Biomechanical Effects of Isolated PCL and Concomitant PCL/Posterolateral Corner Deficiencies of the Knee

INTRODUCTION:
Injury to the posterior cruciate ligament (PCL) of the knee may cause instability, increase the risk for cartilage degeneration and lead to osteoarthritis in the long term.1 The cause for increased risk for osteoarthritis as a result of PCL deficiency is not well understood. Although individuals may have increased knee joint laxity as a result of PCL injury, how this laxity affects knee joint mechanics when combined with muscle and shear loads that occur during walking is not well established. PCL deficiency may alter the biomechanics of the knee and lead to abnormal joint stresses in the medial and lateral compartments. Increased joint contact pressures may eventually lead to osteoarthritis.

The purpose of this study was to examine how isolated PCL deficiency or combined PCL and posterolateral corner (PCL/PLC) injury may impact knee joint biomechanics at knee flexion angles that occur during walking. Specifically, anterior-posterior tibial (AP) translation, internal-external (IE) tibial rotation, valgus-varus (VV) tibial rotation, medial and lateral peak tibio-femoral contact pressure were analyzed for intact, PCL deficient and PCL/PLC deficient conditions using finite element modeling and cadaveric testing.

MATERIALS AND METHODS:
A three dimensional finite element (FE) human knee model developed from CT (axial images at 1 mm per slice) and MRI scans of a healthy 25 year old female was used to simulate intact and PCL deficient conditions (Fig. 1B). The simulations were conducted using ABAQUS 6.7 software (Simulia, Rhode Island, USA). The femur, tibia, fibula and patella were modeled using solid 3D hexagonal brick elements. The PCL, anterior cruciate ligament, medial collateral ligament, lateral collateral ligament, and posterior capsule of the knee were modeled using three dimensional truss elements. The meniscus was modeled as an interaction between tibial and femoral articulating surfaces. The material properties of the cortical and cancellous bone, cartilage, and ligaments were based on data in the literature. Muscle preloads were applied to the semimembranosus (100N), biceps femoris (100N), and quadriceps (400N). A posterior shear-compression load (150N) was applied to the proximal tibia to simulate weight bearing conditions (Fig 1A). A component of this force (parallel to the tibia) acted in compression, while a component (perpendicular to the tibia) acted as a posterior shear load depending on the knee flexion angle. The femur was fixed, allowing the tibia to move freely along the articulation of the femoral condyles. The knee was moved through the range of knee flexion (0-60°). PCL deficiency was simulated by removing the ligament structures from the intact model.

CONCLUSIONS:
Clinical consequences of PCL deficiency are not well understood and surgical reconstruction for PCL injury is controversial.1 The FE data presented in this study are similar to previous cadaveric studies, which indicates that the current FE model is an accurate representation of knee joint biomechanics during different knee flexion angles for intact and PCL deficient conditions. The minimal differences in kinematics and contact pressures between intact and isolated PCL deficient conditions indicate that isolated PCL injury may not be clinically significant. However, the combined PCL/PLC deficient conditions demonstrated much higher peak contact pressures in the medial and lateral compartments compared to isolated PCL deficiency. This higher contact pressure may be an important risk factor for the development of osteoarthritis in the long term. While isolated PCL injury may not significantly alter knee joint biomechanics, concomitant PCL/PLC injury may cause dramatic changes in knee joint biomechanics that necessitate surgical intervention to restore normal kinematic and contact pressure behavior in the knee joint.

REFERENCES:
2. Balasubramanian et al. 2007. 53rd ORS Meeting; Paper 0096.