Published in final edited form as:
Oper Tech Sports Med. 2008 July 1; 16(3): 116–118. doi:10.1053/j.otsm.2008.10.005.

The Kinematic Basis of ACL Reconstruction

Scott Tashman, PhD [Associate Professor, Director of Biodynamics Laboratory], **Sebastian Kopf, MD [Post-Doctoral Research Fellow]**, and **Freddie H Fu, MD [Chairman of the Department of Orthopaedic Surgery, David Silver Professor]**
From Department of Orthopaedic Surgery, University of Pittsburgh, 3471 Fifth Avenue, Pittsburgh, PA 15213, USA

Abstract

The goals of anterior cruciate ligament (ACL) reconstruction are to restore knee stability and function and to preserve joint health. Static tests for antero-posterior laxity (e.g. Lachman test or KT-1000 arthrometer) have typically shown restoration of normal or near-normal laxity with a variety of modern ACL reconstruction techniques. However, ACL reconstruction has failed to prevent early onset of osteoarthritis, and there is growing evidence that traditional single-bundle ACL reconstruction does not restore normal knee mechanics under functional loading conditions. ACL reconstruction may fail to restore normal rotational stability during the pivot shift. Abnormal internal-external rotation and ab/adduction have been reported after ACL reconstruction during normal daily activities like walking and running. Recently, cadaveric studies have shown the potential superiority of ACL double bundle (DB) reconstruction for restoring anatomy and mechanical function. However, clinical data demonstrating the clear superiority of DB reconstruction is lacking, due to the absence of well-controlled clinical studies. Additionally, dynamic knee function after anatomic DB ACL has yet to be assessed comprehensively.

Text

Normal tibio-femoral motion is constrained by articular surfaces, ligaments, capsule and menisci (1). Damage to ligaments or menisci may alter these constraints, permitting abnormal motion that alters cartilage loading patterns and increases risk for osteoarthritis (OA) (2–5). Thus, the goal for anterior cruciate ligament (ACL) reconstruction should be the restoration of normal knee anatomy and mechanics, to return the joint to normal function, reestablish mechanical/biological homeostasis and prevent OA. The most common surgical approach for ACL reconstruction has been a single graft bundle, with the femoral tunnel drilled through the tibial tunnel (trans-tibial). Though this technique has been generally perceived to be successful, several recent meta-analyses have indicated that normal structure and function of the knee is restored only 60% to 70% of the time (6,7). Perhaps of greater concern, 60 to 90% of individuals have radiographic evidence of knee OA within 10 to 20 years after ACL reconstruction (8–14). These and other similar findings have reinvigorated interest in improving our understanding of the anatomy and function of the ACL, and driven investigations into alternative techniques for reconstruction that might better replicate function of the native ACL and improve long-term outcomes.

Corresponding author: Scott Tashman, Department of Orthopaedic Surgery – Biodynamics Laboratory, 3820 South Water Street, Pittsburgh, PA, 15203, E-mail: E-mail: Tashman@pitt.edu, Telephone: 001-412-641-0602, Fax: 001-412-586-3979.

No potential conflict of interest declared.

Much of the current knowledge concerning ACL anatomy and function has been derived from cadaveric studies evaluating normal, ACL-deficient and ACL-reconstructed knees under controlled, laboratory conditions. Linear and rotational transducers have been employed to subject cadaver knees to a variety of dynamically changing flexion and extension torques and tendon forces (15). In full knee extension both bundles are arranged parallel, and in 90° of knee flexion they are twisted. Both bundles resist anterior translation of the tibia relative to the femur, with the anteromedial (AM) bundle more taut from 30° to 90°, and the posterolateral (PL) bundle more tensioned from 0° to 30° of knee flexion. Furthermore, cadaveric studies have shown that the PL bundle plays a more important role for providing rotational stability in comparison to the AM bundle (16,17). Failure of conventional ACL reconstruction to restore knee kinematics has also been demonstrated for cadaveric knees (18). However, it has been difficult to predict clinical outcome based on the results of cadaver studies, which cannot replicate functional loading and represent only the “time-zero” condition (immediately after graft fixation).

In vivo functional assessment after ACL reconstruction is essential for evaluating effectiveness of treatment for restoring dynamic knee function and relating function to long-term outcome. Traditionally, static knee stability tests have been the standard for evaluating graft function after ACL reconstruction. Typically, the thigh is stabilized with the knee held at a fixed flexion angle, and an anterior force is applied to the calf. Most common are the Lachman test and the KT-1000 arthrometer (Med-Metric, San Diego, CA), which involve applying an anterior load and assessing the resulting anterior displacement of the tibia relative to the femur. However, static stability measures are not well correlated with measures of functional outcome for ACL-injured or reconstructed subjects (19–22).

Another functional outcome test commonly performed in clinic is the Pivot shift, which is supposed to evaluate the rotationally stability of the knee (23,24). However, this test is very subjective and observer dependent, and does not simulate a physiological knee activity like walking or running (25–27).

The limited value of these tests for predicting clinical function is likely related to the difference between measures of *laxity* vs. measures of *dynamic stability*. Laxity tests (translational or rotational) measure the maximum displacement of the joint in response to an applied external load, in the absence of muscle forces. These tests attempt to define the limits of the possible envelope of motion for the knee. However, the knee rarely operates at these limits during normal movement. It typically stays within a more limited envelope of function that varies with activity and is controlled by a combination of the passive knee structures and dynamic neuromuscular control (28). Simple laxity tests cannot simulate the complex force magnitudes, directions and rates of application produced at the knee during most human movement (29, 30).

Neuromuscular factors are important contributors to knee function in the ACL injured/reconstructed knee. Muscle forces are known to play a significant role in knee stability, especially in the ACL-deficient and reconstructed individual (31). When the ACL is injured and then reconstructed, the passive structures may be restored but motor control deficits likely remain. Sensory structures have been identified in the intact ACL, which could provide force and length proprioceptive information to the central nervous system (32). Changes in reflex response observed after ACL injury suggest that knee joint control may be adversely affected by ACL loss (33,34). ACL reconstruction technique may influence graft healing and joint proprioception, leading to effects beyond those of passive mechanics alone.

Only in vivo, human studies of dynamic joint function and stability can assess the combined effects of surgical methods, tissue healing/remodeling and neuromuscular control on joint

NIH-PA Author Manuscript

NIH-PA Author Manuscript

function. Even small instabilities can lead to repetitive microtrauma and long-term joint degeneration, if they occur with high frequency during common activities. Thus, measurement of dynamic knee motion during functional tasks, and understanding how it is altered by ACL injury/reconstruction, are fundamental prerequisites for optimizing treatment.

Most data currently available on dynamic knee function was collected using gait analysis techniques employing optoelectronic or video-based systems to track external markers attached to the skin. These techniques have been used to investigate the effects of ACL deficiency and ACL reconstruction on knee function during various movement activities, including walking, jogging, and ascending/descending stairs. ACL deficient knees showed increased internal tibial rotation relative to uninjured knees (35). However, no kinematic differences were found during gait between SB ACL reconstructed and uninjured knees for flexion-extension, abduction-adduction, and internal-external rotation (36,37). With more demanding functional tests, such as pivoting after stair descending and jumping, ACL reconstruction restored antero-posterior stability but not rotational stability (38). The maximum range of tibial rotation was reported to be similar between ACL deficient and reconstructed knees (39).

There is some concern that studies using skin markers may miss subtle but important kinematic abnormalities after ACL reconstruction. Depending on skin and underlying tissue, the movement of these markers can be up to 30 mm relative to underlying bone, especially during high impact activities (40). Radiographic methods such as Roentgen stereophotogrammetric analysis (RSA) enable direct visualization of bone. Dynamic RSA (D-RSA), combining small implanted bone markers with biplanecine radiography, can achieve accuracy of ±0.1 mm or better at high sample rates (250 frames/s) (41). Thus, D-RSA can acquire precise, real-time kinematic data during daily activities like running, incline and decline walking, and stair climbing. A study utilizing D-RSA to evaluate patients after ACL reconstruction found that the reconstructed knees were significantly more externally rotated (3.8 ± 2.3°) and adducted (2.8 ± 1.6) than the contralateral, uninjured joints. Anterior tibial translation did not differ significantly between the reconstructed and the healthy, contralateral knee (41), but did increase over time after surgery (+0.85 mm from five to twelve months) (42). Even these are appear to be small changes; their effect over time may be responsible for the early onset of OA (5).

The studies described above all utilized ACL reconstruction techniques representative of the “standard of care” for the last 10–15 years (single graft bundle, typically trans-tibial drilling of femoral tunnel). The cadaver and in vivo studies described above (and other similar studies) have highlighted limitations of this approach for restoring normal knee anatomy and function, and lead to a surge of interest in anatomical ACL reconstructions that attempt to better reproduce the two-bundle anatomy and its insertion sites. It has been proposed that a more anatomical ACL reconstruction will more effectively restore native ACL kinematics and dynamic knee function. Cadaveric and in vivo MRI studies evaluating knee kinematics after double bundle (DB) ACL reconstruction showed superior results compared to SB ACL reconstruction (43,44). These results were confirmed by a recent meta-analysis, which showed that even in laxity tests like KT-1000 and pivot shift, DB ACL reconstruction achieved superior results in comparison to SB ACL reconstruction (45,46). However, these studies are insufficient to evaluate the efficacy of anatomical ACL reconstruction for restoring normal knee mechanics and preserving long-term joint health. Future studies should include accurate assessment of dynamic knee function, as well as careful assessment of clinical and functional outcomes.

Literature

1. O’Connor JJ, Shercliff TL, Biden E, Goodfellow JW. Proc Inst Mech Eng [H] 1989;203:223–33.

2. Brandt KD, Braunstein EM, Visco DM, O'Connor B, et al. J Rheumatol 1991;18:436–46. [PubMed: 1856812]
3. O'Connor BL, Palmoski MJ, Brandt KD. J Bone Joint Surg Am 1985;67:562–72. [PubMed: 3980502]
4. Pond MJ, Nuki G. Ann Rheum Dis 1973;32:387–8. [PubMed: 4726075]
5. Chaudhari AM, Briant PL, Bevill SL, Koo S, Andriacchi TP. Med Sci Sports Exerc 2008;40:215–22. [PubMed: 18202582]
6. Biau DJ, Tournoux C, Katsahian S, Schranz PJ, Nizard RS. BMJ 2006;332:995–1001. [PubMed: 16603564]
7. Biau DJ, Tournoux C, Katsahian S, Schranz P, Nizard R. Clin Orthop Relat Res 2007;458:180–7. [PubMed: 17308473]
8. Sommerlath K, Lysholm J, Gillquist J. Am J Sports Med 1991;19:156–62. [PubMed: 2039067]
9. Neyret P, Donell ST, Dejour H. J Bone Joint Surg Br 1993;75:36–40. [PubMed: 8421030]
10. Noyes FR, Mooar PA, Matthews DS, Butler DL. J Bone Joint Surg Am 1983;65:154–62. [PubMed: 6687391]
11. McDaniel WJ, Dameron TB. Clin Orthop Relat Res 1983;158–63
12. Kannus P, Järvinen M. Am J Sports Med 1987;15:138–43. [PubMed: 3578635]
13. Lohmander LS, Ostenberg A, Englund M, Roos H. Arthritis Rheum 2004;50:3145–52. [PubMed: 15476248]
14. Fithian DC, Paxton EW, Stone ML, Luetzow WF, et al. Am J Sports Med 2005;33:335–46. [PubMed: 15716249]
15. Reuben JD, Rovick JS, Schrager RJ, Walker PS, Boland AL. Am J Sports Med 1989;17:463–71. [PubMed: 2782529]
16. Gabriel MT, Wong EK, Woo SL, Yagi M, Debski RE. J Orthop Res 2004;22:85–9. [PubMed: 14656664]
17. Sakane M, Fox RJ, Woo SL, Livesay GA, et al. J Orthop Res 1997;15:285–93. [PubMed: 9167633]
18. Loh JC, Fukuda Y, Tsuda E, Steadman RJ, et al. Arthroscopy 2003;19:297–304. [PubMed: 12627155]
19. Barber SD, Noyes FR, Mangine RE, McCloskey JW, Hartman W. Clin Orthop Relat Res 1990:204–14. [PubMed: 2347154]
20. Harter RA, Osternig LR, Singer KM, James SL, et al. Am J Sports Med 1988;16:434–43. [PubMed: 3189674]
21. Seto JL, Orofino AS, Morrissey MC, Medeiros JM, Mason WJ. Am J Sports Med 1988;16:170–80. [PubMed: 3377102]
22. Snyder-Mackler L, Fitzgerald GK, Bartolozzi AR, Ciccotti MG. Am J Sports Med 1997;25:191–5. [PubMed: 9079172]
23. Pinczewski LA, Salmon LJ, Jackson WF, von Bormann RB, et al. J Bone Joint Surg Br 2008;90:172–9. [PubMed: 18256083]
24. Beynnon BD, Johnson RJ, Fleming BC, Kannus P, et al. J Bone Joint Surg Am 2002;84-A:1503–13. [PubMed: 12208905]
25. Lane CG, Warren RF, Stanford FC, Kendoff D, Pearle AD. Knee Surg Sports Traumatol Arthrosc. 2008
26. Noyes FR, Grood ES, Cummings JF, Wroble RR. The American Journal of Sports Medicine 1991;19:148. [PubMed: 2039066]
27. Benjaminse A, Gokeler A, van der Schans CP. J Orthop Sports Phys Ther 2006;36:267–88. [PubMed: 16715828]
28. Dyrby CO, Andriacchi TP. J Orthop Res 2004;22:794–800. [PubMed: 15183436]
29. Markolf KL, Bargar WL, Shoemaker SC, Amstutz HC. J Bone Joint Surg Am 1981;63:570–85. [PubMed: 7217123]
30. Schipplein OD, Andriacchi TP. J Orthop Res 1991;9:113–9. [PubMed: 1984041]
31. Ingersoll CD, Grindstaff TL, Pietrosimone BG, Hart JM. Clin Sports Med 2008;27:383–404. vii. [PubMed: 18503874]
32. Schultz RA, Miller DC, Kerr CS, Micheli L. J Bone Joint Surg Am 1984;66:1072–6. [PubMed: 6207177]

33. Jennings AG, Seedhom BB. J Bone Joint Surg Br 1994;76:491–4. [PubMed: 8175863]
34. Solomonow M, Baratta R, Zhou BH, Shoji H, et al. Am J Sports Med 1987;15:207–13. [PubMed: 3618871]
35. Georgoulis AD, Papadonikolakis A, Papageorgiou CD, Mitsou A, Stergiou N. Am J Sports Med 2003;31:75–9. [PubMed: 12531761]
36. Berchuck M, Andriacchi TP, Bach BR, Reider B. J Bone Joint Surg Am 1990;72:871–7. [PubMed: 2365720]
37. Noyes FR, Schipplein OD, Andriacchi TP, Saddemi SR, Weise M. Am J Sports Med 1992;20:707–16. [PubMed: 1456365]
38. Chouliaras V, Ristanis S, Moraiti C, Stergiou N, Georgoulis AD. Am J Sports Med 2007;35:189–96. [PubMed: 17251174]
39. Ristanis S, Stergiou N, Patras K, Tsepis E, et al. Clin J Sport Med 2006;16:111–6. [PubMed: 16603879]
40. Reinschmidt C, van den Bogert AJ, Nigg BM, Lundberg A, Murphy N. J Biomech 1997;30:729–32. [PubMed: 9239553]
41. Tashman S, Collon D, Anderson K, Kolowich P, Anderst W. Am J Sports Med 2004;32:975–83. [PubMed: 15150046]
42. Tashman S, Kolowich P, Collon D, Anderson K, Anderst W. Clin Orthop Relat Res 2007;454:66–73. [PubMed: 17091011]
43. Seon JK, Song EK, Bae BH, Park SJ, et al. Int Orthop 2007;31:623–8. [PubMed: 17061128]
44. Yagi M, Wong EK, Kanamori A, Debski RE, et al. Am J Sports Med 2002;30:660–6. [PubMed: 12238998]
45. Meredick RB, Vance KJ, Appleby D, Lubowitz JH. Am J Sports Med. 2008
46. Irrgang JI, FU FHF. Am J Sports Med. 2008