Altered Knee Kinematics in ACL-Deficient Non-Copers: A Comparison Using Dynamic MRI

Peter J. Barrance,[1] Glenn N. Williams,[1] Lynn Snyder-Mackler,[1,2] Thomas S. Buchanan[1]

[1]Center for Biomedical Engineering Research, Department of Mechanical Engineering, 126 Spencer Laboratory, University of Delaware, Newark, Delaware 19716

[2]Department of Physical Therapy, University of Delaware, Newark, Delaware 19716

ABSTRACT: Kinematics measured during a short arc quadriceps knee extension exercise were compared in the knees of functionally unstable ACL-deficient patients, these patients' uninjured knees, and uninjured control subjects' knees. Cine phase contrast dynamic magnetic resonance imaging, in combination with a model-based tracking algorithm developed by the authors, was used to measure tibiofemoral kinematics as the subjects performed the active, supine posture knee extension exercise in the terminal 30 degrees of motion. Two determinants of tibiofemoral motion were measured: anterior/posterior location of the tibia relative to the femur, and axial rotation of the tibia relative to the femur. We hypothesized that more anterior tibial positioning, as well as differences in axial tibial rotation patterns, would be observed in ACL-deficient (ACL-D) knees when compared to uninjured knees. Multifactor ANOVA analyses were used to determine the dependence of the kinematic variables on (i) side (injured vs. uninjured, matched by subject in the control group), (ii) flexion angle measured at five-degree increments, and (iii) subject group (ACL-injured vs. control). Statistically significant anterior translation and external tibial rotation (screw home motion) accompanying knee extension were found. The ACL-D knees of the injured group exhibited significantly more anterior tibial positioning than the uninjured knees of these subjects (average difference over extension range $= 3.4 \pm 2.8$ mm, $p < 0.01$ at all angles compared), as well as the matched knees of the control subjects. There was a significant effect of interaction between side and subject group on A/P tibial position. We did not find significant differences in external tibial rotation associated with ACL deficiency. The changes to active joint kinematics documented in this entirely noninvasive study may contribute to cartilage degradation in ACL-D knees, and encourage more extensive investigations using similar methodology in the future. © 2005 Orthopaedic Research Society. Published by Wiley Periodicals, Inc. J Orthop Res 24:132–140, 2006

Keywords: knee; kinematics; ACL; cine phase contrast; magnetic resonance

INTRODUCTION

Anterior cruciate ligament (ACL) injury has been associated with progressive development of knee osteoarthritis,[1,2] and alteration to kinematics causing aberrant joint loading has been put forth as a possible contributor to the disease's prog-

ression.[3] The ACL primarily functions to limit anterior translation of the tibia relative to the femur,[4–6] and also plays a role in governing the coupled tibiofemoral axial rotation with knee flexion.[7,8] In vitro cadaveric studies have provided some insight into the behavior of the ACL-deficient knee under controlled conditions,[9,10] including simulated muscle loads, but here the relationship to the in vivo situation is not straightforward. In order to investigate the potential for ACL injury to elicit changes in knee kinematics during activity, precise dynamic studies of small-scale differences in motions are therefore important. However, such measurements present significant methodological challenges. Relative

---

motion between skin and bone, for example, limits the precision of skin-marker–based motion analysis systems.[11,12] Methods involving periosteal or intracortical pinning of markers to bones, or implantation of radio-opaque materials in order to track motion with fluoroscopy, are invasive.[11–13]

Cine phase contrast magnetic resonance imaging (cine PC MRI), a technique originally developed to image blood and tissue velocity in the beating heart,[14] has been used to measure three-dimensional, in vivo, skeletal motion.[15] In the current study, we used a method we have developed that combines cine PC MRI with a 3-D geometric model-based tracking technique to measure knee kinematics during exercise.[16] A motion phantom experiment which used magnetic tracking of the motion of the device as a reference standard was used to estimate the accuracy of the method. Average differences between the two tracking methods were 2.82 mm RMS for anterior/posterior tibial position, and 2.63° RMS for axial rotation. An inter-trial repeatability study of human knee kinematics produced RMS differences in anterior/posterior position and axial rotation of 1.44 mm and 2.35°.

We compared knee kinematics in ACL-injured subjects and age- and activity-matched healthy control subjects as they performed a closely controlled active task—a supine, repetitive knee flexion/extension activity known as the short-arc quadriceps exercise. This exercise is commonly used for strengthening of the knee extensor mechanism, since it isolates the muscles that control the knee but does not threaten joint stability as the individual is not weight-bearing. The anterior pull of the quadriceps during active knee extension, however, loads the ACL and has the potential to elicit changes in knee kinematics in ACL-deficient (ACL-D) knees.[4]

We measured two determinants of the kinematics of the tibia relative to the femur: the anterior/posterior location of the tibia relative to the femur (A/P tibial positioning), and the internal/external rotation of the tibia relative to the femur (I/E rotation angle). Because of the increase in anterior laxity associated with ACL deficiency, as well as the anterior force produced by the quadriceps, we hypothesized that ACL-D knees would display more anterior tibial positioning than either the uninjured knees of the same subjects or knees in the control group. In light of the previously reported associations of ACL deficiency with changes in tibial axial rotation, we also hypothesized that significant differences would be seen between I/E rotation angles in ACL-D knees and these subjects' uninjured knees, as well as between the I/E rotation angles in the injured knees and the knees in the control group.

METHODS

Sixteen subjects (ACL-injured group, 12 male, 4 female, age = 25.8 ± 11.1 years) had sustained a complete isolated unilateral ACL rupture less than 6 months prior to testing. Before injury, all were regular participants in athletic activities that involve quick changes of direction and/or jumping, such as football, basketball, and soccer. ACL rupture was confirmed by diagnostic MRI, manual laxity testing (KT1000, MEDmetric, San Diego, CA) and subsequently arthroscopy. Exclusion criteria included a previous ACL injury, concomitant ligament pathology, fractures, greater than a trace knee joint effusion, and the presence of hip or ankle pathology. All ACL-injured subjects had experienced post-injury episodes of joint instability, and had failed a screening examination designed to identify potential ACL-injured *copers*,[17] i.e., candidates for nonoperative treatment of the injury. These subjects were therefore classified as ACL-injured non-copers.

Sixteen uninjured subjects (control group, age = 22.1 ± 6.9 years) matched by gender, activity, and age to the ACL-injured group were also tested. All subjects signed a statement of informed consent approved by the University of Delaware's Human Subject Review Committee.

Data Collection

The subjects lay supine in a magnetic resonance scanner (Signa LX 1.5T, General Electric Healthcare, Milwaukee, Wisconsin), with the thigh elevated by a positioning jig. A series of five axial plane static localizing images was acquired through the knee (scan parameters: slice spacing 15 mm, field of view 240 mm, matrix size 256×256, repetition time TR = 100 ms, echo time TE = 3.4 ms). These images were used to locate a sagittal localizing image (scan parameters: field of view 300 mm, matrix size 256×256, repetition time TR = 100 ms, echo time TE = 3.3 ms), prescribed through the femoral intercondylar notch. Cine-PC MRI data (scan parameters: field of view 300 mm, encoding velocity 35 cm/s, matrix 256×256, repetition time 17 ms, echo time 6.4 ms) were acquired on this plane while the knee was actively moved between full extension and approximately 30° flexion for approximately 5 min and 30 s duration. Subjects voluntarily coordinated their repetitions to the beats of a metronome played through headphones. The specified frequency of repetition was 35 cycles per minute, and data acquisition was synchronized by an optical trigger positioned under the ankle of the subject. No loading additional to the weight of the foot and shank was applied.

Subsequently to the dynamic scan, a static high resolution 3-D scan was acquired of each knee, yielding a series of 124 serial anatomical images in the axial plane (scan parameters: slice thickness 1.0 mm, matrix 256×256, field of view 180×180 mm).

Data Processing

The peripheries of the distal femur and proximal tibia/fibula were traced on the serial images of the 3-D scan using a digitizing tablet and the IMOD (University of Colorado, Boulder, Colorado) software package.[18] Custom developed software was combined with a surface triangulation package[19] to reconstruct 3-D geometric models of the femur and tibia/fibula for each knee. The cine phase contrast imaging protocol provides a sequence of 24 frames of data through the cycle on the specified image plane. Each resulting data frame yields four separate images on the plane: one is the usual anatomical cross-section (magnitude image), and the others are encoded with velocity information in each of the three principal directions of the image[14] (Fig. 1). The cine-PC rigid body tracking technique was used to optimally register modeled trajectories of the geometric bone models to the cine-PC magnitude and velocity data.[16] In this method, the intersection of each bone model with a simulated image

plane is calculated as the model moves along a computed trajectory, and cine-PC velocity data are sampled from the regions of the velocity images within the area of this intersection. From the sampled velocity data, the instantaneous linear and angular velocities of a coordinate system fixed to the bone model are estimated, and integration of the linear and angular velocities is used to predict updated trajectories. An iterative process proceeds until an optimal agreement is reached between the computed trajectory and the velocity and observed intersection along the path. Incorporation of through-plane velocity data enables the measurement of rotations around the in-plane axes of the images; it is this that allows the detection of tibial axial rotation from velocity data on a single sagittal plane.

Anatomically based coordinate systems were fixed within each geometric bone model. Landmarks were digitized in the sagittal localizing images to establish the directions of the long axes of the femur and tibia in that plane. The medial/lateral axis of the femur was aligned parallel to planes tangent to the posterior and inferior extents of the condyles. The tibia's medial/lateral axis was oriented parallel to both the posterior aspect of the tibia/fibula and the tibial plateau.

A joint coordinate system[20] was used to calculate tibial A/P positioning and I/E tibial rotation angle
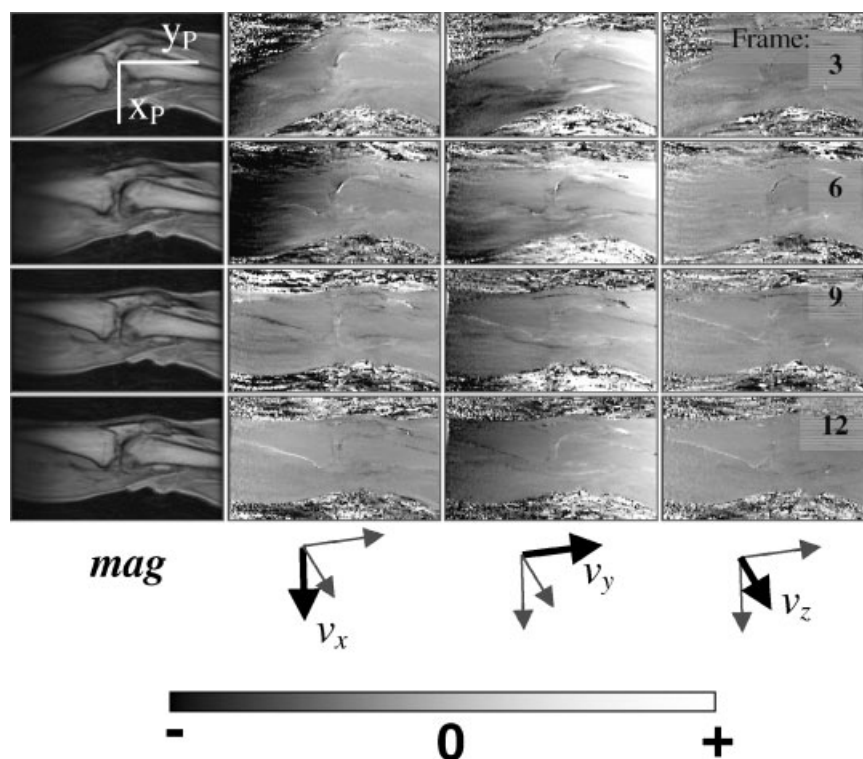

Figure 1. A sequence of cine-PC magnitude *(mag)* and velocity image frames, acquired through a static mid-sagittal plane of the knee during the short arc quadriceps exercise. Velocity components at each pixel location in vertical, horizontal, and through-plane directions *(vx, vy, vz,* respectively) are represented as positive and negative values according to the gray scale shown at the bottom of the figure.

parameters. The femoral and tibial landmarks for measuring joint position parameters were placed at the most distal point in the femoral notch and the midpoint of the tibial intercondylar eminences, respectively. The A/P tibial positioning parameter is a measure of the distance from the femoral origin point to the tibial origin point along the anteriorly directed floating axis.[20] Since the tibial origin is posterior to the femoral origin, the A/P position parameter values are negative; they become less negative as the tibia moves anterior. To compare between the kinematics in the ACL-injured and control groups, one knee in each control subject was assigned as the index knee, matched by side to the injured knee of the corresponding age- and activity-matched subject in the ACL-injured subject group. The contralateral knee was termed that subject's reference knee. Interpolating cubic spline functions (MATLAB, The Mathworks, Natick, MA) were fitted to the values of each kinematic parameter as functions of knee flexion angle during the extension phase of the motion cycle. The spline functions were evaluated at five-degree increments in the range from 30° to 5° of knee flexion.

Multifactor ANOVA statistical analyses were used to evaluate the dependence of the kinematic variables on the following two repeated measures: (i) side: injured vs. uninjured (matched by subject in the control group as described above), and (ii) flexion angle at each five-degree increment of flexion. The subject group (ACL-injured vs. control) was incorporated as a between-groups factor. Post-hoc testing with paired and independent t-tests was used to evaluate the sources of main effects and interactions observed. Significance levels were set at $\alpha = 0.05$ for all statistical analyses.

RESULTS

Range of Motion in Knee Flexion

The mean ranges of motion in the knees of each group ranged between 28.9° and 29.7°. The mean minimum knee flexion in the knees of each group varied between 1.8° and 2.4°. There was no statistically significant difference between minimum angles, maximum angles, or ranges of motion between the paired knees of either group ($p > 0.34$).

Tibial Anterior/Posterior Positioning

The repeated measures ANOVA demonstrated a main effect of more anterior tibial positioning with knee extension (Fig. 2). No significant
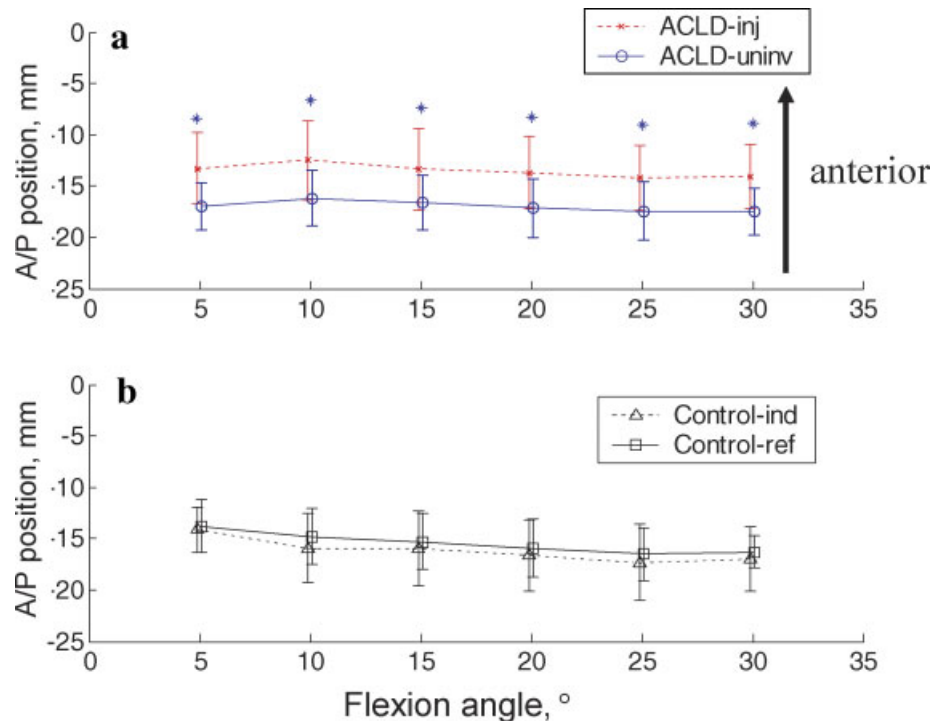

Figure 2. Anterior/posterior position parameter for each 5° comparison knee flexion angle. (a) ACL-D group; (b) control group (ind, index knees, matched to injured knees of ACL subjects; ref, contralateral knees). In the ACL-D group, the tibial positions in the injured knees were significantly anterior to the uninvolved knees at all angles tested, whereas little difference was seen between sides in the control group. Asterisks: significant difference ($\alpha = 0.05$) from paired t-test within groups; error bars: standard deviation.

interaction between subject group and flexion angle was observed, indicating that anterior movement with knee extension was not differentiated by subject group. There was also no significant interaction between side and angle; therefore, the effect was not found to be a function of the side tested. Post-hoc testing with an independent t-test showed that tibial positioning at 5° flexion was significantly anterior to that at 25° flexion.

There was a significant interaction between the effects of the side and group factors on A/P tibial positioning. A significant main effect of side on A/P tibial positioning was found. Post-hoc testing using paired t-tests showed that the injured knees of the ACL-injured subjects had significantly anterior positioning relative to that group's uninvolved knees at all angles tested (Fig. 2). The side-to-side differences in tibial positioning were larger in ACL-injured subjects than in control subjects. In ACL-injured subjects, the mean difference in tibial positioning averaged over the flexion angles tested was 3.4 ± 2.8 mm, and the maximum mean difference was 3.8 ± 3.0 mm, whereas in the control group the average and maximum differences between sides were -0.8 ± 2.5 and -1.2 ± 2.9 mm, respectively. The more anterior tibial positioning of the injured knees was apparent in several of the ACL-injured subjects on visual comparison of the cine-PC magnitude image sequences of each knee (Fig. 3). The tibial positioning in ACL-injured knees was also anterior to that in the index knees of the control subjects, as demonstrated by post-hoc testing with independent t-tests ($p < 0.05$ at 10, 20, 25° flexion; also $p = 0.057$ at 15°).

Tibial Internal/External Rotation

The repeated measures ANOVA analysis on tibial rotation demonstrated only a significant main effect of angle on axial rotation angle. This was caused by the external tibial rotation with knee extension that was recorded in both groups (Fig. 4), similar to previous observations on screw home motion.[21] Post-hoc testing with independent t-tests indicated that knees were significantly externally rotated at 5° flexion relative to the values at 30° flexion ($p < 0.025$). The mean excursion in external rotation during extension from 30° to 5° ranged between 4.8° and 5.8° in the subject group/side categories studied. Tibial external rotation was similar in ACL-D and uninjured knees; the hypothesis that axial rota-
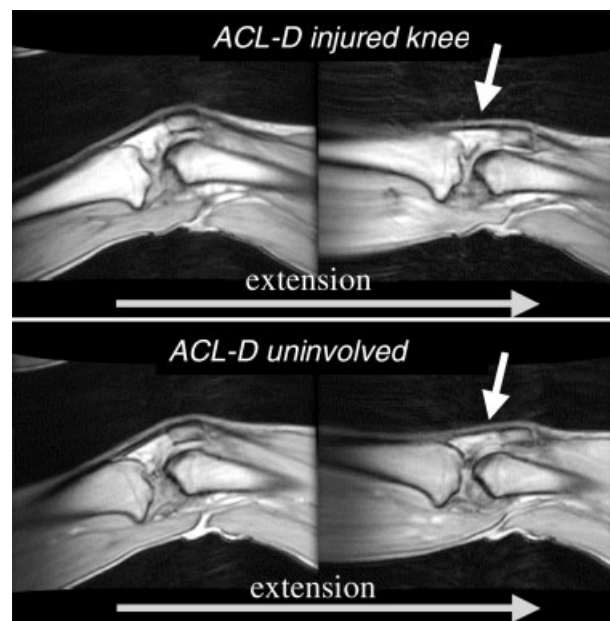

Figure 3. Cine-PC MRI magnitude images indicate a more anterior tibial position in the injured knee of one ACL-deficient subject. The anterior tibial subluxation of the ACL injured knee is most apparent from the decreased angle of insertion of the patellar ligament onto the proximal tibia (arrows).

tion patterns would be affected by ACL deficiency was not supported by the data.

DISCUSSION

Subjects that had sustained ACL injuries demonstrated altered knee kinematics as they performed the dynamic short arc quadriceps extension exercise. Our hypothesis that the ACL-deficient knees of the injured subjects would exhibit significantly more anterior tibial positions than their uninjured knees, as well as the knees of the control subjects, was supported by the data. Anterior motion of the tibia with knee extension was seen in both sides in both groups of subjects. All groups exhibited tibial external rotation motion consistent with the screw home mechanism in terminal extension. Our hypothesis that alterations in tibial external rotation motion would also be exhibited was not supported by the data.

The findings that these individuals have altered kinematics during a knee extension exercise, in the absence of external joint loading, are provocative. Others have used computerized goniometer linkages to measure differences in A/P tibial
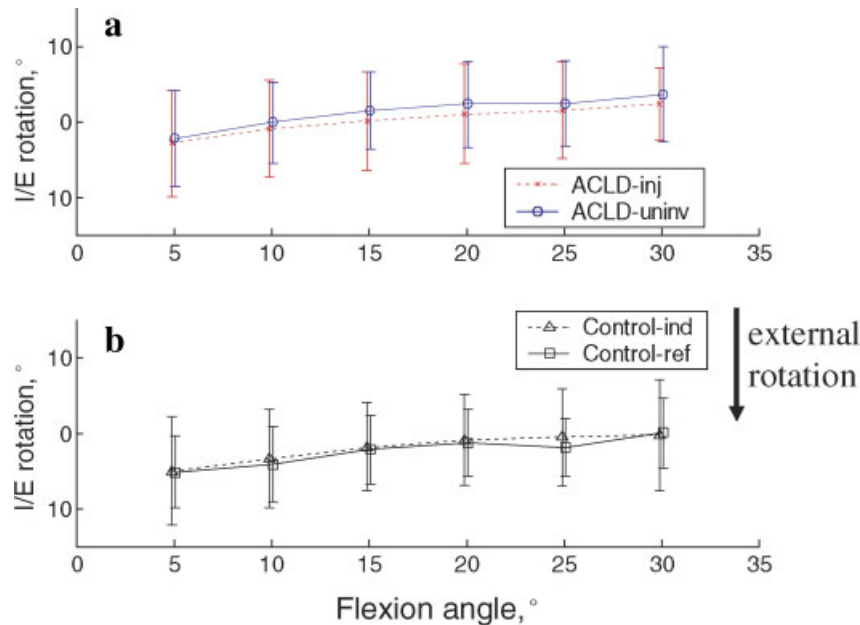
Figure 4. Tibial axial rotation parameter for each 5° comparison knee flexion angle. (a) ACL-D group; (b) control group (ind, index knees, matched to injured knees of ACL subjects; ref, contralateral knees); error bars, standard deviation.

translation in non-loaded and moderately loaded knee extension tasks. Kvist and Gillquist[22] measured an average increase in A/P translation of 2.2 mm in subjects' ACL-deficient knees relative to their uninjured knees as they performed a 90° non-loaded extension exercise in the seated position. Lysholm and Messner[23] measured an average increase in A/P tibial translation (involved vs. uninjured knee) of 2 mm as subjects slowly performed a supine-position quadriceps extension in the last 30° of extension range (a similar exercise to that of the current study). Kizuki et al.[24] reported 4.1 mm more A/P translation in ACL-deficient knees relative to the uninjured knees of their subjects as they performed a non-loaded, seated 90° knee extension task in an exercise dynamometer (Biodex Medical Systems, Inc., Shirley, NY). In the above studies, tibial translation was defined as displacement relative to data collected in reference positions, such as non-loaded full extension,[22,23] or a series of values measured as the knee was passively extended.[24] Joint positioning in such reference configurations may itself be affected by pathology; for instance, fixed joint subluxations have been reported in ACL-deficient subjects during standing by Almekinders and Chiavetta.[25] In that study, tibiofemoral position was determined from landmarks drawn on sagittal plane radiographs. Average tibial position in ACL-injured knees was

reported to be 3.9 mm anterior to the uninjured knees. Tibial subluxation has also been correlated with ACL rupture in diagnostic MRI scans acquired when subjects were supine, relaxed, and in full knee extension.[26] Comparison of goniometer linkage-based studies to medical imaging-based studies such as the current one should be evaluated in this knowledge. Measurement error can also be introduced by relative motion between goniometer linkages and bones during motion.[27] Nevertheless, the finding of anterior tibial displacement in the injured knees of our subjects is consistent with the results of these studies.

Franklin et al.[28] measured A/P tibiofemoral position as subjects performed a quadriceps extension task against the additional load of a 6.8 kg weight suspended from the ankle. Tibiofemoral position was measured using a similar radiographic method to that of Almekinders and Chiavetta.[25] The averaged tibial position was 7.5 mm more anterior in the ACL-injured knees than in the same subjects' uninjured knees. This displacement is considerably larger than the average relative anterior tibial positioning of the injured knees of the ACL-D subjects in the current study (3.5 mm). The greater tibial displacement would be expected given the larger quadriceps force (and hence the larger anterior directed component) necessary to support the weight of the ankle loading used by Franklin et al.[28]

We may be able to shed further light on the mechanism by which this anterior positioning occurs. We studied muscle activation patterns dynamically in ACL-deficient non-copers as they performed the short arc quadriceps exercise in a simulated MRI chamber using electromyography (EMG).[29] In that study, we found impairment in the control of the quadriceps; specifically, whereas uninjured subjects generally reduced activation to approximately zero values when the knees were flexed (and hence the shank's weight was supported by the testing jig), the ACL-D subjects exhibited continuous activation in this phase. This failure to turn off the quadriceps during knee flexion was also observed as subjects performed a static task evaluating neuromuscular activation while generating knee moments in varied directions.[29] The anterior tibial subluxation and evidence of increased dynamic translation observed in the current study may be caused by a combination of the pathological increased activation of the quadriceps and the decreased restraint to anterior tibial motion. No evidence of compensatory activation of the hamstrings muscles to limit anterior tibial translation was found in our electromyographic study.[29]

The amount of external rotation motion observed with extension (the screw home motion) varied between 4.8° and 5.8° in the 25° range of flexion angles tested in the current study. Ishii et al.[21] used an intracortically-fixed goniometer linkage to measure screw home motion in uninjured knees as subjects actively moved their knees between 60° flexion and full extension. These authors measured a steady external rotation with an average excursion of approximately 7° between 30° and 5° of knee flexion. In a study of passive knee movement in cadaver knees, Wilson et al.[8] found an average of approximately 8° internal tibial rotation between 5° and 30° of flexion as cadaver knees were flexed from full extension. The axial rotation levels we observed were therefore in a similar range to those reported previously. The small discrepancy may have been caused by a previously observed tendency of the method to somewhat under-record axial rotation motion.[16]

The relationships between the roles of the cruciate ligaments and tibial axial rotation motion have been explored in model studies[30] as well as in vivo.[31,32] Similarly to Jonsson et al.,[33] the current study reported little change to the screw home kinematics. They speculated that this was because the anterior tibial translation produced was relatively slight. The task in the current study was less challenging to the ACL (non-loaded extension vs. extension against a 30N ankle weight[33]), perhaps explaining why we did not observe an effect of ACL-deficiency on tibial axial rotation.

This study has provided important information on changes in active knee kinematics after ACL injury; however, some limitations of the study warrant mention. We chose the short-arc quadriceps exercise because it allows study of joint kinematics in highly controlled, repeatable conditions that do not challenge knee stability. In the future though, it will be important to develop methods to assess kinematics when the joint is subjected to compressive load, as occurs during more functional activities. Also, we note that the cine phase contrast technique has potential susceptibility to error caused by inconsistencies in subject motion. Although our validation studies have demonstrated that the technique is robust to typical levels of these variations, this will be a more significant concern if the method is used to study more challenging activities.

Biological changes in articular cartilage in response to changes in its mechanical environment have been demonstrated,[3,34] and significant changes in joint contact pressure distribution and cartilage thickness have been observed as early as 16 weeks after transection of the ACL in animal studies.[3] The differences in joint positioning observed between the knees of the ACL-injured subjects in the current study must change the patterns of contact between the articulating surfaces. It therefore seems likely that post-injury impairment of joint stability may indeed be a causal factor in the progression of osteoarthritis in these patients. Further research will be needed to fully explore the contributions of the dynamic and passive aspects of joint stabilization to the kinematic changes seen. Noninvasive measurements of joint kinematics during activity, using medical imaging-based techniques such as the one used in this study, promise to play a crucial role in this work.

ACKNOWLEDGMENTS

REFERENCES

1. Daniel DM, Stone ML, Dobson BE, et al. 1994. Fate of the ACL-injured patient. A prospective outcome study. Am J Sports Med 22:632–644.

2. Maletius W, Messner K. 1999. Eighteen- to twenty-four-year follow-up after complete rupture of the anterior cruciate ligament. Am J Sports Med 27:711–717.

3. Herzog W, Clark A, Wu J. 2003. Resultant and local loading in models of joint disease. Arthritis Rheum 49:239–247.

4. Grood ES, Suntay WJ, Noyes FR, et al. 1984. Biomechanics of the knee-extension exercise. Effect of cutting the anterior cruciate ligament. J Bone Joint Surg [Am] 66:725–734.

5. Markolf KL, Gorek JF, Kabo JM, et al. 1990. Direct measurement of resultant forces in the anterior cruciate ligament. An in vitro study performed with a new experimental technique. J Bone Joint Surg [Am] 72:557–567.

6. More RC, Karras BT, Neiman R, et al. 1993. Hamstrings—an anterior cruciate ligament protagonist. An in vitro study. Am J Sports Med 21:231–237.

7. Andersen HN, Dyhre-Poulsen P. 1997. The anterior cruciate ligament does play a role in controlling axial rotation in the knee. Knee Surg Sports Traumatol Arthrosc 5:145–149.

8. Wilson DR, Feikes JD, Zavatsky AB, et al. 2000. The components of passive knee movement are coupled to flexion angle. J Biomech 33:465–473.

9. Kanamori A, Sakane M, Zeminski J, et al. 2000. In-situ force in the medial and lateral structures of intact and ACL-deficient knees. J Orthop Sci 5:567–571.

10. Sakane M, Livesay GA, Fox RJ, et al. 1999. Relative contribution of the ACL, MCL, and bony contact to the anterior stability of the knee. Knee Surg Sports Traumatol Arthrosc 7:93–97.

11. Manal K, McClay Davis I, Galinat B, et al. 2003. The accuracy of estimating proximal tibial translation during natural cadence walking: bone vs. skin mounted targets. Clin Biomech 18:126–131.

12. Tashman S, Anderst W. 2003. In-vivo measurement of dynamic joint motion using high speed biplane radiography and CT: application to canine ACL deficiency. J Biomech Eng 125:238–245.

13. Ramsey DK, Lamontagne M, Wretenberg PF, et al. 2001. Assessment of functional knee bracing: an in vivo three-dimensional kinematic analysis of the anterior cruciate deficient knee. Clin Biomech 16:61–70.

14. Pelc NJ, Herfkens RJ, Shimakawa A, et al. 1991. Phase contrast cine magnetic resonance imaging. Magn Reson Q 7:229–254.

15. Sheehan FT, Zajac FE, Drace JE. 1998. Using cine phase contrast magnetic resonance imaging to non-invasively study in vivo knee dynamics. J Biomech 31:21–26.

16. Barrance PJ, Williams GN, Novotny JE, Buchanan TS. 2005. A method for measurement of joint kinematics in vivo by registration of 3-D geometric models with cine phase contrast magnetic resonance imaging data. J Biomech Eng 127:829–837.

17. Fitzgerald GK, Axe MJ, Snyder-Mackler L. 2000. Decision-making scheme for returning patients to high-level activity with nonoperative treatment after anterior cruciate ligament rupture. Knee Surg Sports Traumatol Arthrosc 8:76–82.

18. Kremer JR, Mastronarde DN, McIntosh JR. 1996. Computer visualization of three-dimensional image data using IMOD. J Struct Biol 116:71–76.

19. Geiger B. 1993. Three-dimensional modeling of human organs and its application to diagnosis and surgical planning. Sophia Antipolis, France: Institut National de Recherche en Informatique et Automatique. 129 p.

20. Grood ES, Suntay WJ. 1983. A joint coordinate system for the clinical description of three-dimensional motions: application to the knee. J Biomech Eng 105:136–144.

21. Ishii Y, Terajima K, Terashima S, Koga Y. 1997. Three-dimensional kinematics of the human knee with intracortical pin fixation. Clin Orthop Relat Res 343:144–150.

22. Kvist J, Gillquist J. 2001. Sagittal plane knee translation and electromyographic activity during closed and open kinetic chain exercises in anterior cruciate ligament-deficient patients and control subjects. Am J Sports Med 29:72–82.

23. Lysholm M, Messner K. 1995. Sagittal plane translation of the tibia in anterior cruciate ligament-deficient knees during commonly used rehabilitation exercises. Scand J Med Sci Sports 5:49–56.

24. Kizuki S, Shirakura K, Kimura M, et al. 1995. Dynamic analysis of anterior tibial translation during isokinetic quadriceps femoris muscle concentric contraction exercise. The Knee 2:151–155.

25. Almekinders LC, Chiavetta JB. 2001. Tibial subluxation in anterior cruciate ligament-deficient knees: implications for tibial tunnel placement. Arthroscopy 17:960–962.

26. Vahey TN, Hunt JE, Shelbourne KD. 1993. Anterior translocation of the tibia at MR imaging: a secondary sign of anterior cruciate ligament tear. Radiology 187:817–819.

27. Vergis A, Hindriks M, Gillquist J. 1997. Sagittal plane translations of the knee in anterior cruciate deficient subjects and controls. Med Sci Sports Exerc 29:1561–1566.

28. Franklin JL, Rosenberg TD, Paulos LE, et al. 1991. Radiographic assessment of instability of the knee due to rupture of the anterior cruciate ligament. A quadriceps-contraction technique. J Bone Joint Surg [Am] 73:365–372.

29. Williams GN, Barrance PJ, Snyder-Mackler L, et al. 2004. Altered quadriceps control in people with anterior cruciate ligament deficiency. Med Sci Sports Exerc 36:1089–1097.

30. Wilson DR, Feikes JD, O'Connor JJ. 1998. Ligaments and articular contact guide passive knee flexion. J Biomech 31:1127–1136.

31. Andriacchi TP, Dyrby CO. 2005. Interactions between kinematics and loading during walking for the normal and ACL deficient knee. J Biomech 38:293–298.

32. Karrholm J, Selvik G, Elmqvist LG, et al. 1988. Three-dimensional instability of the anterior cruciate deficient knee. J Bone Joint Surg [Br] 70:777–783.

33. Jonsson H, Karrholm J, Elmqvist LG. 1989. Kinematics of active knee extension after tear of the anterior cruciate ligament. Am J Sports Med 17:796–802.

34. Mow VC, Wang CC. 1999. Some bioengineering considerations for tissue engineering of articular cartilage. Clin Orthop 45:S204–S223.