Effect of ACL Deficiency on MCL Strains and Joint Kinematics

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In Press, *Journal of Biomechanical Engineering*
Oct. 25, 2006

Keywords:

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ABSTRACT

The knee joint is partially stabilized by the interaction of multiple ligament structures. This study tested the interdependent functions of the anterior cruciate ligament (ACL) and the medial collateral ligament (MCL) by evaluating the effects of ACL-deficiency on local MCL strain while simultaneously measuring joint kinematics under specific loading scenarios. A structural testing machine applied anterior translation and valgus rotation (limits 100 N and 10 N-m, respectively) to the tibia of 10 human cadaveric knees with the ACL intact or severed. A three-dimensional motion analysis system measured joint kinematics and MCL tissue strain in 18 regions of the superficial MCL. ACL-deficiency significantly increased MCL strains by 1.8% ($p<0.05$) during anterior translation, bringing ligament fibers to strain levels characteristic of microtrauma. In contrast, ACL transection had no effect on MCL strains during valgus rotation (increase of only 0.1%). Therefore, isolated valgus rotation in the ACL-deficient knee was non-detrimental to the MCL. The ACL was also found to promote internal tibial rotation during anterior translation, which in turn decreased strains near the femoral insertion of the MCL. These data advance the basic structure-function understanding of the MCL, and may benefit the treatment of ACL injuries by improving the knowledge of ACL function and clarifying motions that are potentially harmful to secondary stabilizers.

Keywords: MCL, ACL, strain, kinematics, knee, ligament, anterior tibial translation, valgus rotation

INTRODUCTION

The mechanical functions of knee ligaments are interrelated, with multiple soft tissue structures contributing to joint stability under externally applied loading conditions [1, 2]. The overlapping function of the anterior cruciate ligament (ACL) and medial collateral ligament (MCL) is a prime example of this concept, as these ligaments share responsibility in stabilizing anterior translation of the tibia and valgus joint opening [3]. Injuries to the ACL and MCL account for 26% of knee trauma [4], with combined ACL/MCL injuries comprising 70% of all multiligament knee injuries [5]. Isolated MCL injuries often adequately heal without surgical intervention, however conservatively treated ACL injuries have a high incidence of unsatisfactory outcomes [6, 7]. Even ACL reconstructed knees exhibit abnormal kinematics [8-10] that may lead to cartilage degeneration [11]. Due to the relationship between the ACL and MCL, treatment of combined or isolated ACL injuries may be improved by an understanding of the mechanical effects of ACL deficiency on MCL function.

The current knowledge of ligament function in the knee joint is largely based on ligament cutting studies that measured changes in laxity after dissecting a specific structure. Experimental studies in cadaveric knees have demonstrated that the superficial MCL is the primary restraint to valgus rotation, and a secondary restraint to anterior translation [3, 12-16], while the ACL is the primary restraint to anterior translation, and a secondary restraint to valgus rotation [3, 13, 14, 17-19]. In addition, the MCL and ACL both resist internal tibial rotation [20-22], with the MCL also resisting external tibial rotation [22, 23]. Recent experiments have investigated local tissue strains and overall force in the ligament during applied loading conditions. Local MCL strains have been measured for single or combined loading conditions, and with the exception of studies by Fischer et al. [24] and Yasuda et al. [25], all MCL strain studies have focused on intact knees

[26-31]. Fischer utilized strain measurement techniques to determine if function of the superficial MCL was affected when the posterior aspect of the longitudinal parallel fibers was severed. Significant changes in strain were only seen in an ACL deficient knee, prompting future research to look into the interaction between the superficial MCL and the ACL. Yasuda found that the ACL has minimal affect on the dynamic strain behavior of the MCL when a lateral impact load is applied to the knee, and kinematic studies determined that when the MCL is intact, the ACL has only a small influence on valgus laxity near full knee extension [12, 22]. Nevertheless, force measurement studies found that when the MCL is intact, ACL tension significantly increases with the application of a valgus load over a range of flexion angles [20, 32]. These results leave the role of the ACL in resisting valgus rotation in an MCL-intact knee unclear; moreover, it is unknown how ACL-deficiency quantitatively affects regional MCL strain under specific loading conditions.

Interpretation of these interactions may be aided by investigating how ACL-deficiency alters localized MCL strains and joint kinematics. Local measurement of ligament strain provides insight into regional function and the values of strain directly relate to the propensity of the tissue to damage, tear or rupture [33]. Further, local strain measurements on heterogeneous tissue structures are necessary to understand how externally applied kinematic motions are resisted by specific regions [26, 34]. This information would provide a broad visualization of MCL structural behavior and would identify the loading configurations that the MCL resists actively. Finally, studying MCL strain patterns in normal and ACL-deficient knees can afford a physiological baseline to compare the in-vitro efficacy of ACL reconstruction techniques. The objective of this research was to quantify regional MCL strains and joint kinematics in the normal and ACL deficient knee during anterior translation and valgus rotation at varying flexion

angles and tibial axial constraint. Two hypotheses were tested: 1) Strains in the MCL increase following ACL transection during application of anterior translation, and 2) strains in the MCL increase following ACL transection during application of valgus rotation.

MATERIALS AND METHODS

Kinematic tests were performed on human knees before and after ACL transection. Briefly, the tibia of each knee was subjected to cyclic anterior-posterior (A-P) translation and varus-valgus (V-V) rotation at flexion angles of 0°, 30°, 60°, and 90° degrees with tibial axial rotation constrained or unconstrained. MCL tissue strains and joint kinematics were recorded during the entire application of anterior translation and valgus rotation to the tibia. Following testing, the MCL was dissected free from the joint to measure the stress-free strain pattern of the MCL. All tissues were kept moist with 0.9% saline solution throughout dissection and testing.

Specimen Preparation. Ten cadaveric right knees were acquired fresh-frozen from male donors (donor age = 56±7 yrs, range 18-65). Each knee was from mid-tibia to mid-femur and was allowed to thaw for 16 hours prior to dissection. All skin, fascia, muscle, and other periarticular soft tissue surrounding the knee joint was removed, including the patella and patellar tendon. One knee was eliminated from testing due to the absence of a medial meniscus, otherwise all knees showed no sign of arthritis or previous soft tissue injury. The fibula was secured to the tibia with a stainless steel screw to ensure an anatomical position was maintained. The femur and tibia were potted in mounting tubes using catalyzed polymer resin (Bondo, Mar-Hyde, Atlanta, GA). Two L-shaped white blocks (the "kinematic blocks") with three black acrylic markers (4.75 mm dia.) were fastened to the anterior femoral condyle and the posterior

aspect of the tibia using nylon screws. Kinematic blocks were used to record the three-dimensional kinematic motions of the tibia and femur during testing.

A 3 x 7 grid of markers (2.3 mm dia.) was adhered to the MCL using cynoacrylate (Fig. 1). These markers formed 18 gauge lengths for strain measurement, with each gauge length spanning approximately 15 mm along the collagen fiber direction. The markers were teased with tweezers after adhesion to verify that they were attached to the superficial MCL fibers and not to the fascia. The markers in the first and second rows were arranged along the anterior and posterior longitudinal parallel fibers of the superficial MCL, respectively (Fig. 1). Distal to the joint line, the markers in the third row were affixed to the distal oblique fibers of the superficial MCL. Proximal to the joint line, the markers in the third row were affixed to the anterior portion of the posteromedial corner. These naming conventions are consistent with Robinson et al. [35] and Warren and Marshall [36].

Testing Procedure. Each knee was mounted in fixtures on a custom testing machine. The machine and fixtures allowed up to four degrees of freedom through a combination of linear and rotary bearings and actuators (Fig. 2). Flexion was fixed, and either A-P displacement during V-V rotation or V-V rotation during A-P displacement was fixed. The tibial fixture permitted tibial axial rotation to be either constrained or unconstrained. Thirty-two tests were performed on each knee. A-P displacements were applied to a set force limit and V-V rotations were applied to a set torque limit (limits of ±100 N and ±10 N-m, respectively [22, 26, 37]). Both A-P and V-V tests were performed at four flexion angles (0, 30, 60, and 90 degrees), with tibial rotation either unconstrained or constrained, and the ACL either intact or deficient. Ten cycles were run for each test to precondition the soft tissue structures of the knee. Data was analyzed at the 10^{th} cycle during anterior translation and valgus rotation. Linear and angular

velocities (1.5 mm/sec and 1 degree/sec, respectively) were selected to achieve quasi-static test conditions, minimizing tissue viscoelastic and inertial effects. A bus cable (RTSI, Plano, TX) was integrated with LabView software to enable real-time capture of both the loading data from the multiaxial load cell (Futek T5105, Irvine, CA, accuracy ±2.2 N and ±0.056 N-m) and the positional data from the linear or rotary actuators (Tol-O-Matic, Inc, Hamel, MN, linear accuracy ±0.0025 mm, rotational accuracy ±0.002°).

MCL strains and joint kinematics were measured simultaneously using a 3D motion analysis system that tracked the centroids of the markers attached to the MCL and kinematic blocks (Fig. 2) [34]. The associated software used the modified direct linear transformation method to calculate the 3D spatial coordinates of the markers [34]. The 3D motion analysis system consisted of two high-resolution digital cameras (Pulnix TM-1040, 1024x1024x30 fps, Sunnyvale, CA) equipped with 50 mm 1:1.8 lenses and extension tubes, two frame grabbers (Bitflow, Woburn, MA) and digital motion analysis software (DMAS, Spica Technology Corp, Maui, HI). The extra-capsular location of the MCL and its planar geometry facilitated the use of this motion analysis system for strain measurement. Unconstrained tibial axial rotation of the knee was calculated using the established kinematic conventions of Grood and Suntay [38]. Prior to testing, an electromagnetic digitizer (Immersion Corp, San Jose, CA accuracy ±0.085 mm) was used to create "embedded" coordinate systems based on anatomical landmarks [39, 40]. The centroids of the markers on the kinematic blocks were determined by averaging four digitized points around the circumference of each marker. These centroids were used to create marker coordinate systems. The transformation matrix between the femur and tibia could then be calculated by using the transformation matrices formed between the embedded and marker coordinate systems and the video-tracked kinematic block systems [34].

A testing methodology was developed to initiate ACL-deficient tests from the ACL-intact neutral position. This neutral position was defined for each flexion angle by finding the inflection point of the force response resulting from small cyclic A-P and V-V displacements, with tibial axial rotation unconstrained. Actuator translation and rotation positions were logged so that the original neutral positions could be restored after ACL transection. To mimic ACL-deficiency, the ACL was transected through its midsubstance without removing the knee from the fixture. Care was taken to avoid damage to the PCL. To verify that the ACL-intact testing position was reproduced for the ACL-deficient knee, kinematic block positions were measured in relation to each other and the multiaxial test frame for each flexion angle. After ACL transection, positional information was compared at each flexion angle and adjustments were made if necessary.

Establishment of Reference Configuration for Strain Measurement. Following testing, the MCL was dissected from its femoral and tibial attachments for measurement of the stress-free reference lengths (l_o) for all gauge lengths using procedures that were developed and validated [26, 37]. The motion analysis system measured the stress-free configuration after the isolated ligament relaxed for 10 minutes on a saline covered glass plate. This was an important step for the calculation of absolute strain, as force exists in the ligament when it is attached to its insertion sites. Material properties of ligament, including ultimate and substructural failure limits, have been quantified in the literature using stress-free configurations [33, 41]. Accurate interpretation of strain data therefore required the use of stress-free reference lengths. In this study, it was found that basing strain results on *in-situ* gauge lengths measured at 0 and 30 degrees passive knee flexion, on average significantly under predicted strain by 2.7±0.1% ($p<0.001$) and 1.1±0.1% ($p<0.001$), respectively.

Data and Statistical Analysis. The lengths between marker pairs were determined in the stress-free reference state (l_o) and during peak valgus rotation or anterior translation (l). The lengths were also recorded at the neutral position for all test cases. Tensile strain along the fiber direction was calculated as $\varepsilon = (l - l_o) / l_o$. Repeated measures ANOVA analysis with three within-subject factors (ACL state, knee flexion angle, tibial axial constraint) was used in conjunction with Bonferroni adjusted pair-wise comparisons to measure significance of factors, factor interactions and between factor levels. If significance was found ($p \leq 0.05$), adjusted paired t-tests were used for case by case comparisons. A similar analysis was performed for the kinematic data. A power analysis demonstrated that a sample size of 10 was sufficient to obtain a power of 0.8 when detecting a 1.0% change in the strain, a 1.0 degree kinematic rotation, and 1.5 mm kinematic displacement. Data are reported as mean±standard error, unless otherwise stated.

To represent MCL strains graphically, mean values of regional fiber strain were applied to a finite element mesh of a MCL constructed from one of the specimens [37]. This mesh was input to TOPAZ3D (LLNL, Livermore, CA), which was used to perform a least squares interpolation of fiber strain values between discrete measurement locations, yielding a continuous spatial representation of the results (Fig. 3-6).

RESULTS

Effect of ACL Transection

ACL-deficiency significantly increased anterior translation by an average 10.0 ± 1.1 mm ($p<0.001$, Fig. 3A), and MCL strains were significantly greater for ACL-deficient cases at peak anterior translation (Fig. 3B). ACL-deficiency did not significantly affect valgus rotation

(p=0.12, Fig. 4A), and MCL strains were not significantly affected by ACL-deficiency at peak valgus rotation (Fig. 4B). ACL transection increased MCL strains by an average 1.8 ± 0.5% at peak anterior translation. In contrast, ACL transection increased MCL strains by only 0.1 ± 0.1% at peak valgus rotation (Fig. 5). The significant strain increases at peak anterior translation ($p<0.05$) occurred along every region of the superficial longitudinal MCL and the region representing the anterior fibers of the posteromedial corner.

ACL transection caused the largest increase in MCL strain during anterior translation at 30 degrees of knee flexion (2.0 ± 1.5%), corresponding with the greatest increase in anterior laxity (12.4 ± 1.3 mm). During anterior translation, the lowest aggregate strain increases due to ACL transection occurred at 0 degrees of knee flexion (1.4 ± 0.7%); however, even with these lower strain increases, 0 degrees flexion had the greatest absolute strain in both ACL intact and ACL deficient cases. For all anterior translation cases, the largest overall strain increase by region due to ACL transection occurred near the femoral insertion (3.8 ± 1.1%), while the region with the least overall increase was along the distal oblique fibers of the superficial MCL (0.3 ± 0.4%) (Fig. 5).

Effect of Knee Flexion Angle

Knee flexion angle had a significant effect on both anterior translation and valgus rotation ($p<0.001$ and $p=0.01$, respectively). Flexing or extending the knee to 30 degrees from all other flexion angles significantly increased anterior translation (average of 3.1 ± 0.5 mm for all cases, Fig. 3A). Extending the knee to 0 degrees from all other angles significantly decreased valgus rotation (average of 1.5 ± 0.3° for all cases, Fig. 4A). Medial collateral ligament strains in most measurement regions were also significantly affected by flexion angle for all test cases.

Interestingly, MCL strain patterns were changed in a nearly uniform manner with each successive 30 degree flexion, for both loading configurations. This uniform change in strain followed a pattern of small yet significant strain increases along the most anterior row distal to the joint line (0.3 ± 0.2%), coupled with larger and significant decreases in change around the posteromedial corner (-3.5 ± 0.6%). Both ACL-intact knees and ACL-deficient knees exhibited this trend in MCL strain behavior.

Effect of Tibial Axial Constraint

Unconstraining tibial axial rotation significantly increased anterior translation by an average of 0.6 ± 0.1 mm and valgus rotation by an average of 0.7 ± 0.2° ($p<0.001$ and $p=0.001$, respectively), under all test conditions. Overall increases in laxity corresponded with overall decreases in MCL strains of 0.45 ± 0.24% during anterior translation and 0.10 ± 0.17% during valgus rotation. These strain decreases were significant across the majority of longitudinal parallel fibers during anterior translation and near the femoral insertion during valgus rotation.

When tibial axial rotation was unconstrained for the ACL-intact cases, an average internal tibial rotation (ITR) of 9.3 ± 3.8° occurred during anterior translation. Transecting the ACL significantly reduced ITR during anterior translation at 30, 60 and 90 degrees flexion by an average 6.9 ± 3.8° (Fig. 6A). When tibial rotation was unconstrained, the larger ITR in knees with an intact ACL resulted in significantly lower MCL strains in the longitudinal fibers near the femoral insertion (2.5 ± 0.4%, Fig. 6B). In contrast, for ACL deficient knees, ITR was reduced and the decreases in strain near the femoral insertion were insignificant (0.6 ± 0.2%, Fig. 6B). This illustrates that decreased ITR after ACL transection results in increased MCL strains in the longitudinal fibers near the femoral insertion. Statistical analysis further supported this

observation, as there was a significant interaction between tibial axial constraint and ACL-transection along strain regions near the femoral insertion at peak anterior translation ($p<0.05$).

DISCUSSION

Understanding the interdependent functions of the ACL and MCL can clarify the structure-function relationship of both ligaments. This study found that ACL-deficiency significantly increased MCL strains during anterior translation, but had no effect on MCL strains during valgus rotation. Joint kinematics measured simultaneously with MCL strains were consistent with comparable studies [19, 22, 26]. The results support our hypothesis that ACL-deficiency increases MCL strain during anterior translation, which is logical considering the respective primary and secondary roles of the ACL and MCL in restraining anterior translation. Conversely, our hypothesis that ACL-deficiency would increase MCL strain during valgus rotation was rejected. This means that application of a valgus rotation to 10 N-m in the ACL-deficient knee was non-detrimental to the MCL.

The finding that strains in the superficial MCL are insensitive to ACL transection during valgus rotation was surprising considering that the ACL has been shown to be an active stabilizer to valgus rotation when the MCL is healthy [20, 32]. Studies by Fukuda et al. [32] and Miyasaka et al. [20] used force superimposition techniques and strain gauges, respectively, determining that the ACL resists valgus rotation from full extension to 90° flexion. Our results showed that ACL transection produced small, insignificant increases in valgus laxity, yet this increased valgus rotation minimally impacted MCL strains at all flexion angles (average increase was 0.1%, average $p=0.64$). A few explanations on this discrepancy are offered. First, it is possible that the reported ACL force contributions during valgus rotation in an intact knee are easily

accommodated by the MCL after ACL transection. Therefore, MCL strain changes are imperceptible and the integrity of the MCL is unaltered. Another possibility is that other secondary stabilizers might increase their contribution to resisting valgus rotation after ACL transection, allowing the MCL to continue to function normally. Yet, the most likely explanation involves differences in degrees of freedom between testing systems. The testing machine and fixtures in this study permitted up to 4 DOF, while experiments by Fukuda et al. [32] used a 5 DOF system. The 5 DOF system permitted A-P translation during V-V rotation, and demonstrated that coupled A-P translation during V-V rotation increases after ACL transection. Therefore, in an intact knee, the function of the ACL during valgus rotation may be to resist coupled anterior translation, and the ACL only resists pure valgus rotation after the MCL is compromised.

To make clinical interpretations, it was necessary to identify loading conditions that generate increased strains, which was feasible since a stress-free reference was used for strain calculation. A stress-free reference allows direct comparison with material properties reported in the literature. Ligament rupture typically occurs at ~18% strain [41], and the onset of microtrauma or substructural failure in ligament occurs at 5.2% strain [33]. During valgus rotation, maximum absolute strains in the mid-longitudinal MCL fibers remained around 4.4% in both the intact and ACL-deficient knee, below the microtrauma threshold. During anterior translation, ACL transection significantly increased maximum absolute strains along the mid-longitudinal fibers from 2.9 to 5.7%, a strain level that could induce microtrauma. These results show evidence that longitudinal MCL fibers in ACL-deficient knees are initially predisposed to damage from anterior translation. This finding is useful in interpreting results from a study by Tashman et al. [42] who measured kinematic gait changes over two years in ACL-deficient and

ACL-intact canines. Consistent with our results and the literature [19, 22, 43], ACL transection immediately caused large translational increases during anterior translation and small rotational increases during valgus rotation. In the ACL-deficient knee, anterior translation significantly escalated with time. Our data suggest that the MCL initially assisted in stabilizing anterior translation; however, the MCL became strained over time leading to increased anterior tibial displacements. This potential increase in MCL laxity may be one factor in the unsatisfactory outcomes characteristic of conservatively treated ACL injuries [44].

Interestingly, the strains of around 10% in the anterior posteromedial corner during both loading conditions greatly exceed the reported substructure failure threshold. However, these results are deceiving. The material tests that defined damaging strains [33, 41] were tested along the mid-longitudinal MCL fibers and therefore are not directly comparable with regions of the posteromedial corner. Considering that the posteromedial corner has been shown to play a limited role in resisting valgus rotation [22], this tissue is likely less stiff with greater failure strains than the adjoining longitudinal fibers.

Relating joint kinematics and local strains has enabled a better understanding of functional MCL regions and is applicable to clinical diagnosis. Following ACL transection, increased anterior laxity was resisted by fibers near the femoral insertion and along the mid-substance of the parallel longitudinal fibers. The greatest average increase in MCL strain following ACL transection occurred at 30 degrees flexion, consistent with the largest increase in anterior translation. Yet, 0 degrees flexion held the distinguished position of having the greatest absolute strains both before and after ACL transection. Increasing the flexion angle, for both loading conditions, slightly stretched the fibers distal to the joint line along the anterior superficial MCL. Meanwhile, the posterior regions of the superficial MCL and anterior

posteromedial corner uniformly slackened with this increased flexion. This behavioral pattern and the corresponding magnitude of the strain changes were unaffected by ACL transection, and were insensitive to the discrepancies between anterior translation and valgus subluxation patterns observed at different flexion angles. Therefore, regardless of directional loading or ACL condition, progressive flexing of the knee will reduce overall MCL strain. These findings support using a knee flexion angle of 15-30 degrees when administering the Lachman test rather than performing the test at full extension [45]. At a slightly flexed angle, the MCL will not be overstressed, and deviations in joint laxity with the contralateral knee are maximized.

The relationship between tibial axial rotation and the MCL and ACL was further developed in this study. When tibial axial rotation was constrained, knee laxity decreased under both loading conditions. This was at least partially due to increased resistance along the longitudinal fibers of the MCL, which experienced significantly higher strains, particularly during anterior translation. Unconstraining tibial axial rotation permitted internal tibial rotation, which in turn decreased MCL strains. Internal tibial rotation during anterior translation was reduced after the ACL was transected. Thus, the ACL encourages internal rotation, perhaps by "unwinding" during anterior translation [46]. In summary, in an intact knee, the ACL promotes internal tibial rotation, which in turn reduces MCL strains along the longitudinal parallel fibers of the superficial MCL near the femoral insertion.

The specific limitations of the methods used in this study deserve discussion. Joint kinematics may have been altered due to the dissection necessary for strain measurement or because joint compressive forces and stabilizing muscle activity were not represented. Muscle activity has been shown to reduce knee laxity [47]. Therefore, to reproduce the magnitudes of strains from this study in-vivo, greater force and torque limits would likely be required.

Removing the patella may have also influenced MCL strain patterns and joint kinematics. Tests were performed in a controlled environment, and did not undergo high speed motions or combined loading configurations that would have been more analogous to injury causing mechanisms. Strain measurement was based on changes to gauge length between marker pairs. This assumed that strain was homogeneous over the length of these discrete regions. For graphical representation, MCL strain values were interpolated between marker rows, which may not accurately account for inhomogeneities orthogonal to the fiber direction. Finally, strains in the deep MCL were not measured, although previous studies have shown that the deep MCL is half as stiff as the superficial MCL [48] and has a minimal contribution to valgus restraint when the superficial MCL is intact [49].

Through measurement of tissue strain and joint kinematics, this research has improved the understanding of how the ACL and MCL interact. Additionally, results from this study can be used to validate finite element models and improve the governing constitutive equations. The present results and methods can also serve as a baseline to verify that a specific ACL reconstruction technique not only returns the knee to regular joint kinematics, but is capable of returning normal functionality to the intact MCL.

ACKNOWLEDGMENTS

Financial support from NIH grant #AR47369 is gratefully acknowledged.

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FIGURE CAPTIONS

Figure 1: Twenty-one markers defined eighteen regions for strain measurement. The markers in row 1 and row 2 were affixed to the anterior and posterior longitudinal fibers of the superficial MCL. Markers in row 3 inferior to the joint line were considered affixed to the distal oblique fibers of the superficial MCL. Markers in row 3 superior to the joint line were considered affixed to the anterior posteromedial corner.

Figure 2: Schematic of the loading apparatus, depicting a medial view of the knee at 0 degrees flexion. Kinematic blocks are rigidly attached to the tibia and femur for 3-D motion measurement. A – applied anterior-posterior tibial translation. B - applied varus-valgus rotation. C – adjustable flexion angle. D - constrained or unconstrained tibial axial rotation. E - unconstrained medial-lateral translation and joint distraction. F – load/torque cell.

Figure 3: A) Anterior tibial displacements at all flexion angles, with unconstrained tibial axial rotation, before and after ACL transection. B) Average MCL strains at peak anterior translation as a function of flexion angle, with unconstrained tibial axial rotation, before and after ACL transection. Knee anterior laxity and MCL strains significantly increased at each flexion angle in the ACL-deficient knee. * $p < 0.05$, error bars = SD.

Figure 4: A) Valgus rotation at all flexion angles, with unconstrained tibial axial rotation, before and after ACL transection. B) Average MCL strains at peak valgus rotation as a function of knee flexion angle, with unconstrained tibial axial rotation, before and after ACL transection. ACL transection had no significant effect on valgus laxity or MCL strains. Error bars = SD.

Figure 5: MCL strain changes due to ACL transection at peak anterior translation and valgus rotation, averaged over all cases. Transection significantly increased MCL strains during anterior translation, but had no effect on MCL strains during valgus rotation. * $p < 0.05$ (within a region).

Figure 6: **A)** Internal tibial axial rotation from neutral to peak anterior translation, 30 degrees knee flexion, before and after ACL transection. B) Average MCL strains at peak anterior translation, 30 degrees knee flexion, with fixed and unconstrained tibial axial rotation, before and after ACL transection. In the ACL-intact knee, unconstraining tibia axial rotation significantly reduced strain along the anterior MCL. After ACL transection, internal tibial rotation was significantly decreased and MCL strain was unaffected when tibial axial rotation was unconstrained. Thus, in the intact knee, the ACL promoted internal tibial rotation during anterior translation, which relieved strain in the MCL. This also occurred at 60 and 90 degrees flexion. * $p < 0.05$

FIGURE 1:

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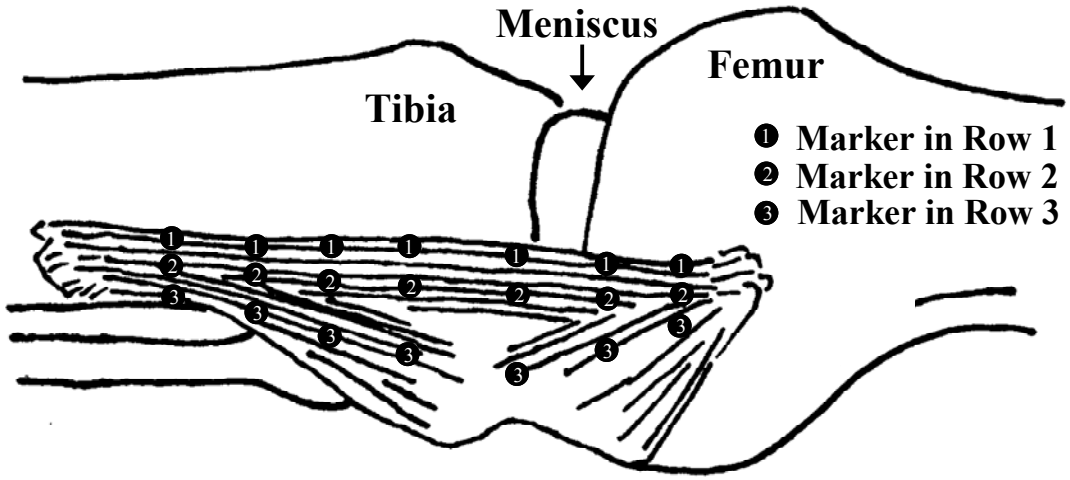

FIGURE 2

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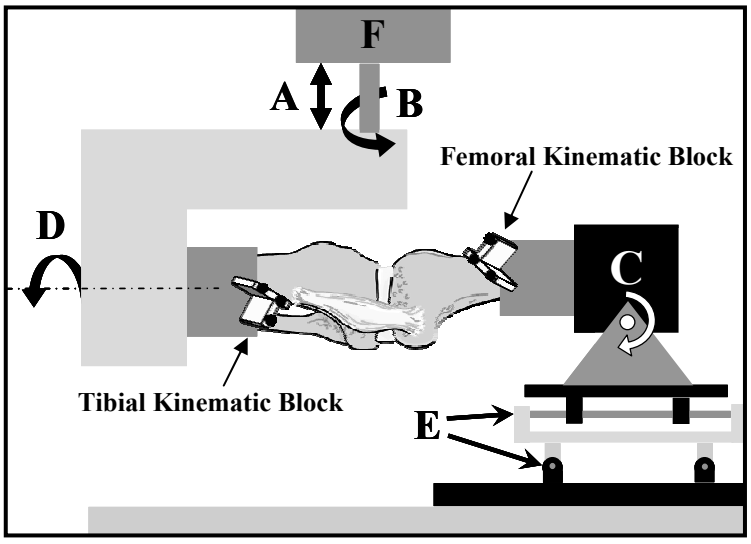

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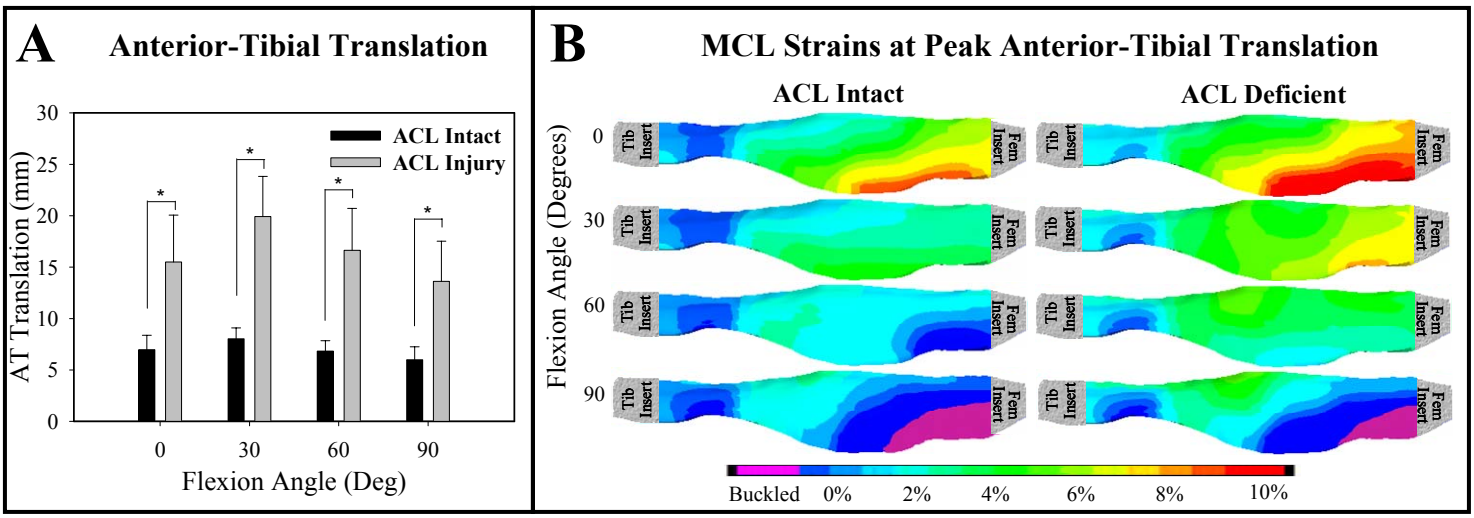

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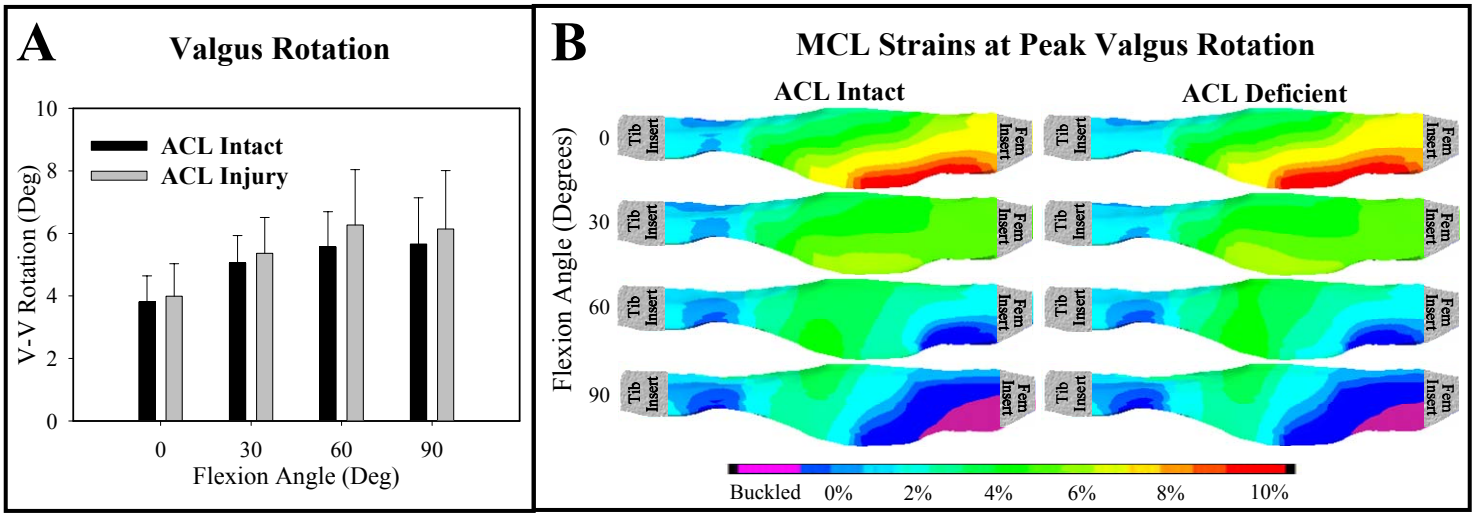

FIGURE 5

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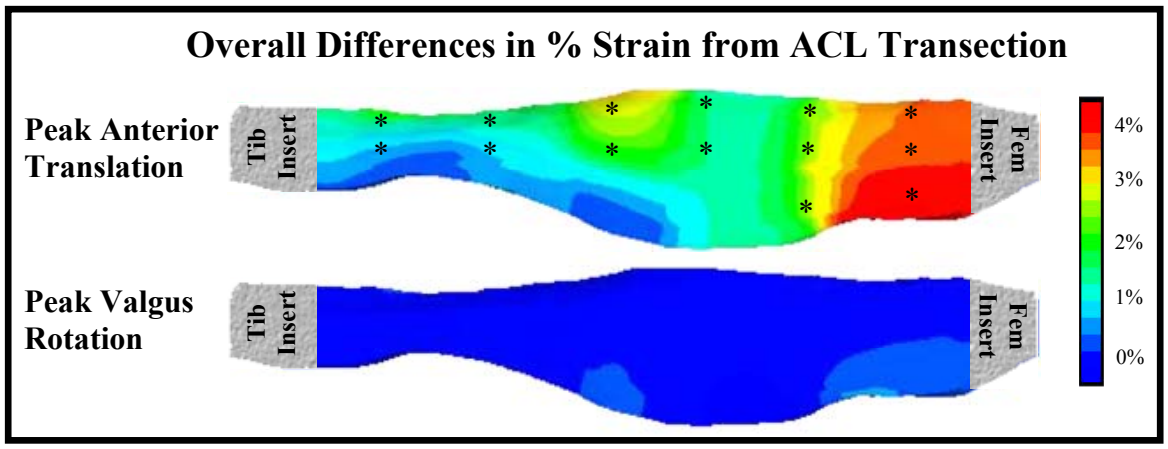

FIGURE 6

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