Motor control after anterior cruciate ligament reconstruction

Gokeler, Alli

Chapter 2

The Relationship between Isokinetic Quadriceps Strength and Laxity on Gait Analysis Parameters in ACL Reconstructed Knees

A. Gokeler, T. Schmalz, E. Knopf, J. Freiwald, S. Blumentritt
Knee Surg Sports Traumatol Arthrosc 2003; 11(6):372–378

ABSTRACT

Gait alterations after anterior cruciate ligament (ACL) reconstruction have been reported in the literature. In the current study, a group of 14 patients who all had an ACL-reconstruction (ACLR) with a patellar tendon autograft were examined. Kinetic and kinematic data were obtained from the knee during walking. The Flexion-Extension-Deficit (FED) calculated from the angular difference between maximal flexion and maximal extension during the stance phase in the ACLR and the normal knee was measured. We investigated whether these alterations in gait are related to quadriceps strength and residual laxity of the knee. It may be that patients modify their gait patterns to protect the knee from excessive anterior translation of the tibia by reducing the amount of extension during stance. On the other hand, persistent quadriceps weakness may also cause changes in gait patterns as the quadriceps is functioning as an important dynamic stabilizer of the knee during stance. Results showed that patients had a significantly higher FED value of 4.9 ± 4.0 when compared to data obtained from a healthy control group (CTRL) in a previous stud (FED 1.3 ± 0.9). This is mainly caused by an extension deficit during mid stance. External extension moments of the knee were significantly lower in the ACLR group -0.27 ± 0.19 T_{ZMAX} Nm/kg when compared to a CTRL group -0.08 ± 0.06 T_{ZMAX} Nm/kg. Correlation coefficient analysis did not show any positive relationship between quadriceps strength and gait analysis parameters. Furthermore no correlation was found between the amount of laxity of the knee and gait. The relevance of this study lies in the fact that apparently the measured gait alterations cannot be solely explained by often used biomechanical indicators such as laxity and strength. Possibly, the measured gait alterations are a result of the surgical procedure with subsequent modified motor programming.

Key words: ACL, Gait analysis, Isokinetic strength, Neuromuscular, Rehabilitation

INTRODUCTION

Anterior cruciate ligament-reconstruction (ACLR) has become a routine surgical procedure in the last 15 years. Since the early nineties more aggressive rehabilitation programs have been advocated including immediate full knee extension, weight bearing as tolerated and early initiation of closed chain exercises emphasizing quadriceps strengthening.[1] Subsequently coordinative exercises are implemented with the goal of return to sports at four-six months after surgery. Several quantitative tests are described in the literature such as arthrometric knee laxity testing[1-3] and isokinetic strength testing[4-6] to evaluate the outcome of these surgical procedures. It has been demonstrated that laxity tests may not necessarily provide information about the functional status of the knee.[7] Furthermore, it is commonly accepted that return of a strong quadriceps muscle after knee injuries is vital for functional and athletic use of the lower extremity[8-11] although others did not observe this correlation.[12,13] Reports about isokinetic peak torque measurements taken approximately six months after ACLR and comparing the involved with the non-involved side show quadriceps ratios ranging from 59.5% to more then 90%.[5,6,14-17] Despite the differences reported, the consensus seems that quadriceps strength has not returned to normal levels at this time after surgery. This is interesting considering that most athletes are able to resume sports approximately six months after surgery.

We know from investigations performed at our gait laboratory[18] that a large percentage of patients show significant abnormalities during gait even at 26 weeks after ACLR, equivalent to the time period when most patients return to sports. In fact, the evidence from our study showed that the return of normal gait may even take more than one year. The most striking differences were an extension deficit and reduced external extension moments in the involved knee in the mid-stance phase of gait. The question arises as to the nature of different biomechanical strategies used – consciously or unconsciously - by patients after ACLR. It may be that patients modify their gait patterns to protect the knee from excessive anterior translation of the tibia by reducing the amount of extension during stance. On the other hand persistent quadriceps weakness may also cause changes in gait patterns as the quadriceps is functioning as an important dynamic stabilizer of the knee during stance.

The purpose of this study was to determine whether gait alterations were present in patients whose ACL-deficient (ACLD) knees were surgically reconstructed with a patellar tendon autograft, and in that case, whether that had a relationship with residual laxity and quadriceps strength. We chose to take the measurements 26 weeks after surgery as we know from a previous study that kinetic and kinematic characteristics of gait are still significantly different from controls.[19]

MATERIAL AND METHODS

Subjects

Fourteen subjects (7 men and 7 women) with a mean age of 24 years (range 21-40), mean height 183 cm (range 162-192) and a mean weight of 74.4 kg (range 56-101) participated in this study. All had a complete rupture of the ACL that was arthroscopically reconstructed using the central third of the patellar tendon. All patients participating in the study were collegiate or recreational athletes. After surgery, they completed an intensive rehabilitation program as outpatients three times a week at the same rehabilitation center. The program included immediate weight bearing, range of motion exercises, pool therapy, stationary bicycle and closed chain strengthening and coordination exercises. Running was permitted after 10 weeks and once dynamic stability was satisfactory, agility and sports specific exercises were started. Return to sports involving pivoting and jumping was allowed after six months. Patients gave their consent to participate in this study.

Experimental Design

Clinical examination

All patients were examined by the same two physical therapists with respectively ten and eight years experience in orthopedics. The examination consisted of passive range of motion measurements of both knees for knee extension and flexion with a standard goniometer and instrumented laxity testing using the KT-1000 arthrometer (MEDmetric Corp., San Diego, Cal. USA) tests with application of a 89-N force. Side-to-side differences (in mm) were reported for comparison.

Isokinetic testing

Muscular performance of both knees was evaluated on an isokinetic testing device (Lido Active, Loredon Biomedical Inc., Davis, CA) of both knees at a velocity of 60 deg/sec. All patients had two-three training sessions on the isokinetic device in the weeks prior to testing to familiarize them with the testing procedure. The subjects did a 15 minute warm-up on a stationary bicycle (Kardiomed Bike, Proxomed, Karlstein, Germany) before the test procedure. Testing was done with the subjects in a seated position with the hip in 90° flexion and the thigh fixated with straps. The ROM for the knee was set at 0° to 90° flexion. The noninvolved side was tested first. Prior to testing 10 sub-maximal repetitions were performed. The test procedure consisted of 10 maximal concentric repetitions for flexion and extension at a speed of 60 deg/sec. The patients received standardized verbal commands but visual information from the curves as displaced on the monitor was withheld. The peak torque of quadriceps and hamstring strength was compared with the noninvolved side and was expressed as a ratio (involved torque/ noninvolved torque x 100).

Gait analysis

Gait analysis was performed for level walking at our gait laboratory using a 4-camera optoelectric system (Primas, Delft Motion Analysis, Delft, the Netherlands) with a 100 Hz frequency for collection of the 2-dimensional data. Reflective markers were placed on the subjects at anatomic landmarks according to the description in a previous paper.[18] The markers were placed at the greater trochanter, lateral femoral epicondyle, lateral malleolus and on the outside of the shoe representing the location of the head of the fifth metatarsal. Thus only sagittal plane motions could be calculated. Two force plates (Kistler Instruments, Winterthur, Switzerland) embedded in a 12 meter long walkway measured the ground reaction forces of both legs with a sampling rate of 400 Hz. The 2-dimensional data derived from the four cameras were synchronized with the collection of data from the force plates. All subjects were instructed to walk steadily during the test procedure. For each subject a specific starting point was determined from test trials so that the subject would contact the platform each time with the same limb without having to consciously focus to touch the plate. All subjects walked with sport shoes. The data used in this study were obtained from the mean values of 10-12 consistent cycles of walking over the walkway. Definitions of the quantitative parameters were described in detail in an earlier publication from our institution.[18] For the purpose of this study we will summarize the most important kinetic and kinematic parameters.
To describe the kinematic changes during the stance phase, we calculated the angular difference between maximal flexion and maximal extension in the ACLR and the normal knee. We defined this as the “Flexion-Extension-Deficit“ (FED) (Figure 1). The differentiation whether a significant FED-value is due to reduced flexion or extension motion during stance can be made with the calculation of joint toques. In 90% of the cases a higher value is associated with an extension deficit in stance.[18]

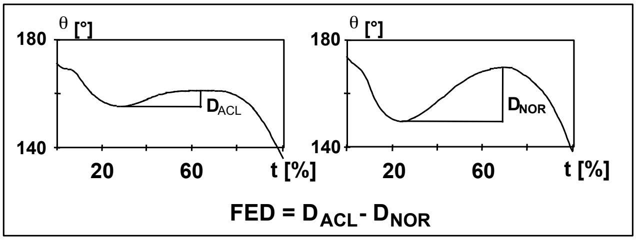

Figure 1. Sagittal knee angles (θ) during the stance phase (t expressed as percentage of stance phase) for the reconstructed knee (left) and normal knee (right). D_{ACL} Difference between maximal knee flexion an extension for the reconstructed knee; D_{NOR} difference between maximal knee flexion an extension for the normal knee; FED D_{ACL}–D_{NOR}

The external, sagittal moment acting on the knee joint was calculated from kinematic data and ground reaction forces. During normal human gait there is an external flexion moment in the first 50% of stance which is followed by an external extension moment in the second half. The difference between the maximal values of the external flexion moments comparing the ACLR knee with the normal knee is defined as T_{ZMIN} whereas the difference between maximal external extension moments is defined as T_{ZMAX} (Figure 2).

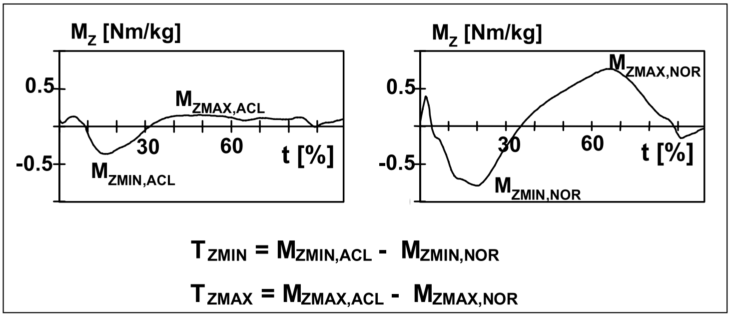

Figure 2. The sagittal knee moments during stance phase (t expressed as percentage of stance phase) normalized to body weight (M_Z). The external flexion (M_{ZMIN}) and extension (M_{ZMAX}) moments are shown for the ACL-reconstructed knee (left) and normal knee (right). The difference between the maximal values of the external flexion moments comparing the ACL-reconstructed knee with the normal knee is defined as T_{ZMIN} whereas the difference between maximal external extension moments is defined as $T_{ZMAX.}$

In this study we only calculated for T_{ZMAX} as this was shown to be a sensitive indicator of gait abnormalities.[18] All measurements were performed 26 weeks after surgery on all subjects.

Statistical analysis

Linear correlation coefficients were calculated with SPSS 10.0 for Windows to determine the relationship between isokinetic strength, laxity measurements and gait analysis.

RESULTS

Gait analysis

The mean value of FED in our patients during stance phase of gait was 4.9° ± 4.0 and was significantly different ($p < 0.01$) when compared to a control group in a previous study. (Figure 3). The mean external extension torque, T_{ZMAX} was - 0.27 ± 0.19 Nm/kg and is also significantly different ($p < 0.05$) when compared to controls (Figure 4).

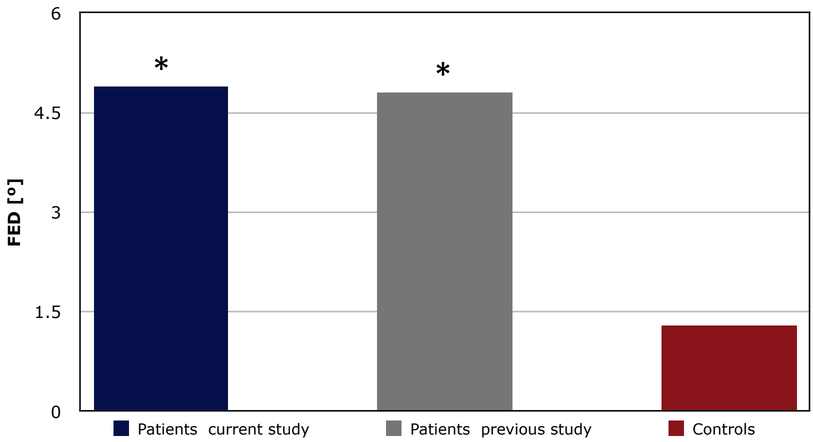

Figure 3. Kinematic flexion extension deficit FED for the current patient group in comparison to the earlier recorded data of a comparable patient group (n=35, mean age 27 years) and a healthy control group (n=30, mean age 28 years)[28] indicating significant difference (*) of the patients in comparison to the natural right-left-differences of uninjured people ($p < 0.01$).

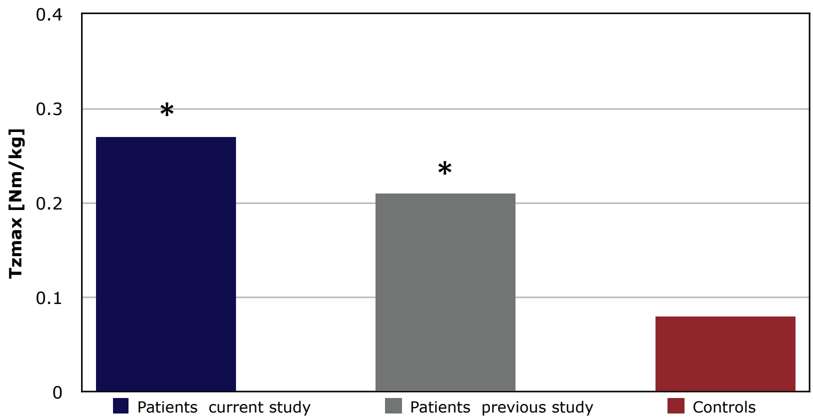

Figure 4. External extension moments T_{ZMAX} for the current patient group in comparison to the earlier recorded data of comparable patient group (n=35, mean age 27 years) and a healthy control group (n=30, mean age 28 years)[28] indicating significant difference (*) of the patients in comparison to the natural right-left-differences of uninjured people ($p < 0.05$).

Laxity examination and Isokinetic Strength

Laxity measurements with the KT-1000 with a 89N force showed a mean side to side difference of 2 ± 0.9 mm. The mean isokinetic quadriceps peak torque ratio at 60 deg/sec for the involved side was 74.9 ± 17.8 % of the non-involved side.

Correlation between Laxity, Isokinetic Strength and Gait Analysis

The linear correlation coefficients between clinical examination, isokinetic strength and gait analysis were calculated and are summarized in Table 1. A correlation exists between FED and T_{ZMAX} ($p < 0.05$). We did not find a correlation between laxity examination, isokinetic quadriceps torque and gait analysis parameters.

Table 1. Correlation coefficients (*r*) between the Gait Analysis Parameters FED and T_{ZMAX} and Isokinetic Quadriceps Peak Torque and Laxity

	T_{ZMAX}	KT-1000	Isokinetic Quadriceps Peak Torque
FED	0.56 (*)	0.005	0.33
T_{ZMAX}	X	0.19	0.24

(*: indicates statistically significant relationship p<=0.05)

We present 4 scatter diagrams: one showing the correlation between FED and isokinetic quadriceps peak torque (Figure 5), one showing the correlation between T_{ZMAX} and laxity (Figure 6), one showing the correlation between FED and laxity (Figure 7) and finally between T_{ZMAX} and isokinetic quadriceps strength (Figure 8).

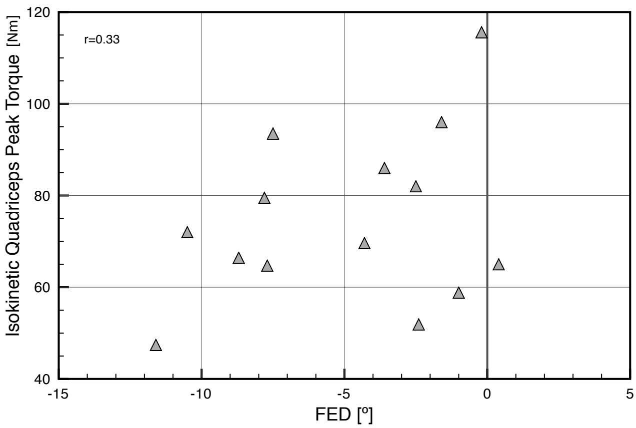

Figure 5. Correlation between the kinematic Flexion-Extension Deficit (FED) and Isokinetic Quadriceps Peak Torque in ACL-reconstructed knees.

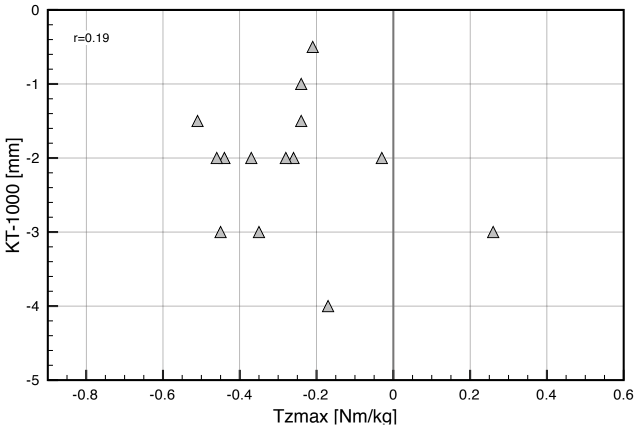

Figure 6. Correlation between T_{ZMAX} and Laxity in ACL-reconstructed knees.

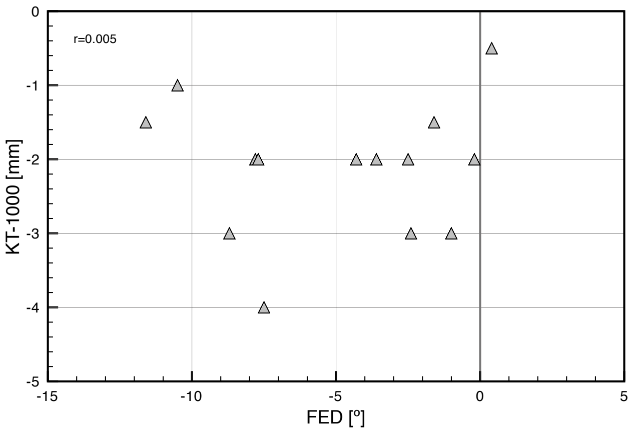

Figure 7. Correlation between FED and Laxity in ACL-reconstructed knees.

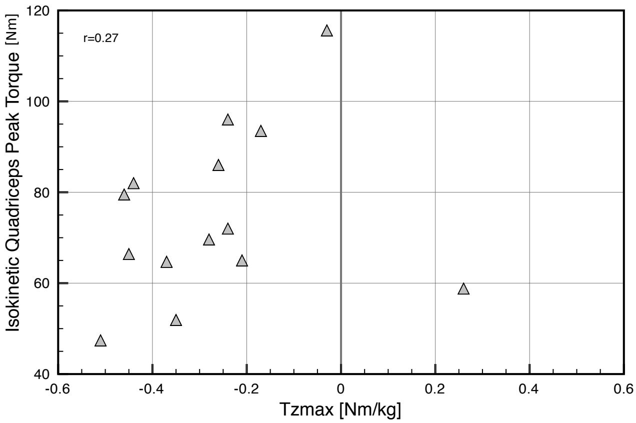

Figure 8. Correlation between T_{ZMAX} and Isokinetic Quadriceps Peak Torque in ACL-reconstructed knees.

DISCUSSION

The results from this study showed that typical kinetic and kinematic deficits are present after ACLR. The abnormalities mainly concern ROM, the extension motion of the knee, and the external extension moments acting on the knee joint during the stance phase of gait. In this context a statistical relationship exists between FED and T_{ZMAX}. In other words, we found that when FED reaches normal values it will implicate that the most important kinetic parameter expressed as T_{ZMAX} , will have also returned to normal values.[18] Neither FED nor T_{ZMAX} were related to quadriceps strength or laxity of the knee. The concept of gait modification in ACLD knees termed quadriceps avoidance as a strategy to reduce anterior displacement of the tibia is perhaps the one most popularized.[20,21] Others did not demonstrate the phenomenon of quadriceps avoidance.[18,19,22-24] Cicotti et al.[25] reported consistent EMG activity of the vastus lateralis muscle during gait in patients with ACLD knees when compared to controls. Our results concerning the absence of a significant relationship between laxity and gait analysis are in general agreement with Rudolph and co-workers.[26] They examined subjects with ACLD knees who were classified as copers and non-copers. The copers compensated well functionally for ACL injury compared to non-copers who were not able to stabilize their knees and were scheduled for surgery. They found that the amount of laxity in their subjects was unrelated to gait patterns. The aforementioned contradictions in the literature may be due to differences in methodology by which kinetic and kinematic data are calculated, examination of ACLD or ACLR knees, time after surgery, sample size and statistical analysis used.

Our study showed that six months after ACLR, patients had a mean isokinetic quadriceps peak torque ratio of 74.9% which is in proximity of values reported by others.[6,14,17] Our results showed no statistical relationships between isokinetic quadriceps strength and gait analysis parameters. Some researchers have found positive relationships between isokinetic quadriceps peak torque and functional performance[8,9,11] others found only a low or no correlation.[12,13,27,28] Several papers examining the effect of strength on gait analysis have been published. Snyder-Mackler and co-workers[10] studied 110 patients after ACLR and showed a relationship between isometric quadriceps strength and lower values of extension and flexion motion during the stance phase. In general the kinematic differences they reported are in agreement with our study, however in contrast to their findings, the differences we measured were unrelated to quadriceps strength. Lewek et al.[29] examined the relationship between isometric strength of the quadriceps on gait mechanics. They classified patients with ACLR knees in two groups of strong quadriceps with strength ratios > 90% and those with ratios < 80%. They found a significant relationship between strength and knee angles and moments during the early phase of stance. Mittlmeier and colleagues[30] found that weakness of the quadriceps measured isokinetically 24 weeks after ACLR was related to gait abnormalities. However they studied gait by assessing plantar pressure distribution which cannot calculate for joint moments as we did in our study. Rudolph et al.[26] did not find a correlation between isometric quadriceps strength and the amount of knee flexion during weight acceptance in subjects with ACL-deficient knees. It has to be noted that isokinetic testing usually involves maximal muscle activation whereas kinetic and kinematic parameters obtained during gait do not place maximal demands on the knee joint. This could be a possible explanation for the lack of relationship between isokinetic quadriceps strength and gait analysis parameters.

Several investigators[22,23,31] have described the dynamic stabilizing function of the hamstrings in ACLD knees. Less is known about the role of the hamstrings in a population with ACLR knees. Cicotti and co-workers[25] reported near normal activity of the hamstrings during the swing phase of gait in ACLR knees when compared to controls. Work at our own institution has shown that the activity of the gastrocnemius muscle is significantly reduced during the stance phase.[32]

Although improvements in surgical techniques and more aggressive rehabilitation programs have been implemented, several authors continue to report persistent deficits in quadriceps strength.[33-35] Engelhardt and co-workers showed that afferent signals from the central nervous system inhibit the activation of the quadriceps muscle after injury or surgery of the knee, causing the often observed atrophy of the quadriceps.[36] Freiwald and colleagues demonstrated that isokinetic torque of the quadriceps was significantly reduced 12 weeks after ACLR when compared to pre-operative measurements.[33,37] At 16 months after surgery the maximal isokinetic quadriceps ratio was 81% in comparison to the normal knee. Interestingly the patients had a Lysholm score > 95 points and had all resumed their pre-operative sports level. Recently, Keays et al. corroborated these findings.[6] They showed that an isokinetic peak torque ratio of the quadriceps of 88% before surgery and decreasing to 72% at six months after surgery despite intensive quadriceps training. Interestingly, functional tests improved by in the same time period. One may conclude that isokinetic quadriceps peak torque is not as important a predictor of function as initially thought. It may be that when a - so far undefined - “peak torque deficit” is crossed, subjective and objective limitations may become noticeable. From the perspective of the theories in motor learning it appears that reprogramming of the central nervous system after ACLR allows for improvement of functional tasks despite weakness of the quadriceps.[38] The clinical implication may be that primarily focusing on return of full quadriceps strength is no longer warranted and rehabilitation should rather implement goal-oriented exercises that replicate the functional demands as in sports or work.[38]

Several limitations have to be addressed about this paper. First, we had a relative small patient population. Second, the data derived can only be applied to patients who underwent the same surgical procedures as in our study population. Third, to the best of our knowledge, the external validity of gait analysis has has not been demonstrated to more athletic functional demands of the knee. The kinematic and kinetic data as measured in this study thus only applies to gait. Studying more strenuous activities such as running, jumping and cutting movements may provide more relevant information about the differences in kinetic and kinematic parameters necessary for sports related function of the knee. They could then be used as indicators of a safe return to sports after ACLR-reconstruction.
Our study clearly indicates that gait analysis parameters in ACLR knees are not related to quadriceps strength and laxity. Central reprogramming of the central nervous system[38] may be the reason why gait is significantly altered after surgical reconstruction of the ACL[39] as these changes cannot be fully explained by quadriceps weakness and laxity of the knee.

Acknowledgments

Otto Bock Research Department, Biomechanics Laboratory, Göttingen, Germany

Declaration

We followed the principles outlined in the Declaration of Helsinki and the experiment complied with the law in Germany. The subjects were free to withdraw from the study at any time.

REFERENCES

1. Shelbourne KD, Nitz P. Accelerated rehabilitation after anterior cruciate ligament reconstruction. *Am J Sports Med.* 1990;18(3):292-299.
2. Bach BR, Jr., Jones GT, Hager CA, Sweet FA, Luergans S. Arthrometric results of arthroscopically assisted anterior cruciate ligament reconstruction using autograft patellar tendon substitution. *Am J Sports Med* 1995;23(2):179-185.
3. Daniel DM, Malcom LL, Losse G, Stone ML, Sachs R, Burks R. Instrumented measurement of anterior laxity of the knee. *J Bone Joint Surg Am.* 1985;67(5):720-726.
4. Witvrouw E, Bellemans J, Verdonk R, Cambier D, Coorevits P, Almqvist F. Patellar tendon vs. doubled semitendinosus and gracilis tendon for anterior cruciate ligament reconstruction. *Int.Orthop.* 2001;25(5):308-311.
5. Carter TR, Edinger S. Isokinetic evaluation of anterior cruciate ligament reconstruction: hamstring versus patellar tendon. *Arthroscopy.* 1999;15(2):169-172.
6. Keays SL, Bullock-Saxton J, Keays AC. Strength and function before and after anterior cruciate ligament reconstruction. *Clin Orthop Relat Res.* 2000(373):174-183.
7. Snyder-Mackler L, Fitzgerald GK, Bartolozzi AR, 3rd, Ciccotti MG. The relationship between passive joint laxity and functional outcome after anterior cruciate ligament injury. *Am J Sports Med.* 1997;25(2):191-195.
8. Barber SD, Noyes FR, Mangine RE, McCloskey JW, Hartman W. Quantitative assessment of functional limitations in normal and anterior cruciate ligament-deficient knees. *Clin Orthop Relat Res.* 1990(255):204-214.
9. Noyes FR, Barber SD, Mangine RE. Abnormal lower limb symmetry determined by function hop tests after anterior cruciate ligament rupture. *Am J Sports Med.* 1991;19(5):513-518.
10. Snyder-Mackler L, Delitto A, Bailey SL, Stralka SW. Strength of the quadriceps femoris muscle and functional recovery after reconstruction of the anterior cruciate ligament. A prospective, randomized clinical trial of electrical stimulation. *J Bone Joint Surg Am.* 1995;77(8):1166-1173.
11. Karlsson J, Kalebo P, Goksor LA, Thomee R, Sward L. Partial rupture of the patellar ligament. *Am J Sports Med.* 1992;20(4):390-395.
12. Anderson MA, Gieck JH, Perrin DH, Weltman A, Rutt RA, Denegar CR. The Relationships among Isometric, Isotonic, and Isokinetic Concentric and Eccentric Quadriceps and Hamstring Force and Three Components of Athletic Performance. *J Orthop Sports Phys Ther.* 1991;14(3):114-120.
13. Delitto A, Irrgang JJ, Harner CD, Fu FH. Relationship of Isokinetic Quadriceps Peak Torque and Work to One Legged Hop and Vertical Jump in ACL Reconstructed Knees. *Phys Ther.* 1993;73(6):S85.
14. Shelbourne KD, Foulk DA. Timing of surgery in acute anterior cruciate ligament tears on the return of quadriceps muscle strength after reconstruction using an autogenous patellar tendon graft. *Am J Sports Med.* 1995;23(6):686-689.
15. Wilk KE, Romaniello WT, Soscia SM, Arrigo CA, Andrews JR. The relationship between subjective knee scores, isokinetic testing, and functional testing in the ACL-reconstructed knee. *J Orthop Sports Phys Ther.* 1994;20(2):60-73.
16. Wilk KE, Keirns MA, Andrews JR, Clancy WG, Arrigo CA, Erber DJ. Anterior cruciate ligament reconstruction rehabilitation: a six-month followup of isokinetic testing in recreational athletes. *Isokinet Exc Sci.* 1991;1(1):36.
17. Wilk KE, Andrews JR. Current concepts in the treatment of anterior cruciate ligament disruption. *J Orthop Sports Phys Ther.* 1992;15(6):279-293.
18. Schmalz T, Blumentritt S, Wagner R, Gokeler A. Gait analysis of patients within one year after anterior cruciate ligament reconstruction. *Phys Med Reh Kurortmed.* 1998;8:1-8.
19. Schmalz T, Blumentritt S, Wagner R, Junge R. [Evaluation with biomechanical gait analysis of various treatment methods after rupture of the anterior cruciate ligament]. *Sportverletz Sportschaden.* 1998;12(4):131-137.

20. Andriacchi TP, Birac D. Functional testing in the anterior cruciate ligament-deficient knee. *Clin Orthop Relat Res.* Mar 1993(288):40-47.

21. Berchuck M, Andriacchi TP, Bach BR, Reider B. Gait adaptations by patients who have a deficient anterior cruciate ligament. *J Bone Joint Surg Am.* 1990;72(6):871-877.

22. Beard DJ, Soundarapandian RS, O'Connor JJ, Dodd CA. Gait and electromyographic analysis of anterior cruciate ligament deficient subjects. *Gait Posture.* 1996;4(2):83.

23. Roberts CS, Rash GS, Honaker JT, Wachowiak MP, Shaw JC. A deficient anterior cruciate ligament does not lead to quadriceps avoidance gait. *Gait Posture.* 1999;10(3):189-199.

24. Timoney JM, Inman WS, Quesada PM, et al. Return of normal gait patterns after anterior cruciate ligament reconstruction. *Am J Sports Med.* 1993;21(6):887-889.

25. Ciccotti MG, Kerlan RK, Perry J, Pink M. An electromyographic analysis of the knee during functional activities. II. The anterior cruciate ligament-deficient and -reconstructed profiles. *Am J Sports Med.* 1994;22(5):651-658.

26. Rudolph KS, Eastlack ME, Axe MJ, Snyder-Mackler L. 1998 Basmajian Student Award Paper: Movement patterns after anterior cruciate ligament injury: a comparison of patients who compensate well for the injury and those who require operative stabilization. *J Electromyogr Kinesiol.* 1998;8(6):349-362.

27. Sekiya I, Muneta T, Ogiuchi T, Yagishita K, Yamamoto H. Significance of the single-legged hop test to the anterior cruciate ligament-reconstructed knee in relation to muscle strength and anterior laxity. *Am J Sports Med.* 1998;26(3):384-388.

28. Kovaleski JE, Heitman RJ, Andrew DP, Gurchiek LR, Pearsall AW. Relationship between closed-linear-kinetic- and open-kinetic-chain isokinetic strength and lower extremity functional performance. *J Sport Reh.* 2001;10(3):196.

29. Lewek M, Rudolph K, Axe M, Snyder-Mackler L. The effect of insufficient quadriceps strength on gait after anterior cruciate ligament reconstruction. *Clin Biomech.* 2002;17(1):56-63.

30. Mittlmeier T, Weiler A, Sohn T, et al. Functional monitoring during rehabilitation following anterior cruciate ligament reconstruction. A novel Award Second Prize Paper. *Clin Biomech.* 1999;14(8):576-584.

31. Liu W, Maitland ME. The effect of hamstring muscle compensation for anterior laxity in the ACL-deficient knee during gait. *J Biomech.* 2000;33(7):871-879.

32. Schmalz T, Freiwald J, Greiwing A, Kocker L, Ludwig H, Blumentritt S. Mechanical and electromyographical gait parameters in the course of rehabilitation after anterior cruciate ligament reconstruction. *Eur J Sports Traumatol Relat Res* 2001;23(4):146-151.

33. Freiwald J, Jager A, Starker M. [EMG-assisted functional analysis within the scope of follow-up of arthroscopically managed injuries of the anterior cruciate ligament]. *Sportverletz Sportschaden.* 1993;7(3):122-128.

34. Yasuda K, Ohkoshi Y, Tanabe Y, Kaneda K. Quantitative evaluation of knee instability and muscle strength after anterior cruciate ligament reconstruction using patellar and quadriceps tendon. *Am J Sports Med.* 1992;20(4):471-475.

35. Natri A, Jarvinen M, Latvala K, Kannus P. Isokinetic muscle performance after anterior cruciate ligament surgery. Long-term results and outcome predicting factors after primary surgery and late-phase reconstruction. *Int J Sports Med.* 1996;17(3):223-228.

36. Engelhardt M, Reuter I, Freiwald J. Alterations of the neuromuscular system after knee injury. *Eur J Sports Traumatol Rel Res.* 2001;23 75-81.

37. Freiwald J, Reuter I, Engelhardt M. Neuromuscular and motor system alterations after knee trauma and knee surgery. A new paradigm. In: Lehmann L, ed. *Overload, Performance Incompetence and Regeneration in Sport.* New York: Kluwer Academic Press/Plenum Publishers; 1999:81-100.

38. Freiwald J, Engelhardt M. Status of Motor Learning and Coordination in Orthopedic Rehabilitation. *Sportorth Sporttraum* 2002;18:5-11.

39. Ferber R, Osternig LR, Woollacott MH, Wasielewski NJ, Lee JH. Gait mechanics in chronic ACL deficiency and subsequent repair. *Clin Biomech.* 2002;17(4):274-285.