

INVESTIGATION OF THE BIOMECHANICS OF UPPER AND LOWER EXTREMITIES USING A ROBOTIC TESTING SYSTEM

CONRAD WANG, MD, GUOAN LI, PhD, HARRY E. RUBASH, MD

BIOENGINEERING RESEARCH LABORATORY, MASSACHUSETTS GENERAL HOSPITAL

INTRODUCTION

The Bioengineering Research Laboratory has established and used a robotic testing system to precisely measure kinematics and kinetics in human cadaveric specimens, both under zero loading conditions and under simulated muscle loads. This testing system provides a significant improvement over traditional, invasive methods of cadaveric biomechanical testing, as forces and displacements can be precisely measured in a non-destructive fashion.⁴ Moreover, a single specimen can be sequentially tested after serial modifications, which reduces the effect of interspecimen variations on statistical power.

The testing system (Figure 1) consists of a robotic manipulator and a six-degree of freedom load cell. The robotic manipulator (Kawasaki UZ150, Kawasaki Heavy Industry, Japan) is a position-controlling device with a high degree of repeatability for position and orientation. The robotic manipulator can learn the complex motion of a specimen both under unloaded conditions and in response to external loads, and can reproduce these motions in subsequent tests after the specimen has been surgically modified. For example, using the setup shown in Figure 1, the robotic manipulator can define a “passive path” of the knee under zero loading by incrementally determining a flexion path that minimizes forces and moments in the remaining five degrees of freedom. Using the principle of superposition, changes in the kinematic behavior of the knee after surgical modification or in response to simulated muscle loading can then be compared to the original passive path. In this manner, the effects these modifications or loading conditions have on the kinematics and contact forces in the knee can be determined.

To date, the robotic testing system has been implemented in testing protocols of the forearm and knee. We would like to present an overview of current research projects in the Bioengineering Research Laboratory using this robotic testing technology.

ROLE OF THE INTEROSSEOUS LIGAMENT IN FOREARM LOAD TRANSMISSION

Louis E. DeFrate, MS, Shay J. Zayontz, MD, Guoan Li, PhD, James H. Herndon, MD, MBA

Fracture of the radial head is a common traumatic injury to the forearm, and may be accompanied by a disruption of the interosseous ligament (IOL), a ligament that connects the radius and ulna throughout most of the length of the forearm. After such an injury, the radius may migrate proximally, leading to chronic wrist pain, loss of forearm motion, and reduced grip strength.⁷ The IOL is thought to be important to the stability of the forearm. Therefore, it has been suggested in the literature that a reconstruction of the IOL in combination with appropriate treatment of the radial head fracture and stabilization of the distal radioulnar joint would result in a better clinical outcome than reconstruction of the radial head alone.¹¹

In order to design a suitable reconstruction, it is important to understand the biomechanical role of the IOL in the intact forearm. Previous studies have used load cells implanted directly in the radius and ulna to quantify the force

Conrad Wang, MD is a Resident, Harvard Combined Orthopaedic Residency Program.

Guoan Li, PhD is an Assistant Professor, Orthopaedic Surgery/Bioengineering Harvard Medical School.

Harry E. Rubash, MD is Chief, Department of Orthopaedic Surgery, Massachusetts General Hospital and Edith M. Ashley, Professor of Orthopaedic Surgery, Harvard Medical School.

Address correspondence to:

Guoan Li, PhD
Bioengineering Laboratory
Massachusetts General Hospital
330 Brookline Ave., RN 115
Boston, MA 02215
Telephone: 617-667-7819
Email: gli@obl.bidmc.harvard.edu

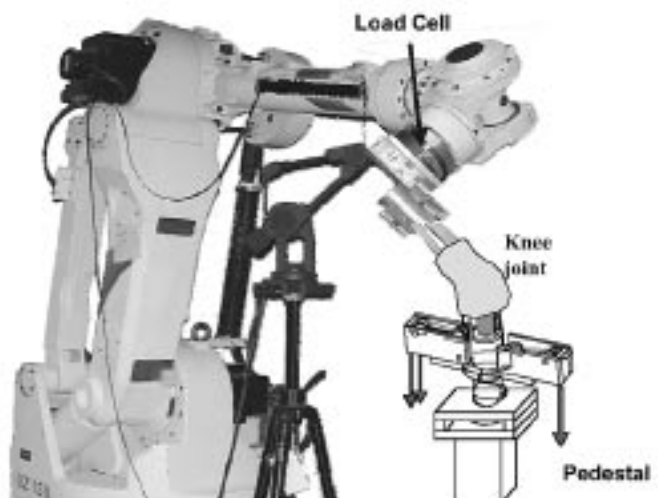


Figure 1. The robotic testing system. Simulated muscle forces are applied via a pulley system with ropes attached to tendons (black arrows)

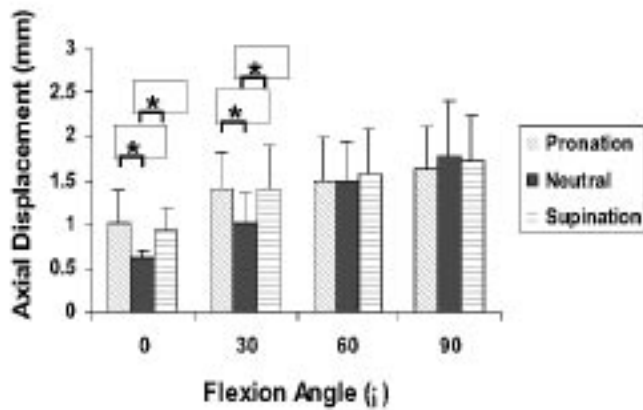


Figure 2. Plot of axial displacement of the radius vs. elbow flexion angle with forearm in pronation, neutral, and supination. * $p < 0.05$.

transferred across the IOL under compressive loads applied to the forearm.^{3,7,10} However, implantation of the load cell requires soft-tissue dissection and disruption of the integrity of the radius and ulna, potentially changing the structural properties of the forearm. These studies have not investigated to what extent disruption of the bony architecture affects the experimental determination of IOL force. Furthermore, most of these studies have only examined the function of the IOL at one elbow flexion angle.^{3,7}

By using the robotic testing apparatus, we have been able to noninvasively test a series of human cadaveric forearms at varying elbow flexion and forearm rotation positions, to determine *in situ* forces in the IOL in response to a 100N compressive load along the long axis of the radius. Testing was performed with the bones and soft tissues of the forearm intact, and transection of the IOL was performed via an incision along the subcutaneous border of the ulna, with minimal disruption of soft tissues and closure of the wound in layers prior to testing.

Our experiments demonstrated that displacement of the radius in response to axial compressive load varied with elbow flexion and forearm rotation (Figure 2). With the forearm in neutral, axial loading resulted in maximal displacement at 90° of elbow flexion. Similar patterns of increasing displacement with increasing elbow flexion were observed with the forearm in supination and pronation.

In neutral forearm rotation, *in situ* IOL force was minimal at full extension and increased to a maximum of 19 N at 90° of elbow flexion. In contrast, loading of the forearms in pronation and supination resulted in maximal IOL force at 30° of elbow flexion; minimal force was again noted at full extension for both forearm supination and pronation. Over all flexion angles, the highest force was observed when the forearm was supinated; the average forces transferred from the radius to the ulna were 36%, 21%, and 15% of the applied forearm load in supination, pronation, and neutral, respectively.

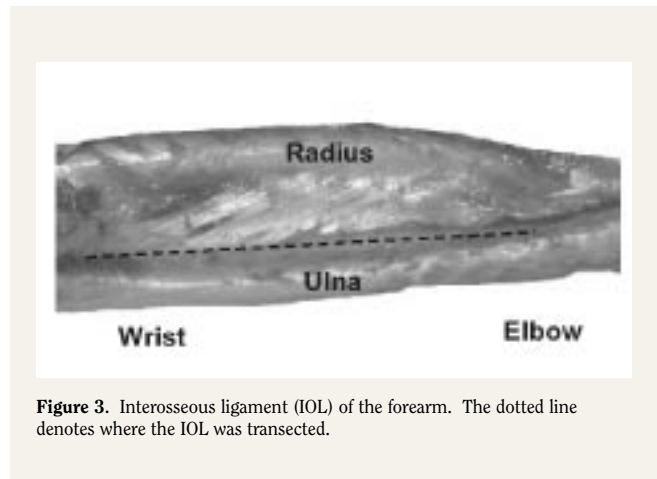


Figure 3. Interosseous ligament (IOL) of the forearm. The dotted line denotes where the IOL was transected.

These findings indicate that the IOL plays a significant biomechanical role in the transmission of force from the radius to the ulna in the forearm. The amount of force that the IOL transfers depends on whether the forearm is in pronation, supination, or neutral, and also varies with flexion of the elbow. At all flexion angles, the force that the IOL transfers from the radius to the ulna was greater in supination than in pronation or neutral rotation. These findings are similar to those reported by others. Morrey *et al.* reported that, under simulated muscle loads, force transferred via the radiocapitellar joint was greater in pronation than in supination, implying that more force was carried in the IOL in supination.⁷ Using load cells directly implanted in the radius and ulna, Birkbeck *et al.* reported that the IOL transferred 17%, 6%, and 6% of applied axial load when the forearm was supinated, neutral, and pronated, respectively.¹

Advantages to our experimental technique include the measurement of forces and displacements with no disruption of the bones and minimal disruption of the soft tissues surrounding the forearm. Previous studies have reported force in portions of the IOL (e.g. the central band).^{5,6,12} Our experiment quantified the force in the entire IOL. Schneiderman *et al.*¹⁰ observed that anatomically, the IOL has a number of thickened bands throughout its length (Figure 3), and suggested that strain in different portions of the IOL is not uniform and varies with different positions of the forearm. Published studies have differed in reported values for IOL central band dimensions,^{3,12} which implies that either there is significant variation in the morphology of the central band, or that proper identification of this structure is difficult. Therefore, measurement of force transferred by the IOL across its entire length may be a more accurate measure of its contribution to forearm biomechanics.

Clinically, most radial head fractures occur with the forearm pronated. We found that, with the elbow in full extension, the IOL carried only 8% of the applied axial load in pronation, compared with 31% in supination. Under axial loading, less force in the IOL with the forearm in pronation

implies greater force transmitted across the radiocapitellar joint. Therefore, in a fall on an outstretched hand with the forearm pronated and elbow extended, the IOL does not function as effectively to bear axial load, resulting in greater load transmission through the radial head and increasing the likelihood of radial head fracture. Similarly, our results suggest that the forearm should be immobilized in supination after isolated radial head fracture, to minimize forces transmitted across the radial head. Finally, the significant role of the IOL in axial force transmission in the forearm suggests that repair or reconstruction of the IOL may be indicated in Essex-Lopresti lesions in order to improve clinical outcomes.

In summary, a minimally invasive technique was utilized to quantify the forces transferred by the IOL from 0° to 90° of

flexion with the forearm in pronation, neutral, and supination. Our results demonstrate that the IOL plays an important biomechanical role in bearing axial load in the forearm. The IOL transfers load from the radius to ulna across the full range of forearm flexion with maximum load transferred in supination at all flexion angles. These findings may help explain the pathogenesis of radial head fractures, and have implications on the treatment of forearm injuries. Further work will focus on the incorporation of simulated muscle loads across the forearm and their effect on load bearing in the IOL.

ACKNOWLEDGEMENTS

This study was supported by an AO/ASIF research grant and a fellowship from the Whitaker Foundation.

References

1. Birkbeck DP, Failla JM, Hoshaw SJ, et al. The interosseous membrane affects load distribution in the forearm. *J Hand Surg* 1997; 22A:975-980.
2. Essex-Lopresti P. Fractures of the radial head with distal radio-ulnar dislocation. *J Bone Joint Surg*. 1951; 33B:244-247.
3. Hotchkiss RN, An KN, Sowa DT, et al. Anatomic and mechanical study of the interosseous membrane of the forearm: pathomechanics of proximal migration of the radius. *J Hand Surg* 1989;14A:256-261.
4. Li G, Rudy TW, Sakane M, et al. The importance of quadriceps and hamstrings muscle loading on knee kinematics and in-situ forces in the ACL. *J Biomech* 1999; 32:395-400
5. Markolf KL, Lamey D, Yang S, et al. Radioulnar load-sharing in the forearm. *J Bone Joint Surg* 1998;80A:879-888.
6. Markolf KL, Dunbar AM, Hannani, K. Mechanisms of Load Transfer in the Cadaver Forearm: Role of the Interosseous Membrane. *J Hand Surg* 2000; 25A:674-682.
7. Morrey BF, An KN, Stormont T. Force transmission through the radial head. *J Bone Joint Surg*. 1988; 70A:250-256.
8. Pfaeffle HJ, Manson TT, Tomaino MM, et al. Role of the forearm interosseous ligament: is it more than just longitudinal load transfer? *J Hand Surg* 2000; 25A:683-688.
9. Rabinowitz RS, Light TR, Havey RM, et al. The role of the interosseous membrane and triangular fibrocartilage complex in forearm stability. *J Hand Surg*. 1994; 19A:385-393.
10. Schneiderman G, Meldrum RD, Bloebaum RD, et al. The interosseous membrane of the forearm: structure and its role in Galeazzi fractures. *J Trauma* 1993; 35:879-885.
11. Sellman DC, Seitz WH, Postak PD, et al. Reconstructive strategies for radioulnar dissociation: A biomechanical study. *J Ortho Trauma* 1995; 9:516-522.
12. Skahan JR, Palmer AK, Werner FW, et al. The interosseous membrane of the forearm: anatomy and function. *J Hand Surg* 1997;22A:981-985.

THE EFFECT OF POSTERIOR STABILIZED TOTAL KNEE ARTHROPLASTY ON KNEE FLEXION

Ephrat Most, MS, Shay J. Zayontz, MD, Guoan Li, PhD, Harry E. Rubash, MD

Posterior-substituting (PS) total knee arthroplasty (TKA) was introduced to improve knee flexion by inducing tibiofemoral rollback (posterior femoral translation), and to prevent posterior subluxation of the tibia.^{7,11} The femoral cam is designed to engage the tibial spine of the prosthesis during knee flexion, in order to prevent excessive posterior tibial translation and posterior tibial impingement with flexion, thus increasing the maximum flexion of the TKA.

However, clinical studies have demonstrated that maximum flexion after PS TKA is usually less than 120°.³ The few *in vivo* biomechanical studies that have investigated the mechanisms of this cam-spine system interaction have yielded

inconsistent results on the ability of PS TKA to restore normal knee kinematics. Most have reported reduced tibiofemoral rollback when compared to normal knees.^{1,2,5,12} One drawback of these studies is that they were not able to compare motion of the same knee before and after TKA. It is therefore difficult to objectively evaluate the ability of TKA to restore normal knee kinematics. Moreover, limited quantitative data have been reported on when and how the cam-spine mechanism is effective during flexion-extension of the knee. This information would be invaluable as a basis for further improvement of TKA design, in order to achieve high flexion of the knee (up to 160°) after TKA.

In this study, we tested a series of human cadaveric knee specimens using the robotic testing system, to investigate the kinematic responses of the knee before and after a posterior substituting total knee replacement (NexGen, Zimmer,

Inc.) under simulated muscle loading conditions. Using the principle of superposition, we also determined the cam-spine contact forces as a function of knee flexion and loading condition. Knee kinematics were defined in terms of translation of the lateral and medial femoral condyles on the tibial plateau, from which both anterior-posterior femoral translation and internal-external tibial rotation could be derived. Quadriceps (400N) and hamstring (semimembranosus, semitendinosus and biceps femoris, 200N total) muscle forces were simulated using a pulley system and weights attached to the appropriate muscle tendons.

We found that, in the unloaded condition, posterior translation of the lateral and medial femoral condyles after TKA was significantly reduced compared to that of the native knee at all flexion angles except at full extension (Figure 4). At 120° of flexion, posterior translation of the lateral femoral condyle was reduced by 23%, and posterior translation of the medial condyle was reduced by 40%. Similar trends were observed under simulated muscle loads, as posterior translation of both medial and lateral femoral condyles were reduced when compared to that of the intact knee. Comparison of the kinematics of unloaded and muscle loading conditions at higher flexion angles (90° and 120°) revealed that medial and lateral condyle translations did not differ significantly with the addition of simulated muscle loads.

Contact forces between the femoral cam and tibial spine ranged from 20N to 90N (Figure 5). Forces were lowest without simulated muscle loads during the passive path. Under simulated muscle loads, increasing contact forces were seen with increasing knee flexion, with highest contact forces observed under combined quadriceps and hamstring loading at 120° and under isolated hamstrings loading at 90°.

Our findings are consistent with reported *in vivo* knee kinematics after TKA during various activities.^{2,12,13} Reduced posterior femoral translation of PS TKA during step-up and gait has been reported in *in vivo* studies,^{9,13} which corresponds with our findings of reduced posterior femoral translation after PS TKA under simulated muscle loads.

The cam-spine contact forces measured at low flexion angles were approximately 20N. Fluoroscopy performed during experiments revealed that the cam-spine mechanism was not engaged at these low flexion angles, suggesting that the recorded forces at these angles were not due to direct contact of the posterior surface of the spine with the femoral cam. We believe that this residual force arose from contact of the sides of the spine with the femoral component.

Engagement of the cam-spine mechanism with knee flexion beyond 70° partially restored posterior femoral translation. Reduced posterior translation of the femoral condyles after PS TKA may limit knee flexion by early impingement of the femoral shaft with the posterior edge of the tibial component. This loss of posterior femoral rollback may account for clinical observations of the inability to achieve knee flexion beyond 120° after PS TKA.

According to our results, reduced femoral translation at

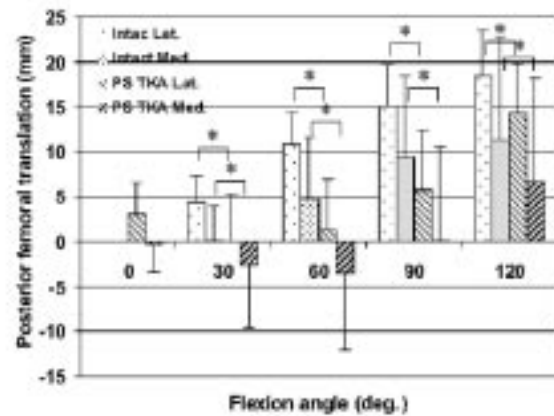


Figure 4. Posterior translation of the lateral and medial femoral condyles in intact and PS TKA knees, under no external load (passive path). * $p < 0.05$. Positions of the trans-condylar line on tibial plateau at different flexion angles were also drawn. The graphic drawings demonstrated mean values of the posterior translation of lateral and medial femoral condyles.

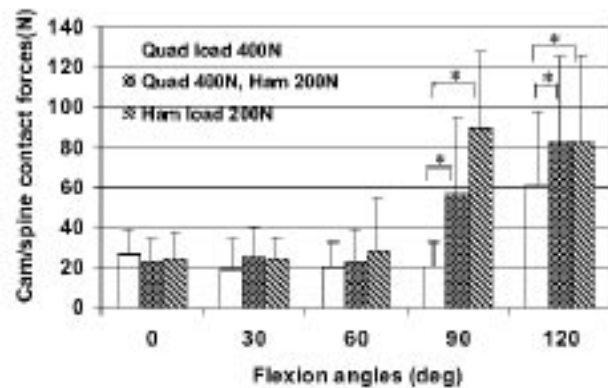


Figure 5. Cam-spine contact forces in posterior-substituting TKA under simulated muscle loads. * $p < 0.05$

high flexion angles after PS TKA is independent of muscle loads. Therefore, optimized prosthetic design and/or surgical technique may be important in determining proper timing of the cam-spine engagement to increase knee flexion. The position of the polyethylene spine on the tibial plateau is an important factor influencing the timing of the cam-spine engagement. Using a computer model, Delp *et al.* reported a posteriorly positioned tibial spine component will cause early cam-spine engagement, thus increasing the extent of posterior femoral translation.⁴ Similarly, different geometrical designs of TKA components as well as variation in surgical techniques could all influence cam-spine behavior and potential knee flexion after PS TKA.

In summary, we investigated the mechanism of the cam-spine system using a single design posterior-substituting total

knee arthroplasty. We found that the cam-spine system was partially effective at restoring tibiofemoral rollback at increasing flexion angles, but did not completely restore posterior rollback to that of the intact knee. This lack of posterior rollback may limit the ability of PS TKA to restore flexion beyond 120° due to early tibiofemoral impingement. We hope that our work will help to develop a rationale for further improvement of surgical

technique and prosthetic designs, with the ultimate goal of restoring normal knee function over the entire range of flexion after total knee arthroplasty.

ACKNOWLEDGEMENTS

This study was supported by a research grant from Zimmer, Inc. and a National Science Foundation (NSF) fellowship.

References

1. Andriacchi TP, Alexander EJ, Toney MK, et al. A point cluster method for *in vivo* motion analysis: applied to a study of knee kinematics. *J Biomech Eng* 1998;120:743-749.
2. Banks SA, Markovich GD, Hodge WA. *In vivo* kinematics of cruciate-retaining and -substituting knee arthroplasties. *J Arthroplasty* 1997;12:297-304.
3. Callahan CM, Drake BC, Heck DA, et al. Patient outcomes following tricompartmental total knee replacement: a meta-analysis. *JAMA* 1994;271:1349.
4. Delp SL, Kocmond JH, Stern SH. Tradeoffs between motion and stability in posterior substituting knee arthroplasty design. *J Biomechanics* 1995;28:1155-1166.
5. Dennis D, Komistek R, Hoff W, et al. *In vivo* knee kinematics derived using an inverse perspective technique. *Clin Orthop* 1996;331:107-117.
6. Hirokawa S, Solomonow M, Lu Y, et al. Anterior-posterior and rotational displacement of the tibia elicited by quadriceps contraction. *Am J Sports Med* 1992;20:299-306.
7. Insall J, Lachiewicz P, Burstein A. The posterior stabilized condylar prosthesis: a modification of the total condylar design. *J Bone Joint Surg* 1982;64A:1317-1323.
8. Li G, Rudy TW, Sakane M, et al. The importance of quadriceps and hamstrings muscle loading on knee kinematics and *in situ* forces in the ACL. *J Biomechanics* 1999;32:395-400.
9. Nilsson KG, Dalen T, Brostrom LA, et al. *In vivo* kinematics in knee replacements with fixed or mobile polyethylene bearings. *Trans ORS* 1997;43:261.
10. Rudy TW, Livesay GA, Woo SL-Y et al. A combined robotics/universal force sensor approach to determine *in situ* forces of knee ligaments. *J Biomech* 1996;29:1357-1360.
11. Stern S, Insall J. Posterior stabilized prosthesis: results after follow-up of nine to twelve years. *J Bone Joint Surg* 1992;74A:980-986.
12. Stiehl J, Komistek R, Dennis D, et al. Fluoroscopic analysis of kinematics after posterior-cruciate-retaining knee arthroplasty. *J Bone Joint Surg* 1995;77B:884-889.
13. Tarnowski LE, Andriacchi TP, Berger RA, et al. Three-dimensional motion of cruciate retaining and posterior stabilized TKAs during walking. *Trans ORS* 1998;44:804.

THE EFFECT OF PCL RECONSTRUCTION ON KNEE KINEMATICS

Thomas J. Gill, IV, MD, Louis E. DeFrate, MS, Conrad Wang, MD, Shay J. Zayontz MD, Bertram Zarins, MD, Guoan Li, PhD

Management of posterior cruciate ligament (PCL) injuries continues to be a controversial topic in sports medicine. Good short-term results have been reported with non-operative treatment, but longer-term studies suggest that late knee arthrosis develops in 36-88% of patients with untreated PCL insufficiency.^{1,7} Appropriate operative treatment for PCL injuries remains unclear. Various methods of PCL repair and reconstruction have met with limited clinical success, and knee arthrosis has been reported in 20% to 60% of patients even after operative treatment.^{3,6,9} Thus, PCL reconstruction continues to be the focus of research efforts.

Experimentally, with the appropriate selection of graft tunnel location and initial graft tension, both single- and double-bundle PCL reconstruction can restore *in vitro* anterior-posterior stability.^{2,10} However, all *in vitro* biomechanical studies

to date have focused on evaluation of PCL reconstruction by measuring anterior-posterior laxity under sagittal plane posterior drawer testing. The effects of surgical reconstruction on the three-dimensional kinematics of the knee remain unclear, especially with regard to rotational kinematics. Few data have been reported on the three-dimensional kinematic behavior of the knee after PCL reconstruction, under functional loading conditions. Our goal in this study was to determine whether *in vitro* PCL reconstruction could restore not only posterior tibial translation but also knee joint rotations under simulated muscle loads.

Using the robotic testing system, a series of human cadaveric knee specimens were tested. A joint coordinate system was established with the knee center defined as the center point of the transepicondylar line and the transepicondylar axis as the flexion/extension axis.

The following testing protocol was applied: the passive flexion/extension path was first determined, and then the kinematic behavior (displacements and rotations) was recorded in response to a posterior drawer load of 130N, a quadriceps

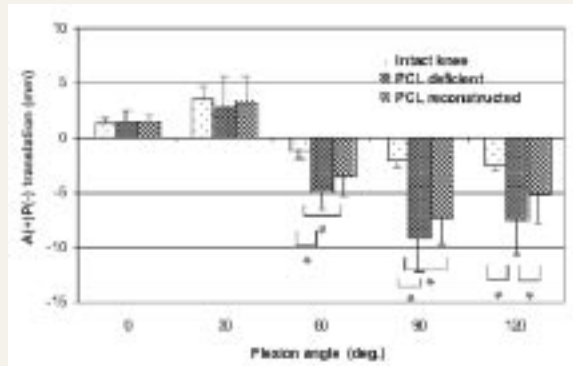


Figure 6. Anterior(+)/posterior(-) tibial translation of the intact, PCL deficient, and PCL reconstructed knee under combined quadriceps and hamstring load. * $p < 0.05$

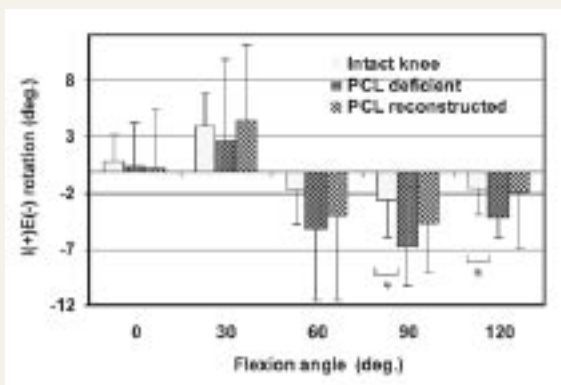


Figure 7. Internal(+)/external(-) tibial rotation of the intact, PCL deficient and PCL reconstructed knee under combined quadriceps and hamstring load. * $p < 0.05$

load (400N), and a combined quadriceps and hamstrings load (400/200N), at selected flexion angles. This protocol was applied to each knee sequentially in intact, PCL resected, and PCL reconstructed states. Reconstruction of the anterolateral bundle of the PCL was performed using an Achilles tendon allograft.⁸ The bone block was fixed on the femoral side using an outside-in interference screw. The tibial tunnel was drilled slightly lateral to midline, 1cm below the articular surface. The posterior capsule was carefully elevated distally off a 2cm area at the tibial tunnel site to facilitate passage of the graft. Tibial fixation was performed with the knee at 90° flexion under a 130N anterior drawer load, using a soft-tissue interference screw augmented with a cortical spiked ligament washer.

In this fashion, each specimen was tested sequentially with the PCL intact, resected and reconstructed, with the serial application of external loads and measurement of knee kinematics. All displacements and rotations were reported with respect to the original, intact knee passive path.

Our results demonstrate that, under posterior drawer loading, posterior tibial translation was greater in PCL resected knees than in intact knees at all flexion angles. PCL reconstruc-

tion significantly reduced posterior tibial translation at all flexion angles when compared to PCL resected knees, to levels not significantly different than those observed in intact knees. These results are in agreement with those reported in the literature.^{2,4,5}

Under both isolated quadriceps and combined quadriceps and hamstring loading conditions, posterior tibial displacement was again greater in PCL resected than in intact knees at high flexion angles. However, the PCL reconstructed knee did not exhibit consistently reduced posterior displacement under simulated muscle loading (Figure 6). This phenomenon demonstrates that after PCL reconstruction, knee kinematics may be restored under posterior tibial drawer loading, but are not consistently restored under muscle loading.

Under simulated muscle loading conditions, increased tibial external rotation was observed at high flexion angles (Figure 7); this again was not consistently reduced by PCL reconstruction. We observed that there was significant variability in rotational knee kinematics after PCL reconstruction, particularly at increased flexion angles. We believe that the location of the tibial tunnel, and therefore the point of graft force application, may play an important role in the rotational kinematics of the reconstructed knee. The precise placement of the tibial tunnel has not been stressed in the current surgical treatment of PCL deficiency.⁸ Further study of the effects of tibial tunnel placement on rotational kinematics of the PCL reconstructed knee is warranted.

Our results demonstrate that PCL reconstruction using an Achilles tendon graft restores normal posterior tibial translation when the knee is examined under posterior drawer loading, but does not completely correct the posterior translation and external rotation of the tibia resulting from PCL deficiency, when assessed under simulated muscle loading. This implies that posterior drawer testing of the PCL reconstructed knee does not accurately evaluate the complex kinematic responses of the knee under simulated physiologic loading conditions. Our ability to successfully reconstruct the PCL and restore knee joint kinematics under physiological loading conditions remains limited. We are currently investigating the influence of the tibial tunnel location on rotational kinematics of the PCL reconstructed knee. In addition, we hope to further elucidate the relationship between kinematics of the PCL reconstructed knee and joint contact pressures. Ultimately, this will help us to predict if PCL reconstruction is successful in preventing the long-term arthrosis of the knee. By understanding the individual benefits and shortcomings of various reconstructive techniques, it is hoped that an optimum PCL reconstruction technique will be developed in order to minimize the onset of degenerative arthritis.

ACKNOWLEDGEMENTS

This study was supported by grants from the Orthopaedic Education and Research Foundation (OREF), the National Football League (NFL) Charity Foundation and a Whitaker Foundation Graduate Fellowship.

References

1. **Boynnton M, Tietjens B.** Long-term follow-up of the untreated isolated posterior cruciate ligament-deficient knee. *Am J Sports Med* 1996; 24:306-311
2. **Burns WCD, LF Pyevich M, Reider B.** The effect of femoral tunnel position and graft tensioning technique on posteior laxity of the posteior cruciate ligament-reconstructed knee. *Am J Sports Med* 1995, 23(4):424-430
3. **Cross MJ, Fracs MB, Powell JF.** Long-term followup of posterior cruciate ligament rupture: A study of 116 cases. *Am J Sports Med* 1984; 12:292-297
4. **Galloway MT, Grood ES, Mehalik JN et al.** Posterior cruciate ligament reconstruction. An in vitro study of femoral and tibial graft placement. *Am J Sports Med* 1996; 24(4):437-445
5. **Harner CD, Janaushek MA, Kanamori A, et al.** Biomechanical analysis of a double-bundle posterior cruciate ligament reconstruction. *Am J Sports Med* 2000; 28(2):144-151
6. **Hughston JC, Bowden JA, Andrews JR et al.** Acute tears of the posterior cruciate ligament: Results of operative treatment. *J Bone Joint Surg* 1980; 62A:438-450
7. **Keller P, Shelbourne K, McCarroll J et al.** Non-operatively treated isolated posterior cruciate ligament injuries. *Am J Sports Med* 1993; 21:132-136
8. **Klimkiewicz JJH, Harner CD, Fu FH.** Single bundle posterior cruciate ligament reconstruction. *Tech Sports Med* 1999; 7:105-109
9. **Lipscomb A, Anderson A, Norwig E, et al.** Isolated posterior cruciate ligament reconstruction: Long-term results. *Am J Sports Med* 1993; 21:490-496
10. **Mannor DA, Shearn JT, Grood ES, et al.** Two-bundle posterior cruciate ligament reconstruction: An *in vitro* analysis of graft placement and tension. *Am J Sports Med* 2000; 28(6):833-845