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Aggressive Quadriceps Loading Can Induce Noncontact Anterior Cruciate Ligament Injury

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Background: The force responsible for noncontact anterior cruciate ligament (ACL) injuries remains controversial. The patella tendon to tibial shaft angle causes an anterior tibial shear force with quadriceps activation.

Hypothesis: An aggressive quadriceps contraction can injure the ACL.

Methods: The authors characterized noncontact ACL injury and kinematics with aggressive quadriceps loading. Thirteen fresh-frozen knees were potted in a jig held in 20° of flexion while a 4500 N quadriceps contraction was simulated. Knee kinematics were recorded. A KT-1000 arthrometer and a simulated active quadriceps test assessed anterior displacement. Statistics were performed using paired *t* tests and 1-way analysis of variance.

Results: Kinematics revealed the following mean values: anterior displacement, 19.5 mm; valgus, 2.3°; and internal rotation, 5.5°. Mean KT-1000 and active quadriceps test differences were 4.0 mm and 2.7 mm, respectively (statistically significant $P = .002$ and $P = .002$). Six knees showed gross ACL injury at the femoral insertion. Based on ACL injury, KT-1000 differences were statistically significant ($P = .029$).

Conclusions: Aggressive quadriceps loading, with the knee in slight flexion, produces significant anterior tibial translation and ACL injury. This suggests that the quadriceps is the intrinsic force in noncontact ACL injuries, producing a model for further investigation.

Keywords: anterior cruciate ligament; kinematics; noncontact; quadriceps

Injury to the anterior cruciate ligament (ACL) can significantly affect a person's activity level and quality of life. Noyes et al[29] found that an ACL rupture can lead to moderate to severe disability in 31% for walking activities, 44% for routine activities of daily living, and 77% during sporting activities demanding frequent cutting and pivoting maneuvers. More than 200,000 new ACL injuries occur in the United States each year, either from direct or indirect forces on the knee.[1,2] Published results show that 70% to 80% of ACL injuries occur via an indirect force and are classified as noncontact injuries.[6,17,24,25,29] Boden et al,[6] through personal interviews and injury video analysis, were able to reveal several common characteristics underlying the noncontact mechanism. At the time of injury, 35% of patients were decelerating, 31% were landing, 13% were accelerating, and 4% were falling backward. In addition, previous kinematic data produced at our institution found that women, who have known increased ACL injury rates, have smaller knee flexion angles (average 22°), more knee valgus, and greater quadriceps activation when compared to males during the stance phase of athletic cutting manuevers.[8] The accumulated data from these studies have led to several essential extrinsic factors that encompass the noncontact ACL injury mechanism. These factors include an abrupt deceleration or change of direction on a

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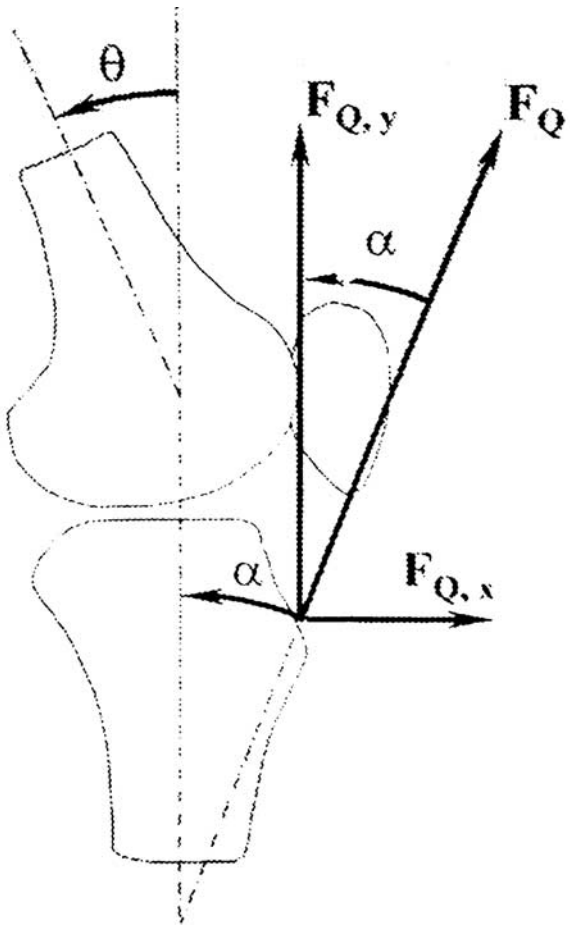

Figure 1. Definition of patella tendon tibial shaft angle (α), knee flexion angle (θ), and the anterior shear force vector ($F_{Q,X}$). F_Q is the total quadriceps muscle force applied to the patella tendon.

planted foot, with the knee near full extension and in slight valgus and the tibia internally rotated, all within the setting of an untoward event in response to minor contact, avoiding contact, or reacting to the ball or play.

Although extrinsic forces involved in noncontact ACL injury are well defined, the role and source of an intrinsic force remains controversial. Numerous cadaveric studies have shown that isometric quadriceps loading produces maximal ACL strain values with the knee near full extension.[21,23,32] The patella tendon to tibial shaft angle can cause an anterior shear force on the tibia relative to the femur with quadriceps activation (Figure 1). This anterior shear force generated by the quadriceps is inversely proportional to knee flexion angle and can be used as an index of load placed on the ACL.[20]

The majority of these studies focus on small quadriceps loads, using open chain models, to discern more favorable postoperative ACL rehabilitation protocols. Through radiographic and mathematical analysis, maximum isometric quadriceps contractions in young trained males have been estimated between 6000 and 7000 N (3000 N normal population),[4,15] and eccentric contraction forces have been estimated to be even higher.[36] Woo et al[38] found the tensile strength of the ACL to be 2100 N in young healthy knees. McConkey first described an eccentric quadriceps contraction as the intrinsic force responsible for ACL injury.[24] He proposed a new mechanism of injury in elite skiers when in a "falling-back" position, which leads to a violent quadriceps contraction in an attempt to regain control. As previously stated, this contraction can create an anterior shear force on the tibia through the patellar tendon causing ACL injury. Therefore, we hypothesize that given the proper circumstances, an intrinsic force generated by a physiologic quadriceps contraction, with the knee in slight flexion, can cause significant anterior tibial displacement and injure the ACL.

The objectives of the study were the following:

1. evaluate changes in anterior laxity of the knee after aggressive quadriceps loading simulating a noncontact ACL injury;
2. evaluate knee kinematics associated with aggressive quadriceps loading and relate these to clinical observation of injuries; and
3. evaluate knee structures for visual evidence of injury after aggressive quadriceps loading and relate these to clinical findings.

To date, no similar biomechanical study has been published.

MATERIALS AND METHODS

Specimens

Thirteen fresh-frozen cadaveric knees (age range, 49 to 93 years) were tested. Eight of the knees were matched pairs with seven female and six male specimens. After thawing at room temperature, all knees were examined for ligamentous injury then dissected to expose the quadriceps tendon, leaving the remaining soft tissue envelope intact. In addition, a small medial arthrotomy was performed to grossly assess ACL integrity. In all knees, no preexisting ligamentous injury was found. After the above initial preparation was completed, the tibia and femur were cut approximately 20 cm from the joint line and secured in aluminum cylinders, using an epoxy potting compound and two crossing 2.0-mm Steinmann pins. They were then mounted in a jig specifically designed to immobilize the femur, with the tibia suspended and allowed to freely rotate in 6° of freedom (Figure 2). Each knee was locked in 20° of flexion, with the remaining degrees of freedom of the tibia unrestricted.

Quadriceps Loading

A 4500 N force (over 1 second), supplied by a servohydraulic material testing machine under load control (8500 Plus, Instron Corp., Canton, Massachusetts), was then

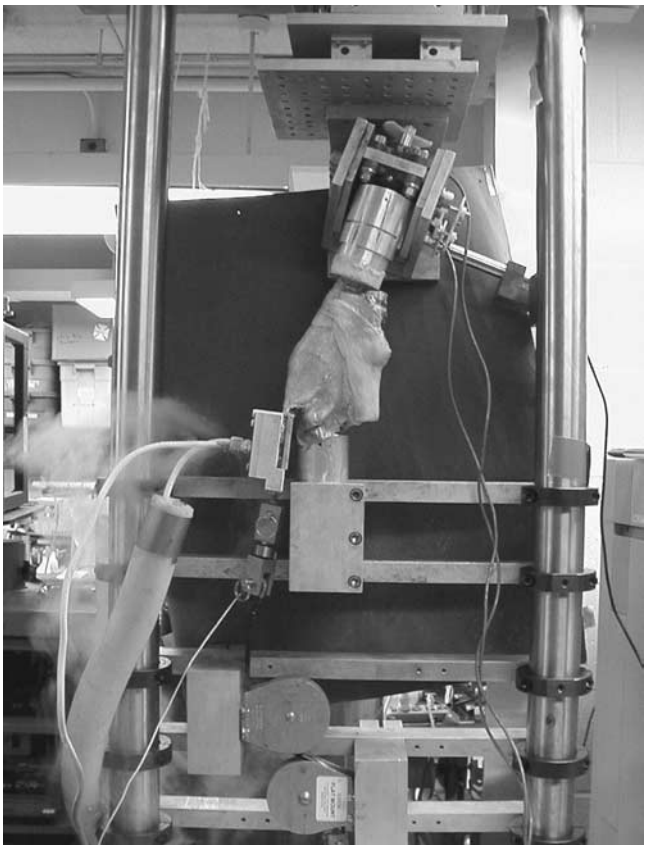

Figure 2. A photograph of the testing jig, with a specimen mounted and the femur positioned inferiorly. A cryoclamp is applied to the quadriceps tendon.

applied to each knee via a liquid nitrogen cryoclamp attached to the quadriceps tendon. An initial static pretension of 40 N was placed on the quadriceps prior to the loading event. The quadriceps contraction was simulated with a ramp loading to a peak load of 4500 N at a rate of 4500 N/sec. The testing system was programmed such that on reaching the 4500 N peak, the load was immediately reduced to 40 N at a ramp rate of 4500 N/sec. As previously stated, Huberti et al.[16] showed that young trained males can produce maximum isometric quadriceps forces of 6000 to 7000 N and normal untrained individuals forces of 3000 N. Therefore, we assumed a 50% increase to produce an aggressive physiologic quadriceps contraction, giving a value of 4500 N, in our untrained specimen population.

Knee Kinematics

During 4500 N quadriceps loading, knee kinematic data were recorded. Anterior, varus/valgus, and internal/external tibial motions were measured by a linear variable differential transformer (Sensotec Inc., Columbus, Ohio) and precision rotary potentiometers (Newark Electronics, Chicago, Illinois), respectively, that were interfaced with the knee jig. Load and kinematic signals were acquired simultaneously at 100 Hz with a 12-bit data acquisition board interfaced with a personal computer. Signals were processed and stored using commercially available software (Labview 6.0, National Instruments Inc., Austin, Texas).

Anterior Displacement Measurement

Anterior tibial displacement was assessed using a KT-1000 arthrometer (Medmetric, San Diego, California) and a simulated active quadriceps test, pre and post 4500 N quadriceps loading. The KT-1000 arthrometer was appropriately secured to the existing tibia and attached aluminum cylinder via a fiberglass mold, which extended distally an additional 25 cm. The femur was secured in a rigid clamp. Each measurement was obtained with the knee in 20° of flexion, as insured by a directly applied goniometer. A 20-pound KT-1000 measurement (89 N) was performed three times on each knee and averaged. Starting from a 10 N pretensioned state, a 400 N force (100 N/sec) was applied by the materials testing system to the quadriceps tendon through the cryoclamp to simulate an active quadriceps test with the knee held in 20° of flexion. The simulated active quadriceps test was repeated three times on each knee with the anterior tibial displacement averaged. The 400 N force produces an estimated 130 N anterior shear force on the tibia, closely matching the displacement force of a 20-pound KT-1000 measurement.

Knee Dissection

After all quadriceps loading and KT-1000 measurements were completed, a formal dissection was performed of the ACL with specific attention to its tibial and femoral insertion sites. In addition, each knee was examined to determine any associated ligamentous or meniscal damage.

Statistical Analysis

Descriptive statistics of kinematic data for all knees were determined. Paired t tests were used to evaluate differences in preanterior and postanterior displacement measurements. One-way analysis of variance (ANOVA) procedures were used to find differences in KT-1000, active quadriceps test measurements, and knee kinematic variables during 4500 N quadriceps loading as a function of the injury status determined at dissection.

RESULTS

Specimen Failures

Two knees sustained tibial plateau fractures during 4500 N quadriceps loading. Gross examination of the fracture sites revealed fatty marrow and osteoporotic bone. As a result, complete data will be reported on the remaining 11 knees.

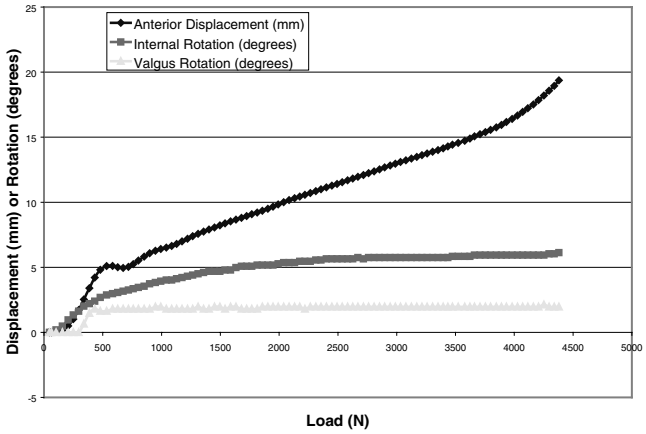

Figure 3. Kinematic data obtained from one specimen that sustained a complete ACL injury.

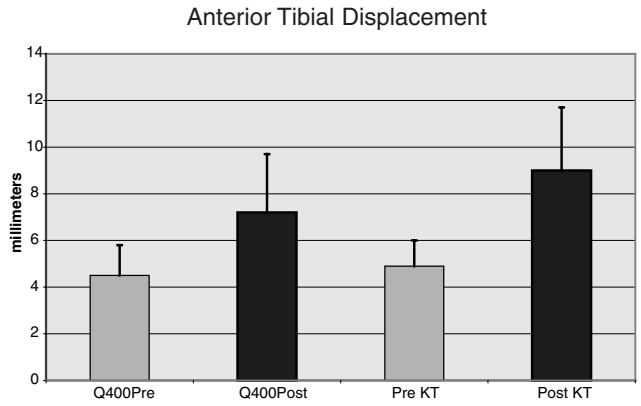

Figure 4. Anterior tibial displacement for 400 N active quadriceps (Q400) and 20-pound KT-1000 (KT) testing, pre and post 4500 N quadriceps loading.

TABLE 1
Mean 4500 N Kinematic Data for All Knees
Based on ACL Injury Status

Gross ACL status	Number of specimens	Anterior displacement (mm)	Valgus	Internal rotation
Complete injury	3	20.60	1.7°	7.4°
Partial injury	3	19.95	3.1°	5.3°
No gross injury	5	18.30	2.0°	3.9°

Knee Dissections

Dissection revealed six knees with gross injury to the ACL at its femoral insertion. Three had complete failures with "empty wall" signs, with no fibers in continuity. The remaining three had partial injuries, one with 25% and two with 50% fiber disruption. All partial injuries revealed a consistent fiber disruption pattern involving the anterior half of the femoral insertion, with the posterior fibers and synovial layer remaining intact. In all specimens, no injury was seen at the ACL tibial insertion. Five specimens revealed no gross change in ACL appearance. In all knees, no associated ligamentous or acute meniscal damage was seen.

4500 N Kinematic Data

All knees displayed similar kinematics of anterior, valgus, and internal tibial rotation (Figure 3). The following mean values were recorded: anterior displacement, 19.5 mm; valgus, 2.3°; and internal tibial rotation, 5.5°. No significant differences were found in kinematic variables based on the injury status of the ACL; however, in knees in which the ACL was grossly injured a trend toward more anterior tibial displacement and internal tibial rotation was found (Table 1).

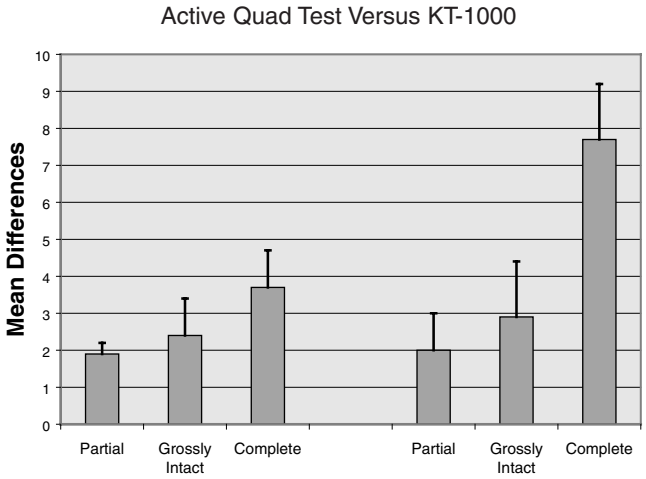

Figure 5. Mean differences for active quadriceps (left) and KT-1000 measurements (right) based on ACL injury pattern. Partial n = 3, grossly intact n = 5, and complete n = 3.

Anterior Displacement

The mean difference for pre and post KT-1000 measurements was 4.0 mm. Likewise, the mean difference for pre and post active quadriceps tests was 2.7 mm (Figure 4). Using a paired t test, these differences were statistically significant (P = .002 and P = .002). Based on ACL appearance at dissection, mean differences in KT-1000 measurements were as follows: grossly intact, 2.9 mm; partial tears, 2.0 mm; and complete tears, 7.7 mm (Figure 5). A 1-way ANOVA found statistical significance when comparing complete versus grossly intact and partial injuries (P = .029). Likewise, mean differences in active quadriceps testing based on ACL appearance were the following: grossly intact, 2.4 mm; partial, 1.9 mm; and complete, 3.7 mm. Statistical analysis of these differences suggests a trend for a difference between complete versus partial or intact

specimens, but no significance was found. During active quadriceps testing, for knees with complete ACL tears following 4500 N loading, we encountered difficulty reproducing a starting reference position. This was most likely secondary to the jig design, which caused the tibia to translate anteriorly due to the weight of the tibia component, coupled with an acute change in ligamentous laxity, resulting in the inability to keep the tibia in a reduced position relative to the femur. As a result, mean differences are probably understated. We attempted to quantify this change by recording the amount of tibial displacement after 4500 N loading from an initial starting position taken before the 4500 N loading. Defined as the *neutral shift,* we found an average displacement for all knees of 13 mm and values based on ACL injury status as follows: intact, 10 mm; partial, 16 mm (results available for one of three knees); and complete, 14 mm. These numbers are not complete for all knees and do not represent total tibial displacement but merely a method to quantify the change in starting reference position during post 4500 N active quadriceps testing.

DISCUSSION

The present study was conducted to examine more closely the noncontact ACL injury mechanism. The ACL is well established as the main restraint to anterior tibial translation. Butler et al[7] have shown that the ACL absorbs 90% of anterior displacement forces. Anterior tibial translation in normal subjects undergoing maximal manual KT-1000 measurements average 8 mm (±2 mm).[9,14,26] Our results show that an aggressive physiologic quadriceps load, with the knee in 20° of flexion, anteriorly displaces the tibia an average of 19 mm. No other studies have been conducted using such an aggressive quadriceps load. It seems intuitive that generating this amount of anterior translation could physically compromise the primary restraining ligamentous structure, the ACL. Daniel and Stone[9] and Mononen et al[27] established a side-to-side KT-1000 difference of 3.0 mm or more as consistent with an abnormal ACL. Based on these data, our mean KT-1000 difference of 4.0 mm is clearly pathologic. The specimens with complete ACL tears at dissection were found to have a mean KT-1000 difference of 7.7 mm, also consistent with Daniel's data, which found mean differences in cadaveric knees after sectioning the ACL of 6.7 mm. However, the results of our active quadriceps test found an overall mean difference of 2.7 mm and in knees with complete ACL tears a mean difference of 3.7 mm. These differences are smaller than would be expected from our KT-1000 results. A plausible explanation for this understated value can be found by our neutral shift data, which showed a mean change in tibial starting position of 13 mm, corresponding to an inability to keep the tibia in a reduced position after 4500 N loading. Therefore, an active quadriceps test is not a reliable method to measure anterior tibial displacement in this model. Future studies will need to take this into account and appropriate changes in jig design and measuring methods made.

Findings at dissection are consistent with clinical and biomechanical results seen elsewhere in the literature. All partial injuries involved the anterior 25% to 50% of the ACL femoral insertion and demonstrated a mean KT-1000 difference of 2.0 mm. Noyes et al[29,31] found, in their series of partial ACL tears, that 28 of 32 involved the anterior femoral insertion, sparing the posterior fiber attachments while producing side-to-side KT-1000 differences of injuries of similar magnitude to our own ranging from 1.0 to 2.5 mm. It appears that partial ACL injuries involving 50% or less of their femoral attachment still provide a restraint to anterior translation. For all complete tears, the femoral attachment site was injured with an "empty wall" sign present, as described by Bach and Warren.[5] Their clinical series of 84 consecutive patients with ACL injuries reported 82% with this arthroscopic finding. Although five knees showed no gross injury at dissection, their KT-1000 data supported a mean difference of 2.9 mm. As previously stated, a 3-mm side-to-side KT-1000 difference is considered abnormal. Noyes et al[30] showed that the ACL could be elongated 50% over its resting length prior to ultimate failure. Kennedy et al[19] loaded the ACL causing a 20% to 30% strain change prior to failure. Gross and histologic inspection of Kennedy et al's specimens revealed a normal outer third of the ligament but underlying interstitial damage within its midsubstance.

Although no histologic studies were performed on our specimens, our anterior displacement data and these studies lead us to conclude that our subset of grossly intact knees sustained interstitial ligamentous damage. This subfailure ligamentous injury, clinically equivalent to a grade I/II sprain, was also examined in an ACL rabbit model. Panjabi et al[32] stretched rabbit ACL ligaments to 80% of ultimate failure load and found no change in overall ligament strength but showed significant changes in load deformation curves, indicating a clear biomechanical change in ligament structure.

The question must arise as to why all knees did not sustain a complete ACL injury. Woo et al[38] demonstrated that the tensile properties of the human ACL are not uniform throughout the population. His results showed that ultimate load to failure decreases with age. In addition, Muneta et al have shown that the cross-sectional area of the ACL is smaller in females compared to males.[28] In the present study, neither specimen age nor sex was controlled. We demonstrated that for all knees, the 4500 N quadriceps force was large enough to cause damage to the ACL; however, for some this force was below the ACL's ultimate load to failure. Future studies need to be done controlling for specimen age and sex to better define ACL failure properties during noncontact injuries.

First characterized by Mink and Deutsch,[26] "bone bruising" is now recognized as an associated MRI finding in the ACL injured knee. Increased MRI signal changes on T2-weighted images reflecting edema and inflammation have been reported at rates of 70% to 90%[11,33,34] in an acute ACL injury. The location of signal changes has been well defined, with the majority occurring in the posterolateral tibial plateau and the terminal sulcus of the lateral

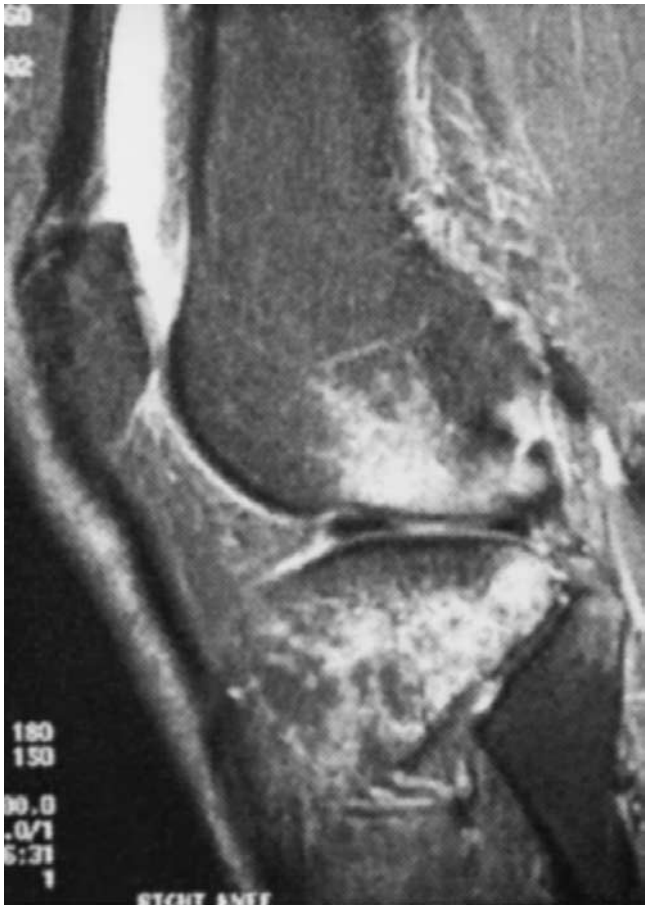

Figure 6. The T2-weighted knee MRI depicts the typical signal change pattern seen in acute ACL injuries at the terminal sulcus of the lateral femoral condyle and the posterolateral rim of the lateral tibial plateau.

femoral condyle. Graf et al[13] found that more than 80% of their patients with acute ACL injuries displayed this pattern (Figure 6).

All knees underwent a violent anterior translation of the tibia an average of 2 cm. This amount of anterior displacement, resulting in injury to the ACL, coupled with slight valgus and internal tibial rotation, can cause the posterolateral tibial plateau to strike the lateral femoral condyle at the level of the terminal sulcus. This "index pivot shift" mechanism was initially proposed by Speer et al[35] and can be further explained by examining the kinematics and static constraints of the knee. During early knee flexion, the rolling-gliding ratio of the femur is 1:2. This places the surface contact point of the femur and tibia posterior. As we have shown, with the knee in early flexion, a large anterior shear force generated by an aggressive quadriceps contraction can injure the ACL. Following ACL rupture, a disruption in the normal rolling-gliding movement of the knee takes place, leading to excessive rolling of the femoral condyle and further displacing the femoral and tibial contact point posteriorly.[3] In addition, based on geometric anatomy, preferential displacement of the lateral tibial plateau occurs. The posterior tibial slope, smaller convex surface, and more mobile meniscus of the lateral tibial plateau, in contrast with the larger, more concave, and well-fixed meniscus of the medial tibial plateau, provide the basis for this argument. With greater displacement of the lateral plateau, the tibia will be internally rotated, and the axially loaded knee can collapse into further valgus due to the development of relative knee laxity. A pivot-shift test performed on a chronic ACL-deficient knee under an open arthrotomy showed that the posterolateral rim of the lateral plateau strikes the terminal sulcus of the lateral femoral condyle.[22] A case report by Vellet et al[37] of a gymnast sustaining an acute ACL injury during landing depicts a subluxated knee with the posterolateral rim locked in the terminal sulcus. The pivot-shift phenomenon is a well-defined sequela of acute and chronic ACL-deficient knees.[10,12] Its potential presence, within milliseconds following the tearing of an ACL, may produce a radiographic snapshot as evidenced by MRI findings of the first event of instability seen in an acute ACL-deficient knee.

Our kinematic data provide further support for the index-pivot-shift mechanism, as knees with gross injury to the ACL showed a trend toward more anterior displacement and internal tibial rotation. However, as hypothesized, significant tibial valgus displacement was not found. This finding can be explained by looking more closely at the testing model. Although a closed chain system was designed, with the femur fully constrained and the tibia locked in 20° of flexion, the actual weightbearing load on the extremity that occurs at the time of ACL injury was not fully reproduced. As a result, the axial load on the knees was minimized. This factor introduces a limitation to the study and may be considered as a possible modification in future designs. The inability to accurately axially load each knee is likely the reason why only isolated ACL injuries were found without associated medial collateral ligament or meniscal pathology. However, this would suggest that the essential kinematic motions of an isolated ACL injury are anterior tibial displacement and internal tibial rotation, without the need for significant valgus displacement.

Johnson et al[18] described a "boot-induced" ACL injury during ski jumping. An extrinsic load is applied to the tibia by the posterior shell of the boot top on landing, when the tails of the skis make first contact and the skier's center of gravity falls backward. The load results in an anterior draw transmitted to the tibia via the boot top, which is attached to the ski. This injury mechanism proposes a purely extrinsic force leading to ACL failure. McConkey provided further insight into this mechanism by proposing that injury resulted when both extrinsic and intrinsic loads were combined.[24] The extrinsic boot-induced forces, described by Johnson et al,[18] preload the ligament, whereas an intrinsic force, from an eccentric quadriceps contraction in an attempt by the skier to regain a more upright position, produces a violent anterior tibial draw and ACL injury.

Although initially described in a skiing model, the same fundamental principles hold true when addressing non-

contact ACL injuries in other sports. We strongly agree with the concept of extrinsic and intrinsic forces underlying the mechanism of all noncontact ACL injuries. We have shown, using a cadaveric model, that an aggressive quadriceps load, with the knee near full extension, produces significant anterior tibial translation causing injury to the ACL. This suggests that the quadriceps can serve as the major intrinsic force in a noncontact ACL injury. Indeed, other possible mechanisms of noncontact ACL injury exist. However, the key to prevention lies within our ability to determine which factor is the limiting step to ACL injury based on the individual and their competing athletic population. To further define and quantify the parameters of the intrinsic and extrinsic forces involved in noncontact ACL injuries, further biomechanical studies should be conducted using similar cadaveric models.

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