**INTRODUCTION:**
Knee hyperextension is a serious and disabling injury in both the athletic and general population. Understanding the mechanism of injury and the resulting biomechanical and anatomical defects are crucial for surgical soft tissue reconstructions. For example biomechanical analysis has shown that untreated injuries to the posterolateral corner of the knee may contribute to posterior cruciate ligament graft failure.\(^1\) Knee stability results from a complex interplay between dynamic and static soft tissue restraints. The purpose of this study was to quantify the effects of knee hyperextension injury on knee laxity in a human cadaver model and qualitatively assess the anatomical injury pattern.

**METHODS:**
Six fresh-frozen cadaveric knees were used with an age range of 73–95 years. All specimens were determined to be free from gross pathology or instability. Specimens were then dissected leaving only the extensor mechanism, surrounding retinaculum, collateral ligaments, capsule, anterior cruciate ligament (ACL), posterior cruciate ligament (PCL), and posterior lateral corner (PLC) intact. Digitizing markers were consistently placed on both the femur and tibia. Both the femur and tibia were rigidly potted in plaster of Paris. Specimens were next rigidly mounted to a custom knee testing system that simulates clinical laxity tests. Following preconditioning, the knee laxity was quantified by measuring the movement of the digitizing markers with a Microscribe 3D LX (Immersion Corp., San Jose, CA). The knee laxity measurements consisted of anterior/posterior (AP) laxity, internal/external rotational (IE) laxity, and varus/valgus (VV) laxity. Clinical laxity tests were reproduced with a 30 N load for AP testing, 2.9 N-m torque for VV testing, and 1.9 N-m torque for IE rotational testing. The laxity data were collected at both 30 and 90 degrees of knee flexion angle for the intact specimens and then following 15 and 30 degrees hyperextension injury.

Isolated knee hyperextension injury to 15 and 30 degrees was produced with a custom testing system mounted to an Instron machine (Instron Corp., Canton, MA). (Figure 1) Load deformation curves were generated to quantify the structural integrity of the knee following hyperextension injury. Following biomechanical assessment, a detailed dissection was performed to document the injured structures in the knee. Repeated measures ANOVA with a Tukey-Kramer Post Hoc Test (p < 0.05) was used for statistical comparison.

**RESULTS:**
**Biomechanical Results**
Knee hyperextension of 15\(^{\circ}\) resulted in significantly (p < 0.05) increased rotational laxity at 90\(^{\circ}\) KFA (3.7 ± 0.2\(^{\circ}\)) and VV laxity at 30\(^{\circ}\) KFA (1.8 ± 0.1\(^{\circ}\)). Knee hyperextension of 30\(^{\circ}\) resulted in the following significant (p < 0.05) increases: AP laxity at 90\(^{\circ}\) KFA (4.5 ± 0.2mm), AP laxity at 30\(^{\circ}\) KFA (4.6 ± 0.2mm), rotational laxity at 90\(^{\circ}\) KFA (8.4 ± 0.1\(^{\circ}\)), rotational laxity at 30\(^{\circ}\) KFA (14.4 ± 0.5\(^{\circ}\)), and VV laxity at 30\(^{\circ}\) KFA (3.0 ± 0.1\(^{\circ}\)). Stiffness, calculated from initial load/deformation curves, decreased significantly from 15\(^{\circ}\) to 30\(^{\circ}\) knee hyperextension injuries (p < 0.05).

**Anatomical Dissection**
The ACL was intact on two specimens and incompetent with partial femoral avulsion on four specimens. The PCL was intact on all specimens. The MCL was intact on all specimens. The posterolateral corner of the knee revealed the following injuries: LCL was intact on four specimens and two specimens had LCL avulsions at the fibula. The popliteus tendon was intact on two specimens, completely avulsed at the femur in one specimen, and partially avulsed at the femur at the posterior aspect of the fossa in three specimens. The popliteofibular ligament was intact in all specimens. The fabellofibular ligament was intact on all specimens.

**DISCUSSION:**
The results from this study suggest progressive damage to translational and rotational knee soft tissue restraints with increasing hyperextension. Knee hyperextension to 30\(^{\circ}\) caused the most significant increase in anterior/posterior and rotational laxity. The absence of gross PCL injury with increased AP translational injury may signify midsubstance deficiency in the cruciate ligaments. Previous study has shown, with a cadaveric knee hyperextension model, that posterolateral structures failed at 23\(^{\circ}\) hyperextension, followed by PCL tearing and no ACL deficiencies.

(2) Our study showed a general injury pattern to the posterolateral corner, partial femoral ACL avulsion in four specimens, and no PCL injuries. The injury pattern was mostly isolated to the posterolateral corner structures. Knee hyperextension injury damages multiple soft tissue structures in the knee. Therefore, these injuries must be evaluated with appropriate mechanism of injury history and multi-directional clinical laxity tests to prepare for surgical soft tissue reconstructions.