

University of Nebraska at Omaha DigitalCommons@UNO

Journal Articles

Department of Biomechanics

1-2005

Three-dimensional kinematics of the tibiofemoral joint in ACL-deficient and reconstructed patients shows increased tibial rotation

Anastasios D. Georgoulis University of Ioannina

Stavros Ristanis University of Ioannina

Constantina O. Moraiti University of Ioannina

Argyris Mitsou University of Athens

Manfred Bernard

See next page for additional authors

Follow this and additional works at: https://digitalcommons.unomaha.edu/biomechanicsarticles Part of the <u>Biomechanics Commons</u>

Recommended Citation

Georgoulis, Anastasios D.; Ristanis, Stavros; Moraiti, Constantina O.; Mitsou, Argyris; Bernard, Manfred; and Stergiou, Nicholas, "Three-dimensional kinematics of the tibiofemoral joint in ACL-deficient and reconstructed patients shows increased tibial rotation" (2005). *Journal Articles*. 142.

https://digitalcommons.unomaha.edu/biomechanicsarticles/142

This Article is brought to you for free and open access by the Department of Biomechanics at DigitalCommons@UNO. It has been accepted for inclusion in Journal Articles by an authorized administrator of DigitalCommons@UNO. For more information, please contact unodigitalcommons@unomaha.edu.



Authors

Anastasios D. Georgoulis, Stavros Ristanis, Constantina O. Moraiti, Argyris Mitsou, Manfred Bernard, and Nicholas Stergiou

Three-Dimensional Kinematics of the Tibiofemoral Joint in ACL-Deficient and Reconstructed Patients Shows Increased Tibial Rotation

Anastasios D. Georgoulis, MD,* Stavros Ristanis, MD,* Constantina Moraiti, MD,* Argyris Mitsou, MD,† Manfred Bernard, MD,‡ and Nick Stergiou, PhD§

*Department of Orthopaedic Surgery, Orthopaedic Sports Medicine Center of Ioannina, Medical School, University of Ioannina, Ioannina, Greece.

[†]Department of Orthopaedics, Medical School, University of Athens, Athens, Greece.

[‡]Priv.-Doz., Klinik Sanssouci, Berlin, Germany.

§HPER Biomechanics Laboratory, University of Nebraska at Omaha, Omaha, NE.

Address reprint requests to Anastasios Georgoulis, MD, Methodiou Anthrakitou 1, Ioannina 45221, Greece. E-mail: oaki@cc.uoi.gr

ABSTRACT

The study of the altered knee joint movement patterns that follow anterior cruciate ligament (ACL) rupture can be very insightful in the development of prevention and therapeutic strategies concerning this injury. This can be achieved through three-dimensional kinematic analysis, because it provides an objective evaluation in vivo of the knee joint function. It has been demonstrated that ACL-deficient patients develop functional adaptations (ie, quadriceps avoidance gait) and walk with the knee in a more extended position to compensate for the ACL loss. Furthermore, it has been shown that ACL rupture results in anterior tibial translation and excessive tibial rotation while performing everyday activities. Although anterior tibial translation is restored with ACL reconstruction, tibial rotation seems to be restored only during low-demanding activities, whereas it remains increased during highdemanding activities. A possible explanation for the lack of restoration of tibial rotation to normal levels is the absence of complete reinstatement of the actual anatomy of the ACL. Reconstruction techniques should become more anatomic and try to approximate both ACL bundles. Two-bundle reconstruction may have advantages over single-bundle reconstruction, with respect to regaining a structure that morphologically and functionally better resembles a normal ACL. This technique however, has not been investigated dynamically, and future research should be performed. Therefore, long-term follow-up studies should focus on the advantages and disadvantages of different surgical procedures, whether it is the graft material or the tunnel positioning, so that dynamic knee function is restored and future pathology of the knee joint is prevented.

KEYWORDS increased tibial rotation, ACL reconstruction, 3D kinematics, high-demanding activities, pivoting, anatomic tunnel placement

The anterior cruciate ligament (ACL) is an important structure in controlling knee joint stability and movement. It has been widely demonstrated that the ACL stabilizes the tibia from anterior translation relative to the femur and limits excessive tibial rotation.1 Furthermore, it functions as a secondary restraint to varus or valgus angulation at full extension.¹

ACL rupture is one of the most common sports-related injuries that leads to deterioration of knee joint function, with development of pathological anterior drawer, rotatory instability, poor control of muscle function, and muscle weakness.²⁻⁵ Specifically, it has been demonstrated that there is a deficit in the quadriceps strength; it has been shown that hamstring weakness might indicate low functional levels in ACL-deficient knees.³ Long-term follow-up studies have also



Figure 1 Variables evaluated with 3D kinematics. Top panels: flexion/extension; bottom panels: external/internal rotation.

shown that ACL rupture is associated with the development of chondral injuries,⁶ meniscal tears, degeneration of the articular cartilage, and eventually posttraumatic arthritis of the medial compartment.⁷⁻¹⁰

Functionally, patients respond to ACL loss in different ways. Specifically, some patients (noncopers) modify their lifestyle and don't take part in high-risk activities to avoid further episodes of giving way, whereas a small percentage of ACL-deficient individuals (copers) continue exercising at their preinjury activity level.¹¹ Noyes et al¹⁰ observed that approximately one third of patients who sustain an ACL injury compensate enough to pursue recreational activities, another one third make compensations but discontinue many activities, and one third do not respond to conservative treatment and require surgical reconstruction. Currently, patients with ACL deficiency (especially the young and those with high level of activity) generally undergo ACL reconstruction. This is because surgical techniques of ACL reconstruction have been improved with the aid of knowledge gained from basic science and clinical research. ACL reconstruction has become a common procedure, and good-to-excellent clinical results have been widely reported.

However, there seems to be room still for improvement in the treatment of ACL deficiency. This improvement can be achieved through a profound understanding of the function of the injured knee during daily living activities. This understanding can be gained through gait analysis.

Gait Analysis

Gait analysis allows the quantification of gait parameters and provides the objective measures necessary to evaluate dynamic functional levels of patients performing everyday activities. Gait analysis results from the study of kinetic, kinematic, and electromyographic parameters.

Kinetics is the study of the forces that cause movement, whereas kinematics refers to the description of motion independent of the forces that cause movement to take place. Linear and angular displacements, velocities, accelerations, and joint angles constitute some kinematic parameters. With three-dimensional (3D) kinematic analysis to study the tibiofemoral joint, all six degrees of freedom (three rotations, ie, internal/external, abduction/adduction, flexion/extension and three translations, ie, anterior/ posterior drawer, medial/lateral shift, distraction/compression) can be discerned (Fig. 1).

3D kinematic analysis of the tibiofemoral joint can be obtained through roentgen stereophotogrammetric analysis (RSA).¹²⁻¹⁴ But, although RSA provides a direct measurement of bony motion in vivo, it is limited by the exposure to radiation and invasive nature of the procedure.

Study of 3D tibiofemoral kinematics has also been conducted with six-degrees-of-freedom electrogoniometers.¹⁵⁻¹⁷ With goniometers that mount on the leg surface, the accuracy of the measurement is affected by skin and soft tissue movement, as well as by the precision by which the linkage is defined with respect to the internal bony structures of the knee joint.¹⁸ To overcome these problems, researchers have used goniometers attached to intracortical pins inserted into the tibia and femur, which is limited by its invasive nature.¹⁹

Mainly, 3D kinematic analysis is conducted with video cameras and opto-electronic digitizers. Specifically, markers are placed on specific anatomic bony landmarks, depending on the biomechanical model used (Fig. 2),²⁰ and the subject performs a given motor task. The position of the markers during the task is recorded and the signal is then converted it into a digital format for computer processing.



Figure 2 The retroreflective marker set required for the motion data collection tests (model by Davis et al²⁰).

Like goniometers, surface markers may not accurately represent the underlying bone motion during highdynamic activities,²¹ because the relative movements between skin markers and the underlying bone may introduce errors.^{18,19,21,22} Especially, subtle movements such as internal/external rotation and adduction/abduction are affected more. It can certainly be supported that this source of error represents one of the most important unsolved problems in in vivo kinematic analysis, because such perturbations are difficult to remove with low-pass filtering, as their frequency content is close to that of the motion.

One way to avoid these limitations is to directly measure skeletal motion in vivo²¹ with intracortical pins. According to Cappozzo et al,²³ the motion of the marker with respect to the underlying bone due to skin movement ranges from a few millimeters to as high as 40 mm. Attaching the reflective markers on intracortical pins can eliminate this source of error. Certainly, the applicability of such methods is limited, because the implantation of intracortical pins is a highly in- vasive procedure that may cause discomfort or pain to the patient and result in restriction of movement. In addition, we believe that implantation of intracortical pins is a method that is limited by the sample size, because an effective number of volunteers cannot be found.

Tibiofemoral Kinematics in the Sagittal Plane in ACL-Deficient and ACL-Reconstructed Subjects

ACL rupture and its effects on knee kinematics have been investigated extensively with regard to the sagittal plane. In most cases, to achieve a thorough evaluation of the joint function, kinematic data were combined with kinetic and electromyographic data that were collected simultaneously.^{15,24-28}

Loss of the ACL causes excessive anterior tibial translation relative to the femur ranging from 30° of knee flexion to full extension.²⁹ It has been shown by several studies that after ACL rupture, patients may use stronger

contraction of the hamstrings to pull the tibia posteriorly³⁰ or walk with weaker contraction of the quadriceps to avoid pulling the tibia anteriorly.^{24,31}

A variety of studies have examined the gait of ACL-deficient patients, but the study by Berchuck et al³¹ is one of the most widely cited. They studied the gait of 16 ACL-deficient patients and found consistent abnormalities in their walking pattern. At mid stance, the ACL-deficient patients were found to exhibit an external knee extension moment, requiring internal flexing moments to maintain equilibrium, that was different from the external flexion moment found in controls. The investigators interpreted this tendency toward an increased internal flexing moment as a reduction in the force generating the extending moment, ie, the quadriceps force. Thus, they used the term "quadriceps avoidance gait" to characterize the walking pattern of these patients.

The exact mechanism by which avoidance of quadriceps contraction reduces anterior tibial translation has been investigated by studying the effects of strain on ACL cadaver knees with a transducer placed on the ACL.³² The ACL strain depended on whether the knee flexion angle was changed passively or after contraction of the quadriceps muscle. Simulated isometric quadriceps contraction increased significantly the anterior-medial ACL strain, above the normal resting level, through the first 45° of knee flexion. During 60° of flexion or greater, the same contractions produced lower ACL strain. This reduction in strain was significant at 105° of flexion and at 120° of flexion. It is obvious that excessive anterior translation of the tibia during gait would be avoided if the patients were able either to avoid excessive activation of the quadriceps by walking with the knee in a more extended position or to avoid quadriceps activation when the knee is near full extension.

Wexler et al²⁸ found that 7.5 years after injury, ACL-deficient patients walked with increased knee extension angles during the terminal stance. This gait pattern, with the knee in a more extended position, results in lower demands placed on the quadriceps. This can be considered an additional mechanism that produces the quadriceps avoidance gait pat- tern in chronic ACL-deficient knees as the nervous system adapts to the injury.³¹ Patel et al^{26,27} also reported that 72% of the patients with a quadriceps avoidance gait walked with a significantly reduced mid-stance knee flexion angle that allowed them to reduce the demand placed on the quadriceps during the stance phase. Therefore, the anterior pull on the tibia was reduced, and the knee was more stable. However, in the remaining 28%, Patel found an increased peak external hip flexion moment. The authors hypothesized that a forward trunk lean by these patients probably produced the increase in the hip flexion moments, thereby helping to decrease the strain placed on the quadriceps during mid stance.

Beard et al²⁵ examined ACL-deficient patients approximately 2 years after injury and reported that they walked with significantly greater terminal knee flexion angle. They also found a prolonged period of average hamstrings activity for the deficient side relative to the intact side during the stance phase. Furthermore, contrary to other studies,^{24,27,28} they found quadriceps activity duration to be similar in the ACL-deficient and control groups.

Investigations involving ACL reconstructed subjects suggest that time since surgery may play an important role in the return of normal gait patterns.³³⁻³⁶ Devita et al³⁵ examined ACL-reconstructed patients 3 weeks and 6 months postoperatively. They found a reduced but prolonged hip extensor moment pattern and a sustained knee extensor moment 3 weeks postoperatively. However, at 6 months after surgery, the ACL-reconstructed subjects demonstrated knee and hip moment patterns more similar to the control group, suggesting that ACL-reconstructed subjects can regain preinjury gait characteristics over time.

Bush-Joseph et al³⁴ studied ACL-reconstructed subjects 8 months postoperatively and reported only slight reductions in the peak knee extensor moment during gait. Timoney et al³⁶ reported that 10 months after surgery, ACL-reconstructed subjects walked with a significantly reduced knee extensor moment compared with control subjects, suggesting that not all patients demonstrate a time-related return of normal gait patterns during the first year after ACL reconstructive surgery. Bulgheroni et al³³ studied the gait patterns of ACL-reconstructed subjects 2 years postoperatively and reported no significant differences in sagittal plane knee or hip moments, suggesting that, given time, ACL-reconstructed subjects can regain normal knee moment gait patterns.

Tibiofemoral Kinematics in the Frontal and Transverse Planes in ACL-Deficient and ACL-Reconstructed Subjects

However, less is known regarding the transverse and the frontal plane movements of the tibia with respect to the femur. This is probably because of the complexity and technical limitations of 3D analysis. Thus, although flexion-extension knee kinematics have been extensively investigated during gait in ACL-deficient and ACL-reconstructed patients, tibial adduction/abduction and internal/external rotation have not received similar attention.

Karrholm et al³⁷ recently used RSA to show that tibial rotation during active extension in healthy individuals ranges from 9.9° of internal to 1.6° of external rotation. Using markers fixed on intracortical pins, Lafortune et al¹⁸ examined tibial rotation during gait and found similar patterns but different magnitudes. Possible explanation to this difference in the ranges of rotation are the variable accuracies of the methods applied as well as the fact that

knee joint loading is different during gait and active extension. Therefore, great caution should be taken when interpreting such results.

Using RSA, Jonsson et al¹⁴ found no significant difference in tibial rotation or adduction/abduction between injured and intact knees in active extension. With the use of six-degree-of-freedom goniometers, Zhang et al¹⁵ reported that ACL-deficient patients walk with more tibial external rotation and more abduction than healthy subjects, which may help these patients compensate for the rupture by avoiding positions where the knee would be unstable because of the loss of the ACL constraint. On the other hand, Marans et al¹⁷ found no differences in tibial rotation in ACL-deficient knees during walking. Using an optoelectronic system, Andriacchi et al³⁴ showed that ACL-deficient knees exhibit increased internal tibial rotation throughout the entire gait cycle. This finding is in line with our results.

Using an optoelectronic system, we evaluated the 3D kinematics of ACL-deficient and ACL-reconstructed patients during a low-demanding activity such as walking.³⁸ We examined 13 patients with unilateral ACL deficiency (time from injury, 7.6 ± 4.3 weeks), 21 patients who had undergone ACL reconstruction (time from reconstruction, 30 ± 16.9 weeks), and 10 healthy controls. ACL reconstruction was performed arthroscopically with BPTB (bone patellar tendon bone) autograft.³⁹ Data collection was conducted with an optoelectronic system sampling at 50 Hz. Reflective markers were placed on both lower limbs according to the model developed by Vaughan. The subjects walked on a 10-m walkway at their self-selected pace. Twelve strides from six trials (two consecutive strides from each side) were averaged for the calculation of the gait variables. Specifically, we examined knee flexion at toe-off, maximum knee flexion during swing, knee flexion at heel-strike, maximum knee flexion during loading response (mid stance), maximum tibial internal/external rotation during the gait cycle, maximum tibial adduction/abduction during the gait cycle, cadence, and average gait velocity.

We found statistical significant differences only for the internal/external tibial rotation variable. Specifically, a significant difference was found in maximum tibial rotation angle in the ACL-deficient group compared with the ACL-reconstructed group and the control group. The mean value of this variable in the ACL-deficient group was $9.6^{\circ} \pm 8.66^{\circ}$ of internal rotation. In the ACL-reconstructed and control groups, the mean value was $0.3^{\circ} \pm 9.9^{\circ}$ of external rotation and $3.6^{\circ} \pm 6.22^{\circ}$ of external rotation, respectively. The tibial rotation angle during swing reached its maximum value during the initial swing phase in all groups (Fig. 3).





Therefore, our results demonstrated that during walking, ACL-deficient knees exhibit internal tibial rotation, which is excessive and statistically significant in the initial swing phase. This increased rotation seems to be restored to normal values after ACL reconstruction. Therefore, it seems that ACL reconstruction might contribute to the prevention of future degenerative changes (ie, meniscal damage) through protection against repeated episodes of rotational instability during low-demanding activities. However, further investigation of this matter is definitely required.

In an attempt to further illuminate this issue, we studied tibial rotation in ACL-reconstructed patients during high-stress activities (descending stairs and subsequent pivoting).⁴⁰ For that purpose, we examined 18 patients with ACL-reconstructed knees and 15 controls. ACL reconstruction was performed arthroscopically with autologous BPTB. The evaluation was performed at an average of 10 months after reconstruction. Clinically (Lachman test, pivot shift test, and KT-1000 measurements), knee joint stability was regained in all patients. The patients were asked to descend three steps, pivot (externally rotate) on the landing leg at 90°, and walk away from the stairway (Fig. 4). Data collection was performed with our six-camera optoelectronic system. Reflective markers were placed on both lower limbs according to the model described by Davis. We examined the maximum range of motion of tibial rotation during the pivoting period.

Statistical significant differences were found between the ACL-reconstructed and the contralateral intact leg within the ACL-reconstructed group. Specifically, we found that the maximum range of motion of tibial internal/external rotation during the pivoting period in the ACL-reconstructed group was 22.60° \pm 2.85° for the ACL-reconstructed knees, whereas it was 18.97° \pm 4.31° for the intact knees in the same group (Fig. 5). On the other hand, no significant differences were found between the healthy leg of the control group and the intact leg of the ACL-reconstructed group. Therefore, our results demonstrated that tibial rotation remained a problem during pivoting activities 1 year after ACL reconstruction.







Figure 4 A stick figure describing the task. The subjects descend three steps at their own pace. The descending period is concluded on initial foot contact with the ground. After foot contact, the subjects pivot (externally rotate)

on the landing leg at 90° and walk away from the stairway. While pivoting, the contralateral leg swings around the body and the trunk is oriented perpendicularly to the stairway.



Figure 5 Group mean and standard deviation values for maximum range of motion of the tibial internal/external rotation during the pivoting period. An asterisk (*) indicates significant difference within the ACL-reconstructed group.

The tibial rotational values reported in our study are in close agreement with the in vitro study of Loh et al.⁴⁰ In that study, a rotational load was applied on ACL-reconstructed cadaveric knees; tibial rotation was reported to be 16.7° (\pm 9.9°) and 22.8° (\pm 12.6°) when the knee was placed at 15° and 30° flexion angles, respectively. With intact cadaveric knees, they found these values to be 16.1° (\pm 8.3°) and 20.6° (\pm 11.1°).

These results are also confirmed by other studies. Andriacchi et al³⁴ demonstrated that although successfully ACL-reconstructed patients displayed no abnormality during low-demanding activities, persistent adaptations were present during high-demanding activities (ie, jogging, pivoting). This finding is also supported by in vitro studies. Specifically, it has been indicated that tibial translation is restored after ACL reconstruction, but tibial rotation is not improved.⁴¹⁻⁴³

In a cadaveric study, Woo et al⁴³ demonstrated that the current reconstruction procedures with single (BPTB) or multiple (semitendinosus and gracilis tendon) grafts are effective in restoring anterior tibial translation but fail to reduce the coupled anterior tibial translation in response to a combined valgus and internal tibial torque. Furthermore, Brandsson et al,12 who examined in vivo tibial rotation in patients with ACL rupture before and after ACL reconstruction, demonstrated analogous results.

There are many hypotheses as to why this phenomenon occurs. The ACL is composed of two major bundles—the anteromedial and posterolateral. Each bundle contributes to different aspects of stability and stresses.^{44,45} In a healthy ACL, each bundle has a different tension pattern; there seems to be functional cooperation between the two.⁴⁴ In vitro studies have shown that the posterolateral bundle plays a major functional role when the knee undergoes extension,^{46,47} whereas forces in the anteromedial bundle are relatively constant throughout flexion/extension. Such anatomic complexity of the ACL is difficult to reproduce with current ACL-reconstruction procedures.

This seems to be the most probable explanation for the lack of restoration of tibial rotation to normal levels. Most ACL-reconstruction procedures have focused only on replacing the anteromedial bundle^{46,48,49}; the other functional bundle—the posterolateral— has not received sufficient attention. Recently, reports have been published that describe the outcomes of more anatomically correct ACL reconstructions designed to reconstruct both ACL bundles.^{47,50}

For single-bundle procedures, the preferred placement of the femoral tunnel in current reconstruction procedures is at the 11-o'clock position, which is designed to reproduce mostly the anteromedial bundle. Thus, because the graft is placed near the center of rotation of the knee joint, it may be unable to resist rotatory loads because of lack of a moment arm. This is why most surgeons tend to use a more lateral femoral tunnel (closer to the 10-o'clock position) to increase the moment arm.⁵¹

A two-bundle graft sounds like a better solution, because it seems to better simulate the morphology of the original ACL.^{47,49} However, few studies with this technique are reported in the literature. Muneta et al⁵⁰ reported the clinical results of a 2-year follow-up of a two-bundle procedure in 54 patients and demonstrated good anterior stability with no serious complications. Using a robotic system, Yagi et al⁴⁷ demonstrated very good biomechanical results with an anatomic reconstruction procedure with two bundles. Theoretically, a two-bundle

reconstruction has several advantages over a single-bundle reconstruction with respect to regaining a structure that morphologically and functionally more closely resembles a normal ACL. During the last few years, many orthopaedic surgeons have started performing two-bundle reconstructions. This technique, however, has not been investigated dynamically, and future research work with external loading conditions should be performed to determine the advantages of two-bundle anatomic reconstruction.

Conclusions

Through 3D kinematic analysis, which enables us to objectively evaluate the functional levels of the knee, it has been found that in the ACL-deficient knee there is anterior tibial translation and excessive tibial rotation during everyday activities. ACL reconstruction is successful in restoring these functions when low-demanding activities such as walking are performed. However, during high-demanding activities, ACL reconstruction seems to fail to restore excessive tibial rotation, which may be the cause of further degeneration in the medial compartment even after ACL reconstruction.

This could be due to the nature of the current graft, which cannot imitate the anatomy and function of the ACL. Reconstruction techniques should become more anatomic and try to approximate both ACL bundles. Moreover, the improvement and development of new surgical procedures and grafts should also contribute to restoring not only the pathological anterior drawer, but also the increased tibial rotation. Long-term follow-up studies need to be performed that focus on the advantages and disadvantages of different surgical procedures, whether it is the graft material or the tunnel positioning, keeping always in mind the importance of reproducing the actual ACL anatomy during the reconstruction.

References

- 1. Markolf KL, Burchfield DM, Sapiro MM, et al: Combined knee loading states that generate high anterior cruciate ligament forces. J Orthop Res 13:930-935, 1995
- 2. Tsepis E, Giakas G, Vagenas G, et al: Frequency content asymmetry of the isokinetic curve between ACL deficient and healthy knee. J Biomech 37:857-864, 2004
- 3. Tsepis E, Vagenas G, Giakas G, et al: Hamstrings weakness as an indicator of poor knee function in ACL deficient patients. Knee Surg Sports Traumatol Arthrosc 12:22-29, 2004
- 4. Papadonikolakis A, Cooper L, Stergiou N, et al: Compensatory mechanisms in anterior cruciate ligament deficiency. Knee Surg Sports Traumatol Arthrosc 11:235-243, 2003
- 5. Stergiou N, Moraiti C, Giakas G, et al: The effect of the walking speed on the stability of the anterior cruciate ligament deficient knee. Clin Biomech 19:957-963, 2004
- 6. Mankin HJ: The response of articular cartilage to mechanical injury [current concepts]. J Bone Joint Surg Am 64A:460-466, 1982
- 7. Finsterbush A, Frankl U, Matan Y, et al: Secondary damage to the knee after isolated injury of the anterior cruciate ligament. Am J Sports Med 18:475-479, 1990
- McDaniel WJ, Dameron TJ: The untreated anterior cruciate ligament rupture. Clin Orthop 172:158-163, 1983
- Noyes F, Matthews D, Mooar P, et al: The symptomatic anterior cruciate-deficient knee. Part II: the results of rehabilitation, activity modification, and counseling on functional disability. J Bone Joint Surg Am 65:163-174, 1983
- 10. Noyes F, Mooar P, Matthews D, et al: The symptomatic anterior cruciate-deficient knee. Part I: the long-term functional disability in athletically active individuals. J Bone Joint Surg Am 65-A:154-162, 1983
- 11. Eastlack ME, Axe MJ, Snyder-Mackler L: Laxity, instability and functional outcome after ACL injury: copers versus noncopers. Med Sci Sports Exerc 31:210-215, 1999
- Brandsson S, Karlsson J, Sward L, et al: Kinematics and laxity of the knee joint after anterior cruciate ligament reconstruction: pre- and postoperative radiostereometric studies. Am J Sports Med 30:361-367, 2002
- 13. Brandsson S, Karlsson J, Eriksson BI, et al: Kinematics after tear in the anterior cruciate ligament: dynamic bilateral radiostereometric studies in 11 patients. Acta Orthop Scand 72:372-378, 2001
- 14. Jonsson H, Karrholm J, Elmqvist LG: Kinematics of active knee extension after tear of the anterior cruciate ligament. Am J Sports Med 17: 796-802, 1989
- 15. Zhang LQ, Shiavi RG, Limbird TJ, et al: Six degrees-of-freedom kinematics of ACL deficient knees during locomotion—compensatory mechanism. Gait Posture 17:34-42, 2003
- 16. Li XM, Liu B, Deng B, et al: Normal six-degree-of-freedom motions of knee joint during level walking. J Biomech Eng 118:258-261, 1996
- 17. Marans HJ, Jackson RW, Glossop ND, et al: Anterior cruciate ligament insufficiency: a dynamic threedimensional motion analysis. Am J Sports Med 17:325-332, 1989
- 18. Lafortune MA, Cavanagh PR, Sommer HJ, et al: Three-dimensional kinematics of the human knee during walking. J Biomech 25:347-357, 1992

- 19. Ishii Y, Terajima K, Terashima S, et al: Three-dimensional kinematics of the human knee with intracortical pin fixation. Clin Orthop 343:144- 150, 1997
- 20. Davis R, Ounpuu S, Tyburski D, et al: A gait analysis data collection and reduction technique. Hum Mov Sci 10:575-587, 1991
- 21. Reinschmidt C, van den Bogert AJ, Nigg BM, et al: Effect of skin movement on the analysis of skeletal knee joint motion during running. J Biomech 30:729-732, 1997
- 22. Lafortune MA: Three-dimensional acceleration of the tibia during walking and running. J Biomech 24:877-886, 1991
- 23. Cappozzo A, Catani F, Leardini A, et al: Position and orientation in space of bones during movement: experimental artefacts. Clin Biomech 11:90-100, 1996
- 24. Andriacchi T, Birac D: Functional testing in the anterior cruciate ligament-deficient knee. Clin Orthop 288:40-47, 1993
- 25. Beard DJ, Soundarapandian RS, O'Connor J, et al: Gait and electromyographic analysis of anterior cruciate ligament deficient subjects. Gait Posture 4:83-88, 1996
- 26. Patel RR, Hurwitz D, Andriacchi TP, et al: Mechanisms by which patients with anterior cruciate ligament deficiency generate the 'quadriceps avoidance gait'. Proceedings of the 21st Annual Meeting of the American Society of Biomechanics, 1996, pp 210-211
- 27. Patel RR, Hurwitz DE, Andriacchi TP, et al: Mechanisms for the 'quadriceps avoidance gait' seen in ACL deficient patients. Gait Posture 5:147, 1996
- 28. Wexler G, Hurwitz DE, Bush-Joseph CA, et al: Functional gait adaptations in patients with anterior cruciate ligament deficiency over time. Clin Orthop 348:166-175, 1998
- 29. Grood ES, Suntay WJ, Noyes FR, et al: Biomechanics of the knee ex- tension exercise. J Bone Joint Surg Am 66-A:725-734, 1984
- 30. Solomonow M, Baratta R, Zhou BH, et al: The synergistic action of the anterior cruciate ligament and thigh muscles in maintaining joint stability. Am J Sports Med 15:207-213, 1987
- 31. Berchuck M, Andriacchi TP, Bach BR, et al: Gait adaptations by patients who have a deficient anterior cruciate ligament. J Bone Joint Surg Am 72:871-877, 1990
- 32. Arms SW, Pope MH, Johnson RJ, et al: The biomechanics of anterior cruciate ligament rehabilitation and reconstruction. Am J Sports Med 12:8-18, 1984
- 33. Bulgheroni P, Bulgheroni MV, Andrini L, et al: Gait patterns after anterior cruciate ligament reconstruction. Knee Surg Sport Traum Arthr 5:14-21, 1997
- 34. Bush-Joseph CA, Hurwitz DE, Patel RR, et al: Dynamic function after anterior cruciate ligament reconstruction with autologous patellar tendon. Am J Sports Med 29:36-41, 2001
- 35. Devita P, Hortobagyi T, Barrier J: Gait biomechanics are not normal after anterior cruciate ligament reconstruction and accelerated rehabilitation. Med Sci Sports Exerc 30:1481-1488, 1998
- 36. Timoney JM, Inman WS, Quesada PM, et al: Return of normal gait patterns after anterior cruciate ligament reconstruction. Am J Sports Med 21:887-889, 1993
- 37. Karrholm J, Brandsson S, Freeman MA: Tibiofemoral movement 4: changes of axial tibial rotation caused by forced rotation at the weight-bearing knee studied by RSA. J Bone Joint Surg Br 82:1201-1203, 2000

- Georgoulis AD, Papadonikolakis A, Papageorgiou CD, et al: Three-dimensional tibiofemoral kinematics of the anterior cruciate ligament-deficient and reconstructed knee during walking. Am J Sports Med 31:75-79, 2003
- 39. Georgoulis AD, Papageorgiou CD, Makris CA, et al: Anterior cruciate ligament reconstruction with the press-fit technique. 2-5 years fol- lowed-up of 42 patients. Acta Orthop Scand Suppl 275:42-45, 1997
- 40. Ristanis S, Giakas G, Papageorgiou CD, et al: The effects of anterior cruciate ligament reconstruction on tibial rotation during pivoting after descending stairs. Knee Surg Sports Traumatol Arthrosc 11:360-365, 2003
- 41. Loh JC, Fukuda Y, Tsuda E, et al: Knee stability and graft function following anterior cruciate ligament reconstruction: comparison between 11 o'clock and 10 o'clock femoral tunnel placement. Arthroscopy 19:297-304, 2003
- 42. Kanamori A, Woo SL, Ma CB, et al: The forces in the anterior cruciate ligament and knee kinematics during a simulated pivot shift test: a human cadaveric study using robotic technology. Arthroscopy 16:633-639, 2000
- 43. Woo SL, Kanamori A, Zeminski J, et al: The effectiveness of reconstruction of the anterior cruciate ligament with hamstrings and patellar tendon. A cadaveric study comparing anterior tibial and rotational loads. J Bone Joint Surg Am 84A:907-914, 2002
- 44. Girgis FG, Marshall JL, Monajem A: The cruciate ligaments of the knee joint. Anatomical, functional and experimental analysis. Clin Orthop 106:216-231, 1975
- 45. Odensten M, Gillquist J: Functional anatomy of the anterior cruciate ligament and a rationale for reconstruction. J Bone Joint Surg Am 672: 257-262, 1985
- 46. Sakane M, Fox RJ, Woo SL, et al: In situ forces in the anterior cruciate ligament and its bundles in response to anterior tibial loads. J Orthop Res 15:285-293, 1997
- 47. Yagi M, Wong EK, Kanamori A, et al: Biomechanical analysis of an anatomic anterior cruciate ligament reconstruction. Am J Sports Med 30:660-666, 2002
- 48. Fu FH, Bennett CH, Ma CB, et al: Current trends in anterior cruciate ligament reconstruction. Part II. Operative procedures and clinical correlations. Am J Sports Med 28:124-130, 2000
- 49. Hara K, Kubo T, Suginoshita T, et al: Reconstruction of the anterior cruciate ligament using a double bundle. Arthroscopy 16:860-864, 2000
- Muneta T, Sekiya I, Yagishita K, et al: Two-bundle reconstruction of the anterior cruciate ligament using semitendinosus tendon with endobuttons: operative technique and preliminary results. Arthroscopy 15: 618-624, 1999
- 51. Steadman JR, Mair SD: Anterior cruciate ligament reconstruction with bone-patellar tendon-bone autograft two-incision technique. Oper Tech Orthop 9:273-280, 1999