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The Effects of Grade III Posterolateral Knee Complex Injuries on Anterior Cruciate Ligament Graft Force

A Biomechanical Analysis*

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ABSTRACT

To determine if untreated grade III injuries of the posterolateral structures contribute to increased force on an anterior cruciate ligament graft, we measured the force in the graft in cadaveric knees during joint loading after reconstruction with otherwise intact structures and in the same reconstructed knees after selected cutting of specific posterolateral knee structures. Tests were first performed on the knee with the posterolateral structures intact and then after sequential sectioning of the fibular collateral ligament, popliteofibular ligament, and popliteus tendon. The graft force was significantly higher after fibular collateral ligament transection during varus loading at both 0° and 30° of knee flexion than it was for the same loading of the joint with intact posterolateral structures. In addition, coupled loading of varus and internal rotation moments at 0° and 30° of flexion further increased graft force beyond that with varus force alone. The increase in graft force remained significant with additional sequential cutting of the popliteofibular ligament and popliteus tendon. We believe this study supports the clinical observation that untreated grade III posterolateral structure injuries contribute to anterior cruciate ligament graft failure by allowing higher forces to stress the graft.

Injuries to the posterolateral structures of the knee can easily be missed on knee examinations, especially when there is a concomitant ACL tear. While grade III posterolateral knee structure injuries have been recognized as a cause of frank instability,[1] they have also been implicated as a leading cause of ACL graft failure.[21]

Over the past few years, the complexity of the posterolateral corner of the knee has become better understood. The unique biomechanics of the convex lateral tibial plateau and its articulation with the convex lateral femoral condyle create a setting for marked instability in the face of injury to the posterolateral structures. Seebacher et al.[24] described the anatomy of the posterolateral aspect of the knee as a three-layered complex. More recently, articles describing the anatomy of the individual structures of the posterolateral aspect of the knee and the diagnosis of posterolateral rotatory instability have provided a clearer understanding and, in turn, have sparked further study of injuries to these structures.[11–14,25,27,28]

Anterior cruciate ligament reconstruction is one of the most frequently performed surgical procedures in orthopaedics. Failure rates for primary reconstructions are reported to be between 0.7% and 8%.[8] Results of graft revisions are much less predictable,[7] with failure rates of 5% to 52%.[8,19] Many authors have suggested that the main cause of graft failures is unrecognized, and therefore untreated, posterolateral rotatory instability.[9,13,21]

Markolf et al.[17] measured the force in a simulated intact ACL with and without the posterolateral knee structures and found increased force on the ACL with varus loading after cutting the posterolateral structures. The findings of that study may not be applicable to the ACL-reconstructed knee, because the reconstruction procedure alters the joint kinematics and load sharing between ligamentous structures (F. Wentorf, unpublished data, 1998).

The goal of this study was to determine if untreated grade III injuries of the posterolateral structures of the knee contribute to increased force on the graft after ACL

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reconstruction. This information would support the clinical suspicion that untreated grade III posterolateral structure injuries contribute to ACL graft failure.

MATERIALS AND METHODS

We studied eight fresh-frozen cadaveric knees with no evidence of previous surgeries, instability by clinical examination, or more than superficial chondromalacia at the time of dissection. The specimens were frozen at −20°C and allowed to thaw overnight before testing. The specimens were cut to within 15 cm proximal and distal to the joint line. Skin and subcutaneous tissues were then removed from the knees and the following structures were identified: iliotibial band and its components, fibular collateral ligament, fabellofibular ligament, components of the long and short head of the biceps femoris muscle, midthird lateral capsular ligament, popliteofibular ligament, popliteal aponeurotic attachments to the lateral meniscus, and the ligament of Wrisberg. Polymethyl methacrylate was used to pot the femur and tibia/fibula, which were each drilled separately with metal screws at their ends before potting to prevent any rotational slippage in the knee test machine.

The central third of the patellar tendon (10 mm), along with 10-mm wide by 25-mm long bone blocks off the patella and tibial tubercle, was harvested for the ACL graft. A small medial arthrotomy provided visualization of the tibial and femoral attachment sites of the ACL after resection of the fat pad. The anterior intermeniscal ligament was carefully preserved. The native ACL was then resected, and a reconstruction was performed with the femoral and tibial tunnels placed at the attachment sites of the native ACL. Arthrex ACL guides (Arthrex, Naples, Florida) were used to drill the tibial and femoral tunnels.

The femoral bone block was secured in the femoral tunnel with a 9 mm by 25 mm cannulated Arthrex interference screw. The tibial bone plug was secured to a 5-cm length of ligament augmentation device (LAD, 3M, St. Paul, Minnesota), which was looped at both ends with No. 5 nonabsorbable double-looped suture. The looped sutures were attached to a tensioning jig, with a screw mechanism to apply tension, that was rigidly secured to the proximal tibia with K-wires. The jig was centered over the tunnel such that the force was applied to the graft complex parallel to the tunnel. A buckle transducer was placed on the ligament augmentation device using techniques similar to those reported previously from this laboratory to measure the forces seen in the ACL graft (Fig. 1).[10, 15] The knee was then mounted in the testing machine and the ACL graft was secured to the tensioning jig after it had been cycled through a series of flexion/extension and anterior/posterior forces on the knee.

The baseline tension on the graft was determined by using a calibrated spring scale to apply a 67-N (15-pound) distal traction load to the ACL graft-ligament augmentation device complex with the knee in 0° of flexion and the graft fixed to the tibial jig. This duplicated the force on an ACL graft when fixation of the tibial bone block is performed during an in vivo ACL reconstruction. Once this load was set, it was allowed to counterbalance with the proximal tibia, as would occur during the fixation of the tibial bone block. The baseline calibration force on the graft was established as the force present on the graft when an anterior force of 67 N (15 pound) at 30° of knee flexion (Lachman test) was applied to the tibia. The force on the graft, as measured by the buckle transducer, was recalibrated via the screw tensioning device to this baseline force using this same technique (as necessary) after each testing cycle for each knee flexion angle to account for any graft or other tissue creep.

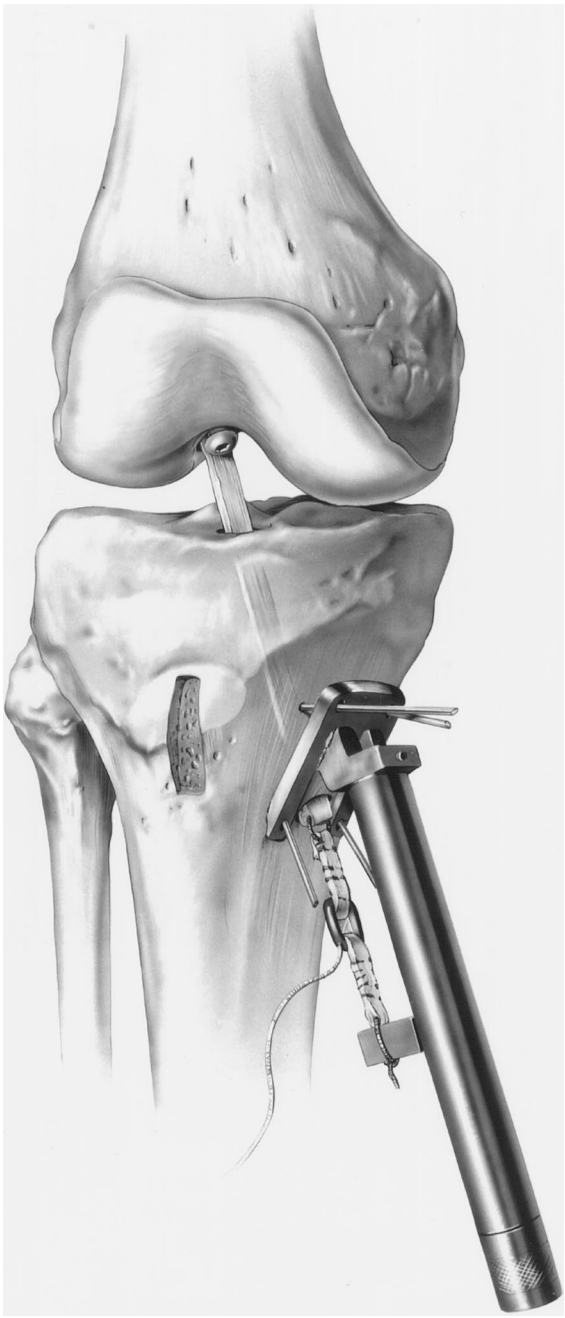

Figure 1. The force-measuring jig for loading and force measurement on the ACL graft during testing.

A pilot study was performed on four knees before the initiation of the study to determine the specific joint loads for this study. Loads tested in the pilot study but not included as part of this study because no graft loading was seen were posterior drawer at 0°, 30°, and 90°, and valgus force with coupled internal or external rotation at 0° and 30°. Although external rotation moments and coupled posterior drawer/external rotation moments did not increase the force on the ACL graft, they were included in this study because they are used clinically to assess abnormal motion at the posterolateral aspect of the knee.[3,4,6,29] In addition, the force and moment values chosen for loading these knees were based on the pilot data. We found that applied forces or moments higher than those performed during the pilot study resulted in graft failure (bone block fracture in three specimens and bone-tendon interface failure in one) for those motions that increased graft force because of the large increase in motion that occurred during the sequential cutting studies.

The following loads were applied to the knee testing machine[15]:

At full extension: 67-N anterior drawer force, 5-N·m internal and external tibial rotation moments, 12-N·m varus and valgus moments, and a 5-N·m internal rotation moment coupled with a 12-N·m varus moment.

At 30° of flexion: 67-N anterior drawer force, 5-N·m internal and external tibial rotation moments, 12-N·m varus and valgus moments, a 12-N·m varus force coupled with a 5-N·m internal rotation moment, and a 67-N posterior drawer force coupled with a 5-N·m external rotation moment.

At 90° of flexion: 67-N anterior drawer force and a 67-N posterior drawer force coupled with a 5-N·m external rotation moment.

The force on the ACL was recalibrated by the screw mechanism on the tibial tensioning jig back to the baseline graft force after each testing sequence for each angle of flexion. The entire testing sequence was performed a total of three times. An instrumented spatial linkage[10] was used to measure six degrees-of-freedom joint motion.

Joint loading and motion testing were performed on the knees after simulated ACL reconstruction in the normal state and after ACL reconstruction and sequential cutting of selected posterolateral structures. We chose the following as the most important posterolateral structures based on our pilot and prior studies[11–14,27,28]: 1) the fibular collateral ligament (near its attachment on the femur), 2) the popliteofibular ligament (near its attachment on the posteromedial fibular styloid), and 3) the popliteus tendon (at its origin on the femur). We did not investigate the effect of varying the cutting sequence.

Statistical analysis using the Student's *t*-test was performed for differences in the ACL graft force between the ACL-reconstructed knee and the ACL-reconstructed knee after each posterolateral structure was sectioned. Analysis was also performed for the differences in anterior/posterior translation, varus/valgus opening, and internal/external rotation between the normal and sequential cutting states for each motion analyzed. Statistical significance was assumed for P values less than 0.05.

RESULTS

Force on the ACL Graft

The force on the ACL graft was different with joint loading before and after the posterolateral structures were cut (Fig. 2). Figure 3 summarizes the increase (or decrease) in force seen on the ACL graft during joint loading after sequential sectioning of the fibular collateral ligament, popliteofibular ligament, and popliteus tendon compared with the baseline state of ACL reconstruction with the posterolateral structures intact.

After the fibular collateral ligament was sectioned there was a significant increase in force on the ACL graft during varus loading at both 0° ($P < 0.01$) and 30° ($P < 0.01$) of knee flexion. In addition, coupled loading of varus and internal rotation at 0° ($P < 0.02$) and 30° ($P < 0.001$) of flexion further increased graft force relative to varus loading alone for this testing state.

Additional sectioning of the popliteofibular ligament and popliteus tendon (after the fibular collateral ligament was cut) revealed similar increases in force on the ACL graft relative to the knee with posterolateral structures intact, with no significant changes occurring between each additional cutting state. The force on the ACL graft was significantly increased during varus loading at 0° after the fibular collateral and popliteofibular ligaments were sectioned ($P < 0.05$) and after the popliteus tendon was also sectioned ($P < 0.02$). It was increased at 30° of knee flexion after the fibular collateral and popliteofibular ligaments were sectioned ($P < 0.04$) and after the popliteus tendon was also sectioned ($P < 0.01$). In addition, a significant increase in ACL graft force was seen after the posterolateral structures were sectioned when coupled loading of varus and internal rotation was performed at both 0° and 30° of knee flexion.

Changes in Joint Motion

Figures 4 through 6 show a summary of the changes in varus/valgus opening, anterior/posterior displacement, and external rotation/internal rotation after sequential sectioning of the posterolateral structures in the ACL-reconstructed knee. Significant changes in joint motion are noted on the graphs. We reemphasize that the graft was reset to the original setting force, simulating the ACL-reconstructed knee, after each cutting sequence.

DISCUSSION

The joint loads that resulted in a significant increase in force on the ACL graft after cutting of the posterolateral structures were varus moments and coupled varus/internal rotation moments. An internal rotation moment by itself did not result in a significant increase in graft force, and it can be extrapolated that the main load that increased the force on the ACL graft was a varus moment. The loads applied in our study were chosen for convenience of the test setup. To relate these forces to those occurring in vivo, an estimate of these forces can be ob-

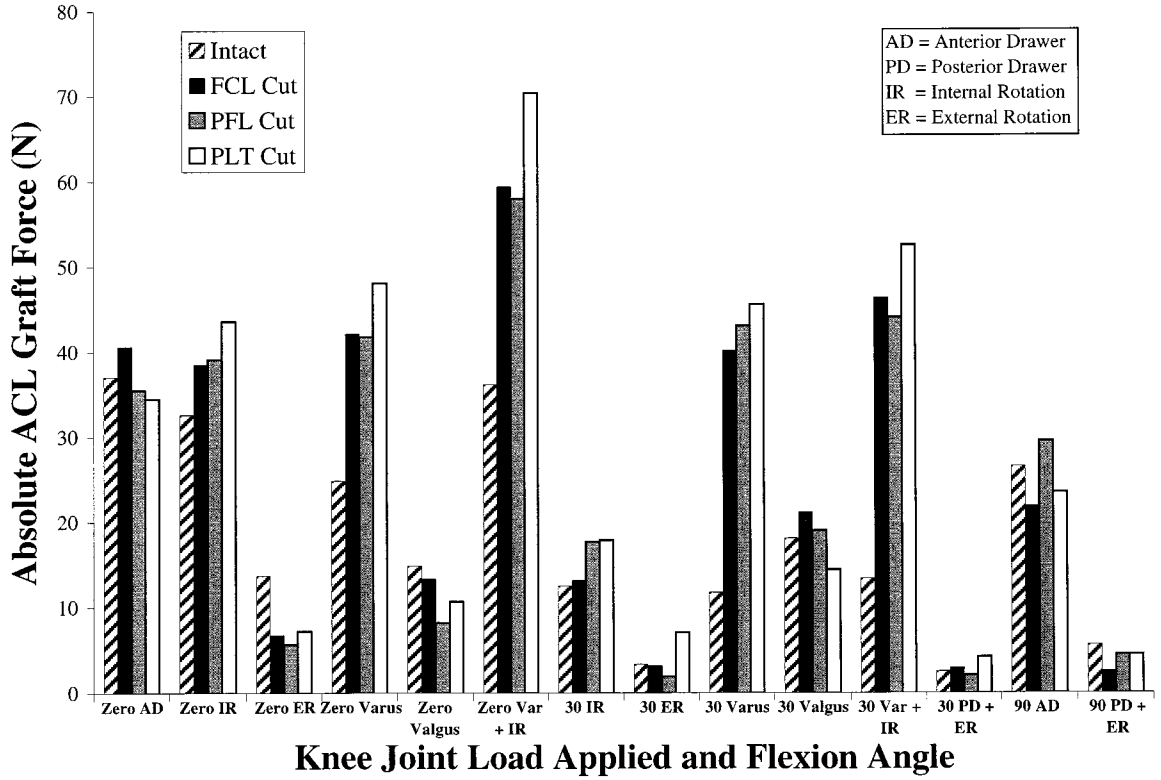

Figure 2. Absolute ACL reconstruction graft force for applied loads with the posterolateral structures intact and with sequential cutting of the fibular collateral ligament (FCL), popliteofibular ligament (PFL), and popliteus tendon (PLT).

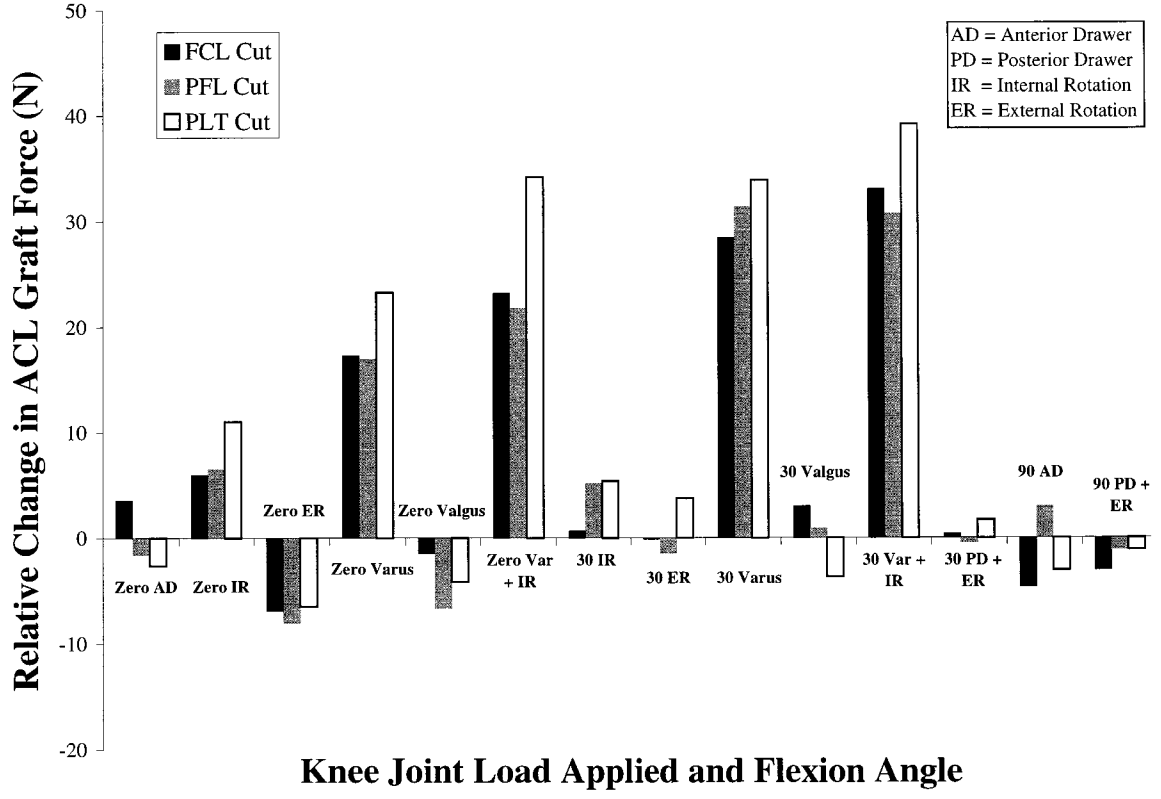

Figure 3. Change in ACL graft force relative to the same load applied to the joint with the posterolateral structures intact and after cutting the fibular collateral ligament (FCL), popliteofibular ligament (PFL), and popliteus tendon (PLT).

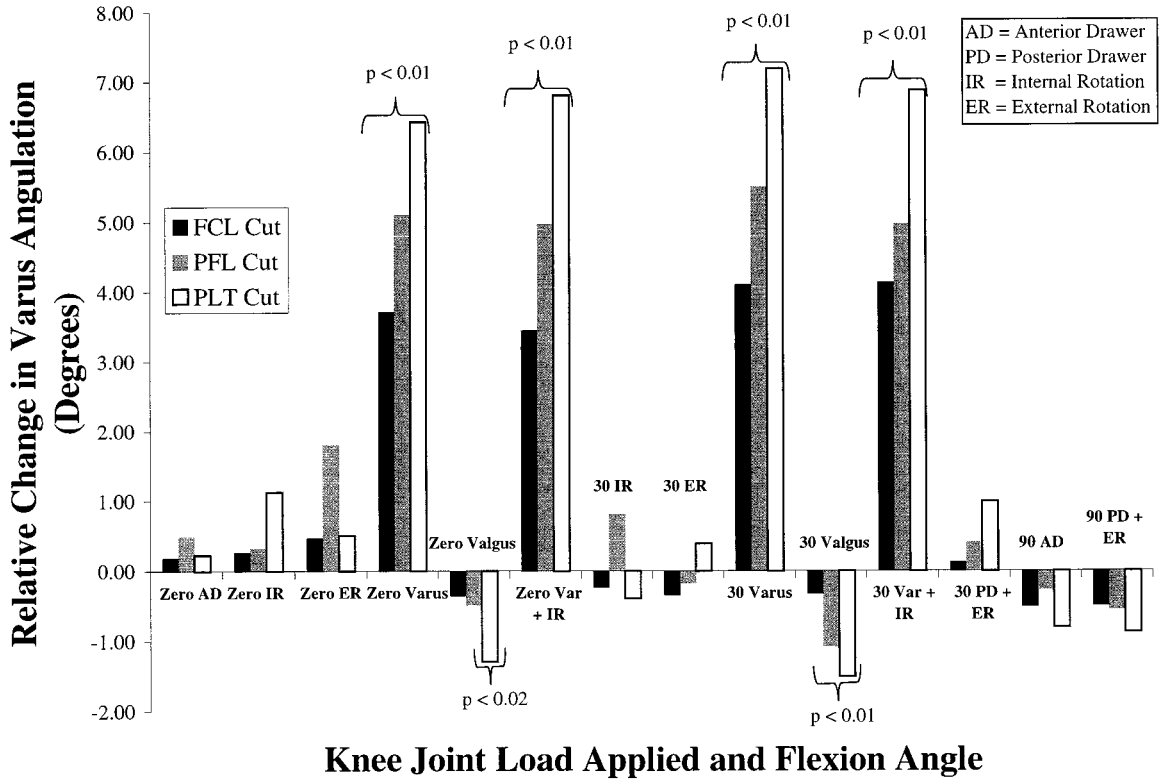

Figure 4. Varus/valgus rotation changes in knee configuration in the ACL-reconstructed knee after sequential sectioning of the fibular collateral ligament (FCL), popliteofibular ligament (PFL), and popliteus tendon (PLT) relative to configuration with the same load on the joint with the posterolateral structures intact.

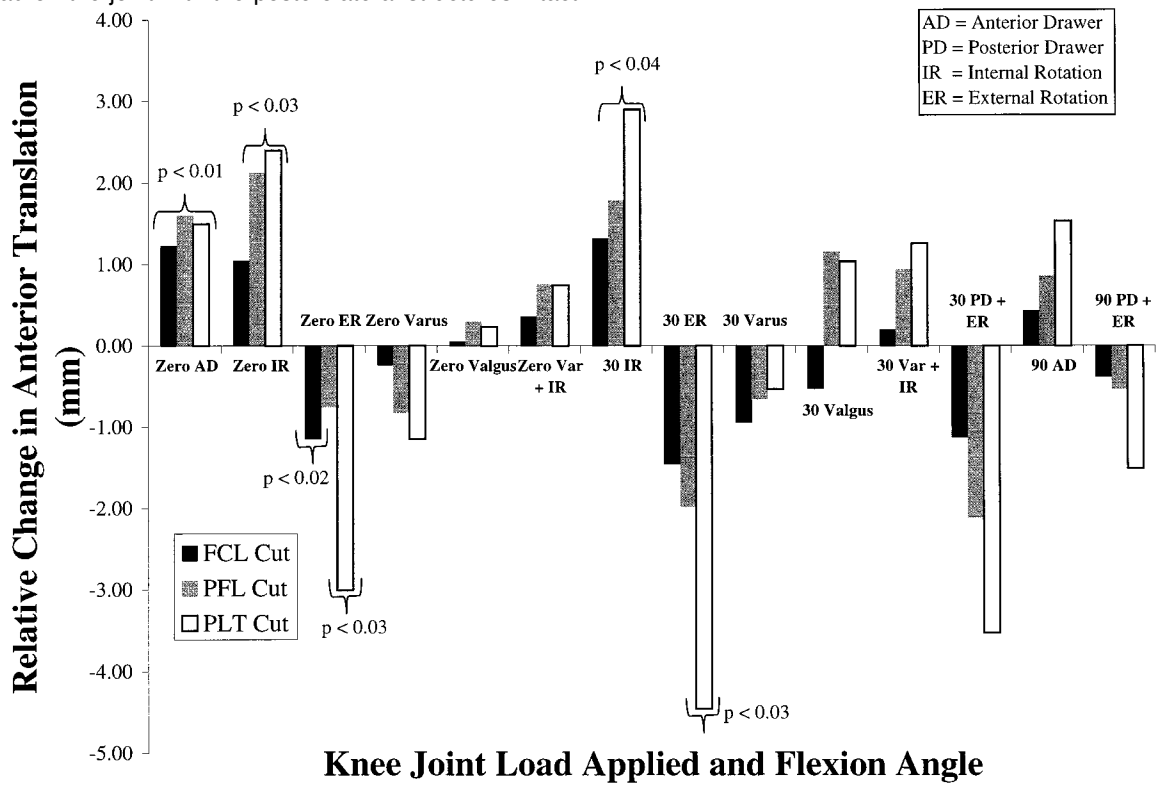

Figure 5. Anterior/posterior translation changes in knee configuration. FCL, fibular collateral ligament; PFL, popliteofibular ligament; PLT, popliteus tendon.

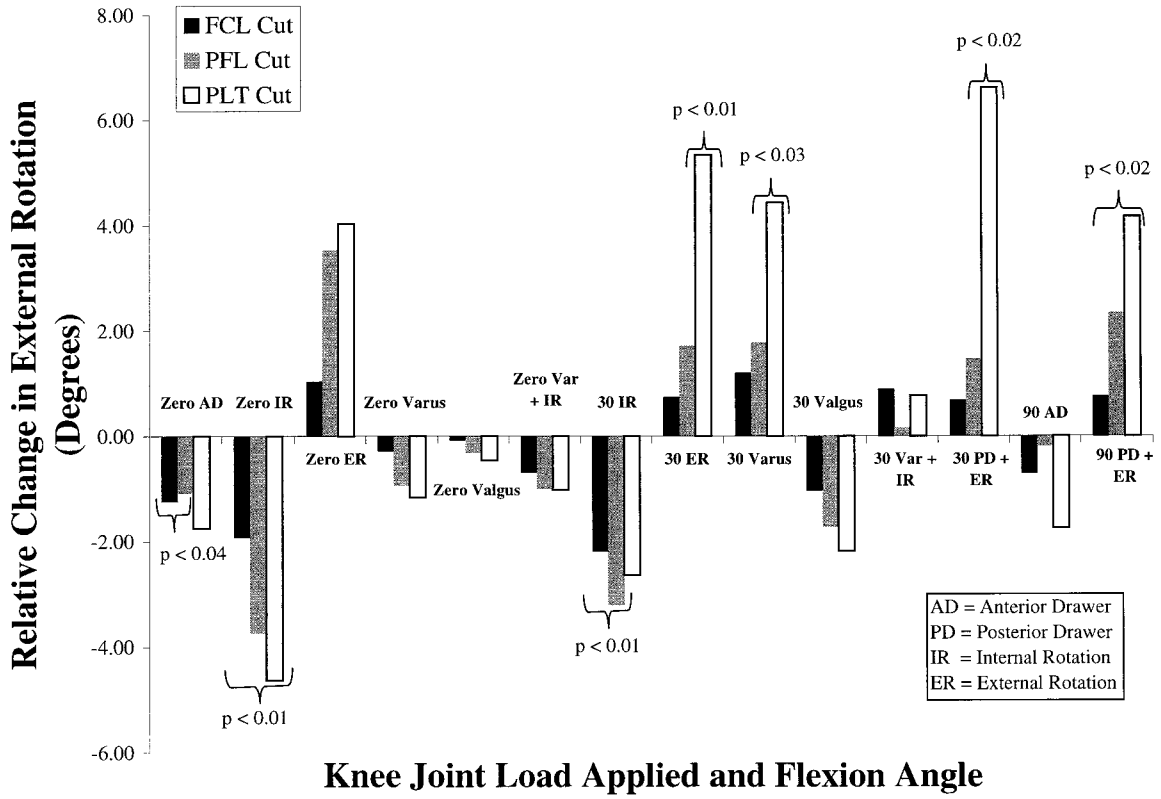

Figure 6. Internal/external rotation changes in knee configuration. FCL, fibular collateral ligament; PFL, popliteofibular ligament; PLT, popliteus tendon.

tained from Prodromos et al.,[22] who measured joint moments in patients before and after high tibial osteotomy. These authors presented adduction (varus) joint moment in terms of body weight (BW) times height (HT). They arbitrarily assigned a value of 4% for normal. The highest adduction moment in their study was 6% BW × HT.

To compare the forces measured by Prodromos et al. with the forces in our study, their values must be converted to newton-meters. A typical body weight is chosen as 155 pounds, or 690 N. A typical height is chosen as 5 feet 10 inches, or 1.8 meters. The normal value of 4% BW × HT then becomes 50 N·m, and 6% BW × HT becomes 75 N·m. In a worst-case scenario, all of this moment would be applied to the passive structures of the knee. For the highest graft force measured in our study with an applied coupled varus and internal rotation moment of 12 N·m at 0° of flexion (71 N) (Fig. 2), if the graft force increased proportionately in relation to the joint varus moment, converting the adduction moment of 50 N·m would mean 50 N·m divided by 12 N·m times 71 N, which would equal 296 N of force on the graft. Seventy-five newton-meters (6% BW × HT adduction moment) would cause 444 N of force on the graft. Rowden et al.,[23] reported that the failure strength of a patellar tendon ACL graft was 416 N immediately after reconstruction. Because animal studies have shown that ACL grafts weaken with time before gaining strength with remodeling,[2,18] the forces that may occur for the patient with a high adduction moment gait (6% BW × HT), such as the varus-thrust gait, could stretch or rupture the graft.

Noyes et al.[20] have argued that the high adduction moment associated with a varus-thrust gait increases lateral soft tissue forces and that this high adduction moment could place an ACL graft at risk. Other studies have demonstrated that the ACL acts as a secondary restraint to varus angulation.[16,17,26] Our study demonstrated that with varus loading, absence of the posterolateral structures did, in fact, significantly increase the load on the ACL graft, which places it at an increased risk for failure. We recommend that the posterolateral structures be repaired or reconstructed at the time of ACL reconstruction in these patients. A complete assessment of the source of the varus moment should also be performed before ACL reconstruction. It is important to determine whether the increase in varus force is solely due to a grade III posterolateral structure injury or is combined with a varus-aligned knee. We recognize that in some knees a proximal tibial osteotomy may also be needed to address increased varus joint line opening in those patients with a mechanical axis that passes medial to the central axis of the knee because of medial compartment arthritis or in patients with chronic injuries who have a congenital tendency to genu varus.

Although posterolateral rotatory instability has been demonstrated to cause an increase in both varus and external rotation forces in the knee,[3–6,11,14,29] an external

rotation force in the ACL-reconstructed knee with sectioned posterolateral structures resulted in an unloading of the ACL grafts in this study. An increase in external rotation force has been demonstrated to cause clinical disability and loss of function in patients,[5,6,13,14,21] but it does not appear to be the main cause of an increase in abnormal loading of the ACL graft in patients with grade III posterolateral rotatory instability. We recognize that our study is a static study and that coupled muscle forces and increases in external rotation force could contribute to ACL graft failure clinically through loading forces that we could not measure in the in vitro state.

Analysis of motion differences for knees with ACL reconstructions only and ACL reconstructions with sequential sectioning of the posterolateral structures revealed significant increases in varus opening for an applied varus moment after each structure was sectioned. This finding indicated that all three of the sectioned structures (fibular collateral ligament, popliteofibular ligament, and popliteus tendon) contribute to prevention of increased varus joint line opening with the ACL graft present (Fig. 4). Our data also show that abnormal joint laxity can result even with an appropriately performed ACL reconstruction if other structures, in this case the posterolateral structures, are not repaired or reconstructed as well. We used only one sequential cutting sequence for this study (based on our pilot study data), and variation in cutting sequences may yield further information on the importance of each anatomic structure in preventing increases in varus opening and the resultant increase in force seen in the ACL graft. It appears from our data that the main structure preventing increased force on the ACL graft is the fibular collateral ligament. Further studies are planned with variations in the sequential sectioning to determine whether significant increases in force on the ACL graft are seen with isolated popliteus complex injuries.

CONCLUSIONS

The force in an ACL graft increased during varus and coupled varus/internal rotation moments in knees with the posterolateral structures sectioned compared with the force in the same knee with the ACL reconstruction and the posterolateral structures intact. We recommend that strong consideration be given to repairing or reconstructing ruptured posterolateral structures, especially in knees with evidence of varus instability at the time of ACL reconstruction. This should decrease the chance of ACL graft failure.

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REFERENCES

1. American Medical Association: *Standard Nomenclature of Athletic Injuries.* Chicago, IL, American Medical Association, 1968
2. Butler DL, Grood ES, Noyes FR, et al: Mechanical properties of primate vascularized vs. nonvascularized patellar tendon grafts; changes over time. *J Orthop Res 7:* 68–79, 1989
3. Gollehon DL, Torzilli PA, Warren RF: The role of the posterolateral and cruciate ligaments in the stability of the human knee. A biomechanical study. *J Bone Joint Surg 69A:* 233–242, 1987
4. Grood ES, Stowers SF, Noyes FR: Limits of movement in the human knee. Effect of sectioning the posterior cruciate ligament and posterolateral structures. *J Bone Joint Surg 70A:* 88–97, 1988
5. Hughston JC, Jacobson KE: Chronic posterolateral rotatory instability of the knee. *J Bone Joint Surg 67A:* 351–359, 1985
6. Hughston JC, Norwood LA Jr: The posterolateral drawer test and external rotational recurvatum test for posterolateral rotatory instability of the knee. *Clin Orthop 147:* 82–87, 1980
7. Jaureguito JW, Paulos PE: Why grafts fail. *Clin Orthop 325:* 25–41, 1996
8. Johnson DL, Harner CD, Maday MG, et al: Revision anterior cruciate ligament surgery, in Fu F, Harner CD, Vince KG (eds): *Knee Surgery.* Baltimore, Williams & Wilkins, 1994, pp 877–895
9. Kannus P: Nonoperative treatment of Grade II and III sprains of the lateral ligament compartment of the knee. *Am J Sports Med 17:* 83–88, 1989
10. Kirstukas SJ: A model for studying loss of knee extension. PhD Thesis. University of Minnesota, Minneapolis, Minnesota, 1995
11. LaPrade RF: Arthroscopic evaluation of the lateral compartment of knees with grade 3 posterolateral knee complex injuries. *Am J Sports Med 25:* 596–602, 1997
12. LaPrade RF, Hamilton CD: The fibular collateral ligament-biceps femoris bursa. An anatomic study. *Am J Sports Med 25:* 439–443, 1997
13. LaPrade RF, Hamilton CD, Engebretsen L: Treatment of acute and chronic combined anterior cruciate ligament and posterolateral knee ligament injuries. *Sports Med Arthrosc Rev 5:* 91–99, 1997
14. LaPrade RF, Terry GC: Injuries to the posterolateral aspect of the knee: Association of anatomic injury patterns with clinical instability. *Am J Sports Med 25:* 433–438, 1997
15. Lewis JL, Lew WD, Hill JA, et al: Knee joint motion and ligament forces before and after ACL reconstruction. *J Biomech Eng 111:* 97–106, 1989
16. Markolf KL, Burchfield DM, Shapiro MM, et al: Biomechanical consequences of replacement of the anterior cruciate ligament with a patellar ligament allograft. Part I: Insertion of the graft and anterior-posterior testing. *J Bone Joint Surg 78A:* 1720–1727, 1996
17. Markolf KL, Burchfield DM, Shapiro MM, et al: Biomechanical consequences of replacement of the anterior cruciate ligament with a patellar ligament allograft. Part II: Forces in the graft compared with forces in the intact ligament. *J Bone Joint Surg 78A:* 1728–1734, 1996
18. Ng GY, Oakes BW, Deacon OW, et al: Biomechanics of patellar tendon autograft for reconstruction of the anterior cruciate ligament in the goat: Three-year study. *J Orthop Res 13:* 602–608, 1995
19. Noyes FR, Barber-Westin SD: Revision anterior cruciate ligament surgery: Experience from Cincinnati. *Clin Orthop 325:* 116–129, 1996
20. Noyes FR, Dunworth LA, Andriacchi TP, et al: Knee hyperextension gait abnormalities in unstable knees. Recognition and preoperative gait retraining. *Am J Sports Med 24:* 35–45, 1996
21. O'Brien SJ, Warren RF, Pavlov H, et al: Reconstruction of the chronically insufficient anterior cruciate ligament with the central third of the patellar ligament. *J Bone Joint Surg 73A:* 278–286, 1991
22. Prodromos CC, Andriacchi TP, Galante JO: A relationship between gait and clinical changes following high tibial osteotomy. *J Bone Joint Surg 67A:* 1188–1194, 1985
23. Rowden NJ, Sher D, Rogers GJ, et al: Anterior cruciate ligament graft fixation. Initial comparison of patellar tendon and semitendinosus autografts in young fresh cadavers. *Am J Sports Med 25:* 472–478, 1997
24. Seebacher JR, Inglis AE, Marshall JL, et al: The structure of the posterolateral aspect of the knee. *J Bone Joint Surg 64A:* 536–541, 1982
25. Stäubli HU, Birrer S: The popliteus tendon and its fascicles at the popliteal hiatus: Gross anatomy and functional arthroscopic evaluation with and without anterior cruciate ligament deficiency. *Arthroscopy 6:* 209–220, 1990
26. Takeda Y, Xerogeanes JW, Livesay GA, et al: Biomechanical function of the human anterior cruciate ligament. *Arthroscopy 10:* 140–147, 1994
27. Terry GC, LaPrade RF: The posterolateral aspect of the knee: Anatomy and surgical approach. *Am J Sports Med 24:* 732–739, 1996
28. Terry GC, LaPrade RF: The biceps femoris muscle complex at the knee: Its anatomy and injury patterns associated with acute anterolateral-anteromedial rotatory instability. *Am J Sports Med 24:* 2–8, 1996
29. Veltri DM, Warren RF: Anatomy, biomechanics, and physical findings in posterolateral knee instability. *Clin Sports Med 13:* 599–614, 1994