
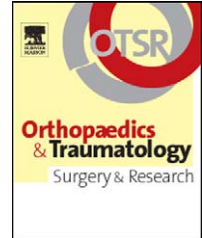




Available online at
 ScienceDirect
www.sciencedirect.com

Elsevier Masson France
 EM|consulte
www.em-consulte.com



REVIEW ARTICLE

ACL injury and reconstruction: Clinical related in vivo biomechanics

A.D. Georgoulis*, S. Ristanis, C.O. Moraiti, N. Paschos, F. Zampeli, S. Xergia, S. Georgiou, K. Patras, H.S. Vasiliadis, G. Mitsionis

Orthopaedic Sports Medicine Center of Ioannina, Department of Orthopaedic Surgery, University of Ioannina, Georgiou Papandreou 2, PO Box 1042, Ioannina 45110, Greece

KEYWORDS

ACL reconstruction;
Tibial rotation;
Electromechanical
delay;
Gait variability;
Muscle strength;
Knee biomechanics;
Sports performance

Summary Several researchers including our group have shown that knee joint biomechanics are impaired after anterior cruciate ligament (ACL) injury, in terms of kinematics and neuromuscular control. Current ACL reconstruction techniques do not seem to fully restore these adaptations. Our research has demonstrated that after ACL reconstruction, excessive tibial rotation is still present in high-demanding activities that involve both anterior and rotational loading of the knee. These findings seem to persist regardless of the autograft selection for the ACL reconstruction. Our results also suggest an impairment of neuromuscular control after ACL reconstruction, although muscle strength may have been reinstated. These abnormal biomechanical patterns may lead to loading of cartilage areas, which are not commonly loaded in the healthy knee and longitudinally can lead to osteoarthritis. Muscle imbalance can also influence patients' optimal sports performance exposing them to increased possibility of knee re-injury. In this review, our recommendations point towards further experimental work with in vivo and in vitro studies, in order to assist in the development of new surgical procedures that could possibly replicate more closely the natural ACL anatomy and prevent future knee pathology.
© 2010 Elsevier Masson SAS. All rights reserved.

Introduction

The anterior cruciate ligament (ACL) is composed of two functional bundles, the anteromedial (AM) and the posterolateral (PL) named by their tibial attachment [1–4]. Tension in the AM bundle increases with knee flexion, whereas the PL bundle takes up greater tension in extension and in response

to coupled internal rotation [5,6]. The ACL is the guide of the screw-home mechanism. This refers to an "automatic" type of axial rotation that is inevitably and involuntarily linked to movements of flexion and extension. When the knee is flexed, the tibia is internally rotated. As the knee is extended, the femoral condyles roll and glide on the tibial condyles, the tibia is gradually externally rotated and at full extension the knee joint "locks" presenting the maximal stability at the upright standing position. This screw-home mechanism is very important for the synchronization of the knee joint to the adjacent joints of the hip and the ankle. Although the principal movement of the knee

* Corresponding author. Tel./fax: +30 26510 64980.
E-mail address: georgoulis@osmci.gr (A.D. Georgoulis).

is flexion-extension, the internal-external rotation plays a very significant role especially in all these activities that include pivoting.

In the clinical setting, the anterior tibial translation is estimated with the Lachman-Noulis test, a reliable noninvasive diagnostic test for the ACL rupture. As Paessler H. and Michel D. reported [7], this test was originally described by George K. Noulis (1849–1919) in his doctoral thesis “*Entorse du genou*” which was defended at the University of Paris in 1875 [8]. This was perhaps the first biomechanical study on knee ligaments in cadavers.

Aristotle (384–322 BC) stated the principal idea that “every movement requires a cause”. This is the main core of interest of the in vivo biomechanics. The movement is studied with the kinematics and the cause is studied with the kinetics. Through its long history, in vivo biomechanics can nowadays be a valuable tool also for the arthroscopic surgeon. In recent years, important findings with clinical relevance have arisen from in vivo biomechanical studies and have improved our understanding for the ACL deficient and the ACL reconstructed knee and more interestingly has influenced even the way that we operate these patients. The current review article presents significant amount of knowledge regarding the in vivo biomechanics of an ACL-deficient patient, starting from the ACL rupture, including the ACL reconstruction and being completed with the return to sports and previous activity level.

ACL injury risk factors and prevention

In team sport settings, 50 to 80% of ACL injuries occur in non-contact situations [9–11]. The risk factors for a non-contact ACL injury can be divided into four categories: environmental, anatomical, hormonal and biomechanical–neuromuscular [12]. From a biomechanical perspective, ACL is loaded not only by extreme anterior translation, but also by both valgus and internal rotation moments. In fact, during landing and sidestep cutting tasks, anterior drawer load in isolation is probably not sufficient to injure the ACL and rather a loading combination on the three movement planes is needed to increase the likelihood of rupture. Besides that, when the knee flexion angle increases, there is a reduction in the resultant strain on the ACL [13–17].

The following biomechanical factors are not directly connected with the actual knee movement patterns, but also seem to play an important role on increasing the injury risk: decreased core stabilization and balance, low trunk and hip flexion angles and high ankle dorsiflexion when performing sport tasks. Furthermore, the combination of lateral trunk displacement with increased knee abduction moments and increased hip internal rotation with tibial external rotation exposes the ACL in high risk [13].

Neuromuscular deficiencies are also commonly observed in female athletes and have been classified in three categories: The ‘ligament dominance’ appears when an athlete absorb a significant portion of the ground reaction force during sports maneuvers with the knee ligaments, rather than the lower extremity musculature [18]. ‘Quadriceps dominance’ is the preferential activation of the knee extensors over the knee flexors during high-torque force movements

[19]. ‘Leg dominance’ is the side-to-side imbalance on strength and coordination between the dominant and the other leg, which may increase the risk for both limbs [19–21].

The prevention programs have focused on neuromuscular training methods to change the above-described modifiable biomechanical and neuromuscular risk factors and to reduce the non-contact ACL injury rates [22]. Most of these effective prevention studies included a combination of proprioceptive, neuromuscular and core balance training, plyometric, closed kinetic chain and other strengthening exercises, in order to modify the sport-specific movement patterns that lead to increased ACL injury risk.

More specific, the intervention programs focus on normalizing the landing technique [23], decreasing the valgus and internal/external rotation moments on cutting maneuvers [23] and increasing the hamstrings [19,24] and gluteal [25] muscles recruitment and strength.

Rupture pattern and injury mechanism of the ACL

ACL injury is very common during athletic performance compared to the incidence in the general population [26]. Recently, the interest on ACL failure has been increased since several studies highlight ACL injury as a risk factor for knee osteoarthritis regardless of ACL reconstruction [27,28]. During ACL injury, the most common symptoms include pain, audible pop, and oedema. The presence and importance of these signs in relation to isolated ACL injury have been evaluated in the past [29]. Several studies have contributed in better understanding of the biomechanical properties of the ACL like strength [30], stiffness [31] and tension patterns in relation to its failure properties [32].

A very interesting point is that a part of ACL fibers fail initially while the rest remain intact and have the ability to withstand load [32]. This condition could represent the partial ACL tear or the tear of only the AM or the PL bundle of the ACL. Three different patterns have been described in both in vitro and in vivo studies. All of these agree that the final pattern is related to the biomechanical features of the ACL and the mechanism of the injury [33,34].

Acute ACL rupture is accompanied in more than 80% by bone bruises, shown in MRI scanning. Spindler et al. [35] showed that 86% and 67% of the contusions involved the lateral femoral condyle (LFC) and lateral tibial plateau (LTP) respectively; in 56% of the cases, bruises at both sides occurred. Lesions in the medial femoral condyle (MFC) and medial tibia plateau (MTP) were less common (7% and 21% respectively). During the ACL injury, the tibia subluxes anteriorly and rotates internally subjecting the anterior parts of femoral condyles and posterior parts of tibial plateaus to direct contact. The excessive internal rotation of the tibia explains why the contusions of the LFC are usually more anterior than those seen on the MFC [36]. The axial and valgus force applied on the knee especially during contact injuries also plays an important role.

Clinical related in vivo biomechanics

Pathological internal-external rotation after ACL injury and reconstruction

During the last few years, the scientific community has given more attention to the role of the internal-external rotation of the knee joint. There is a lot of work in this area from very important centres [3,37–40] which have influenced significantly the reconstruction of the ACL. Our investigations have examined knee joint rotational movement patterns during high- and low-demanding activities in ACL reconstructed patients with a BPTB or a hamstrings graft, using gait analysis. In our first study, we examined 13 ACL-deficient patients, 21 BPTB reconstructed patients and 10 healthy matched controls during walking [41]. We found that during this low-demanding activity, the surgical reconstruction with a BPTB graft restores tibial rotation to normal levels. Next, we wanted to identify if this was also the case in a higher demanding activity, like descending stairs and subsequent pivoting [42] (Fig. 1). We evaluated 18 BPTB reconstructed patients 12 months postoperatively and 15 controls. We found that the tibial rotation during the pivoting period was significantly larger in the ACL reconstructed leg when was compared to the contralateral intact leg and the healthy control. To verify these findings, we examined 11 BPTB reconstructed, 11 ACL-deficient patients and 11

controls during an even higher demanding activity, like landing from a platform and subsequent pivoting (Fig. 2), which could apply increased rotational loading at the knee. We found no significant differences between the deficient leg of the ACL-deficient group and the reconstructed leg of the ACL reconstructed group [43]. In addition, both the reconstructed leg of the ACL reconstructed group and the deficient leg of the ACL-deficient group demonstrated significantly larger tibial rotation values than the healthy control.

Subsequently, we performed a follow-up evaluation [44] in nine BPTB reconstructed subjects that participated in this study [43] and examined them for both the aforementioned high-demanding activities [42,43]. We found that tibial rotation remained significantly excessive even 2 years after the reconstruction. Thus, we questioned whether the configuration and placement of the BPTB graft in the femur had an effect on rotational knee kinematics and we tried to identify if a more horizontal placement of the femoral tunnel can restore rotational kinematics, during these activities. We evaluated 10 patients BPTB reconstructed with the femoral tunnel in the 11-o'clock position and 10 patients with the femoral tunnel in the 10-o'clock position [45]. We noticed that positioning the tunnel at 10-o'clock resulted in slightly decreased rotation values that may have a clinical relevance but did not show a statistical significance.

Following our research work with the BPTB graft, it was logical to question if tibial rotation will remain excessive if

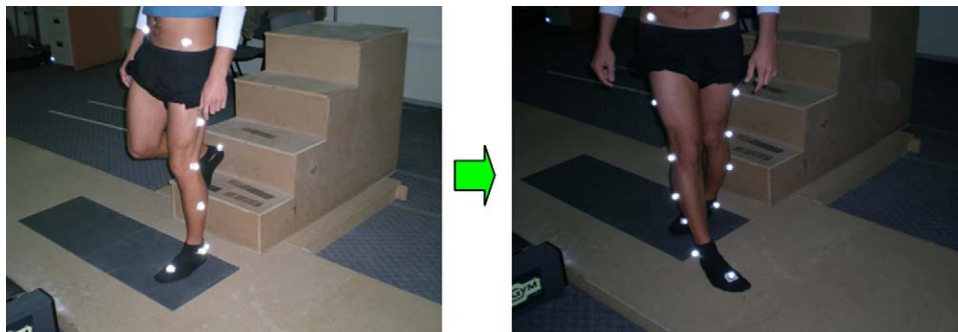


Figure 1 A patient descending the stairway. The descending period was concluded upon initial foot contact with the ground. Following foot contact, the subjects were instructed to pivot on the landing leg at 90 degrees and walk away from the stairway. While pivoting, the contralateral leg was swinging around the body (as it was coming down from the stairway) and the trunk was oriented perpendicularly to the stairway.

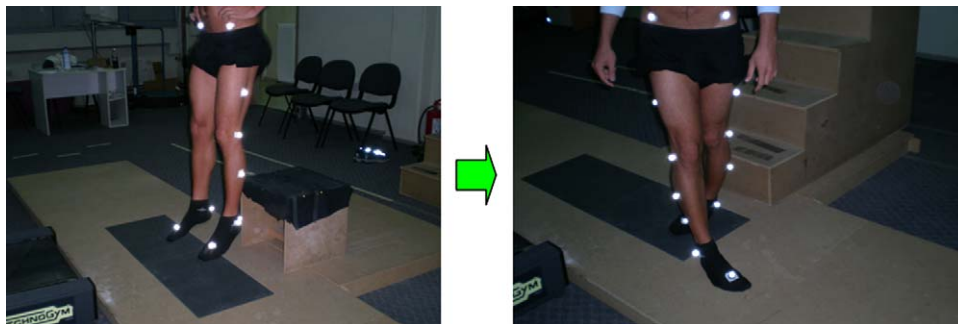


Figure 2 A patient jumping off the platform and land with both feet on the ground. Following foot contact, the subjects pivot (externally rotate) on the right or left (ipsilateral) leg at 90 degrees and walk away from the platform. While pivoting, the contralateral leg is swinging around the body and the trunk is oriented perpendicularly to the platform.

a quadrupled hamstrings graft (semitendinosus and gracilis; ST/G) was used. We examined 11 ST/G and 11 BPTB reconstructed patients along with 11 healthy controls [46], during landing with subsequent pivoting. We found significantly increased tibial rotation in both ACL reconstructed groups when compared with the healthy controls. To verify our findings, we performed an additional experiment where we evaluated 11 ST/G reconstructed patients, 9 months after the surgery and 11 healthy controls during descending and subsequent pivoting [47]. Our results also showed that the ST/G reconstruction did not restore excessive tibial rotation to normal healthy levels. We expected better results from the ST/G graft because it has been demonstrated in several studies [48] that this graft has mechanical properties similar to those of the ACL. It also provides a two-stripe replacement graft that may better approximate the function of the two-bundle ACL.

Our results were in agreement with recent dynamic radiostereometric analysis technique (dRSA) [49] and MRI [50] studies. Brandsson et al. [49] using a dRSA system found that one year after BPTB reconstruction, tibial rotation was not significantly different when compared with the preoperative measurements. In addition, Logan et al. [50] showed with an "open-access" MRI system and during a dynamic weight-bearing activity, that tibiofemoral kinematics are not restored in ACL reconstructed patients with hamstrings, even though sagittal laxity is restored to normative values.

In summary, our research work has showed increased rotation after ACL reconstruction in activities that are more demanding than walking and involve both anterior and rotational loading of the knee (pivoting). These findings persist regardless of autograft selection. Restore of tibial rotation is of great importance, as studies have shown [51] that sagittal plane knee joint forces cannot rupture the ACL during sidestep cutting, and valgus-rotational loading seems to be the most likely injury mechanism. In addition, excessive tibial rotation may cause abnormal loading of cartilage areas which are not commonly loaded in the healthy knee and longitudinally can lead to osteoarthritis [52,53]. The improvement and development of new surgical techniques, that can replicate better the actual anatomy of the natural ACL, like the double-bundle (DB) ACL reconstruction, seems to be a way to address the problem of excessive tibial rotation. Many colleagues have described the details of this technique [54–59]. Although it is a technically demand-

ing procedure, it seems promising towards the consensus effort of the complete restoration of abnormal tibial rotation. However, these new techniques should be rigorously evaluated in terms of in vivo biomechanics and relevant long term results are necessary to support its effectiveness.

Gait variability after ACL injury and reconstruction

Recently a new methodology has been developed which offers a more holistic approach concerning the study of gait. This approach is based on the fact that walking is a rhythmic activity. The legs continuously oscillate forward and backward generating movement. However, a closer examination reveals that each step is not identical to the previous or to the next one (Fig. 3). These variations that exist among subsequent strides are intrinsic deriving from the underlying mechanisms that produce gait [60,61].

Accordingly, variations exist in all human rhythmic functions (i.e. secretion of the hormones, bearing of the heart). Interestingly, the concept of studying variability has been first applied in domains such as endocrinology cardiology and neurology, providing useful information for the function of human body [62–64]. It is remarkable that in some cases changes in variability have been found to be predictive of subsequent clinical changes (i.e. epileptic seizure) [63]. Consequently, new theories have been proposed in order to assess and explain the function of human body [65,66].

Concerning human movement, Stergiou et al. [66] developed the "optimal amount of movement variability" proposition. According to this proposition, under healthy conditions, each motor task is characterized by an optimal amount of variability which provides the human body with flexibility, adaptability and the capacity to respond to unpredictable stimuli and stresses and environmental demands. The achievement of this optimal variability is desired during human motor development and motor learning. On the contrary, aging and disease are characterized by altered (either increased or decreased) variability and are associated with diminished flexibility and capability to respond to stimuli. This approach has been used to examine human gait after ACL rupture [67–69]. Actually the authors examined how knee flexion-extension changes over time over multiple footfalls.

It was observed that when compared to a healthy control knee, the ACL-deficient knee exhibits decreased gait

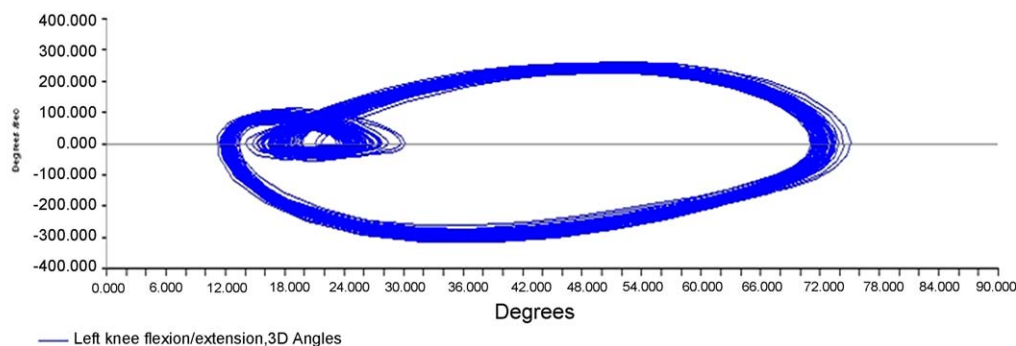


Figure 3 In this graph, knee flexion-extension is plotted versus angular velocity. Each trajectory corresponds to one gait cycle. Trajectories do not overlap revealing that variations exist from one stride to the next.

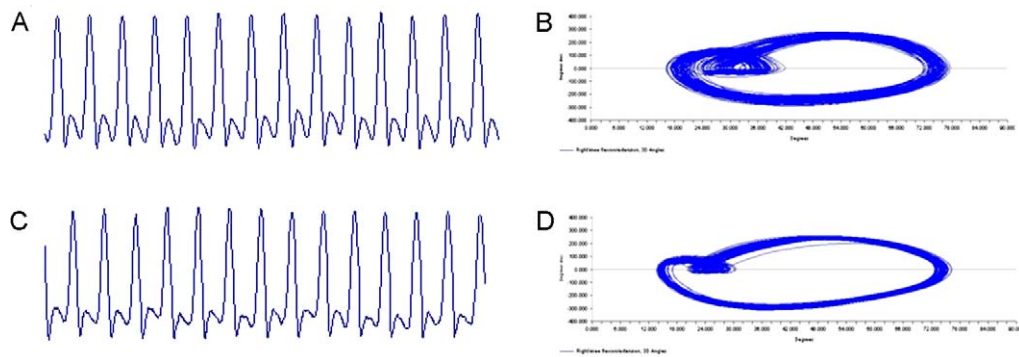


Figure 4 Schematic representation of the decreased gait variability encountered after ACL injury. A and C represent knee flexion-extension versus time over multiple footfalls for a healthy and an ACL-deficient knee, respectively. In the B and D graphs, the knee flexion-extension time series are plotted versus angular velocity. It can be noticed that there is more overlapping between the trajectories in the ACL-deficient knee, implying decreased gait variability.

variability [69]. This indicates that in the ACL-deficient knees, subsequent steps are more similar to each other than in healthy knees (Fig. 4). This could be due to the loss of the mechanical restraint or of the proprioceptive input provided by the ACL [70–74]. This decreased gait variability indicates that a patient with ACL deficiency is more “careful” in the way he or she walks in order to eliminate any extra movements, exhibiting an increased rigidity in movement patterns. According to the optimal amount of movement variability proposition [66], these changes are associated with decreased flexibility and responsiveness to environmental demands, leading possibly to injury and the development of future pathology.

It was assumed that ACL reconstruction could restore gait variability. However, studies have shown that gait variability is greater when compared to a healthy knee, 2 years after ACL reconstruction using either a BPTB or a hamstrings autograft, which indicates decreased flexibility and adaptability to stimuli and stresses [75,76]. This could be due to the altered muscle activity found in ACL reconstructed limbs, which may derive from the altered proprioceptive input [77,78]. Thus, the ACL reconstructed patients exhibit greater divergence in the movement trajectories. This could signify that someone who knows that the ACL is reconstructed feels “secure” to increase and add extra movement. However, because the innate proprioceptive channels are missing, gait variability and the function of the knee are not restored to normative levels.

In addition alterations in gait variability were found in the contralateral intact knee verifying the fact that adaptations do exist in both limbs ACL reconstruction also affected the structure of gait [76]. This supports previous studies that have identified bilateral lower extremity accommodations in gait biomechanics and muscular performance in ACL reconstructed patient [79]. It should be noted that although the typical clinical tests (i.e. Lysholm score, IKDC) were nearly normal in all cases, the study of gait variability showed that the proper function of the knee was not fully restored after ACL reconstruction. This indicates that this method is very sensitive and could prove to be very helpful in the assessment of various conditions as it has already been done in other medical domains.

Gait variability during backward walking

Gait variability can also be used to assess the outcome of rehabilitation protocols after ACL injury. Backward walking (BW) is part of rehabilitation programs [80]. Physical therapists use BW since this task was proved to strengthen the hamstring muscles [81,82] and this can improve the dynamic knee joint stability of ACL-deficient patients. Apart from rehabilitation, BW is used for injury prevention and training [83]. This is because many sports such as soccer, football, basketball and tennis incorporate backward locomotion during competition.

We investigated if ACL deficiency affects gait variability during BW by comparing a group of ACL-deficient patients to a matched control group of healthy individuals. Both knees of the ACL-deficient patients showed a more rigid walking pattern as compared to healthy controls. The contralateral intact knee of ACL-deficient patients showed even more rigid walking pattern as compared to the ACL-deficient knee. These data could imply diminished functional responsiveness to the environmental demands for both knees of ACL-deficient patients, which may result in knees more susceptible to injury.

The results for variability in ACL-deficient patients walking backward seem to be similar to the results presented for walking forward [69]. Therefore, the “rigidity” that was noticed both during forward and backward walking can be related to the ACL deficiency and indicates that these patients have less capability to respond to different perturbations and to adapt to the changing environment.

Neuromuscular control after ACL injury and reconstruction

Muscle strength after ACL injury and reconstruction

One of the most important adaptations after ACL injury is the thigh muscle strength deficit [84,85]. One of the most common used tools for a highly reliable single-joint evaluation is the isokinetic dynamometry [86–88]. The most common parameters that have been used in isokinetic eval-

uation are work per unit, isokinetic curves and mainly the peak torque value [88–90].

In a study of our lab it has been noticed that after ACL rupture quadriceps and hamstring strength deficits exist and there are numerous factors that are responsible for this [91]. After ACL rupture quadriceps muscle strength is decreased due to the loss of afferent reaction from the ACL to gamma motor neurons [92]. The ACL rupture can result in “quadriceps avoidance” gait patterns, which has been explained like a compensator mechanism for preventing anterior subluxation [93,94]. Hamstring muscle firing pattern changes, in an attempt to counteract these anterior shear forces. Specifically, it has been shown that after ACL rupture physiological modifications are developed with the intention to activate hamstrings for diminishing shear forces during knee loading [95]. Strength deficits after ACL rupture are time-dependent and quadriceps deficit persists longer than hamstrings’ [91].

Imbalance of the knee muscle strength, between different muscle groups, has also been identified after ACL reconstruction. Failure to prevent or to effectively treat this imbalance may alter the patient’s functional recovery [96] and predispose patients to re-rupture. Different strength deficits have been reported after different graft types [88,97], which complicate the decision-making in terms of either the type of graft to use or the focus of appropriate rehabilitation [98].

The achievement of muscle strength balance of lower extremities may be one of the most important factors for returning to pre-injury activity levels [96]. Except from the surgery itself, the muscular recovery after an ACL reconstruction may be affected by numerous factors. The preoperative muscle strength [99], the time between injury and reconstruction [100] and the pre- and post-surgery rehabilitation [101] are some of them.

Quadriceps strength weakness has been noticed after harvesting the BPTB autograft and hamstring muscle weakness after harvesting the HST autograft [97]. Muscle function may also be modified due to the attenuation of the gamma loop function caused from the ACL injury, which is not restored after the ACL reconstruction. It is documented that ACL mechanoreceptors play an important role in enhancing the activity of gamma motor neurons contributing to a normal muscle function [92]. It is of crucial importance that, before returning to sport activities after ACL reconstruction, muscle strength deficits have to be diminished. For that reason, rehabilitation after ACL reconstruction must focus on muscle strength recovery in conjunction with a plethora of criteria [96].

Neuromuscular control in terms of electromechanical delay (EMD) in ACL reconstructed patients

However, some researchers have suggested that the actual effectiveness of the muscles to provide mechanical response and protection under real-life situations can be revealed only with the measurement of the time delay between the onset of muscle stimulation by the alpha motoneuron to the development of torque at a given joint [102,103]. This is referred as electromechanical delay (EMD) [102]. Few

studies exist in the literature that documents the effect of ACL reconstruction with BPTB or hamstrings, on the donor-site muscle’s EMD. In our first study, we evaluated 17 ACLR patients with a BPTB autograft, 2 years after the reconstruction [104] under maximally explosive isometric contractions. We recorded the surface electromyographic (EMG) activity of rectus femoris (RF) and vastus medialis (VM). We found that harvesting the medial third of the patellar tendon did not significantly alter the EMD of the knee extensor muscles. In our second study [105], we evaluated 12 ACLR patients with an ST/G autograft and 12 healthy controls, using the same methodology and recording the ST and the biceps femoris (BF) muscles. Our statistical comparisons revealed significant increases of the EMD of the ACLR knee for both investigated muscles.

The EMD measurement is of great functional importance, because regardless of the contractile ability of the muscles, alterations in the EMD of the quadriceps or hamstrings muscle-tendon unit could compromise knee integrity or impair performance by modifying the transfer time of muscle tension to the bones. Our results suggested an impairment of neuromuscular control at the knee flexors after ACL surgery. However, further longitudinal investigation is required to identify how the EMD is tolerated by the central nervous system and if the increased hamstrings EMD can influence patients’ optimal sports performance exposing them to increased possibility of knee re-injury.

The influence of metabolic fatigue on neuromuscular function in ACL reconstructed athletes

ACL rupture is associated with altered neuromuscular function as revealed by diminished EMG activity of the quadriceps and increased or earlier EMG activity of the biceps femoris during walking and running [106,107]. It has been demonstrated that ACL reconstruction re-establishes EMG activation levels of the operated leg towards normative values during low demand activity such as walking and jogging [108,109]. However the effect of metabolic fatigue on EMG activation levels has not been tested. This is important because fatigue is considered to have a cumulative negative effect, resulting in hazardous movement strategies and that knee injuries tend to occur at the later stages of a game where there is accumulation of metabolic fatigue [110].

Our first study examined the effect of metabolic fatigue on EMG activation levels in ACL reconstructed amateur soccer players with a BPTB autograft and demonstrated that metabolic fatigue had a negative effect on EMG activation levels of VL muscle of the operated leg [111] (Fig. 5). Fatigue was verified with measurements of blood lactate and metabolic data [111] simulating conditions that are present in sporting events. Our subsequent study demonstrated that high intensity activities are required to reveal deficits of the operated leg since during low demand activities there are no differences in EMG activation levels between the operated, contralateral intact and control leg [112]. This study further demonstrated that similar levels of metabolic fatigue resulted in increased EMG activity for the control and intact leg but not for the operated leg [112]. An impaired response

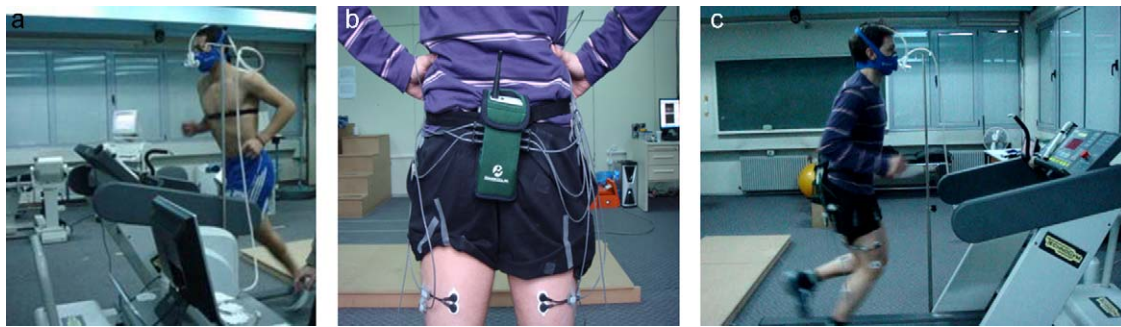


Figure 5 a: athlete performing a test on a treadmill to determine intensities for the subsequent running exercises; b: the EMG transmitter was placed behind the athletes back. EMG traces were collected from vastus lateralis and biceps femoris bilaterally; c: athlete running on treadmill, with simultaneous collection of metabolic and EMG data.

of the operate leg under accumulation of metabolic fatigue has been hypothesized [111].

Endurance training following ACL reconstruction and prior return to high intensity sports may enhance the response of the operated leg as may delay the occurrence of fatigue but this hypothesis remains to be tested [111]. Furthermore, conditions simulating metabolic fatigue under "field" settings may be necessary to reveal deficits in neuromuscular function of ACL reconstructed athletes.

Conclusion

Clinical knee joint biomechanics are impaired after ACL injury, in terms of kinematics and neuromuscular control. Current ACL reconstruction techniques do not seem to fully restore normal physiology of the movement. Excessive tibial rotation is still present in high-demanding activities and impairment of neuromuscular control may persist even though muscle strength is reinstated. These abnormal biomechanical patterns may lead to loading of cartilage areas which are not commonly loaded in the healthy knee and longitudinally can lead to osteoarthritis. Muscle imbalance can also influence patients' optimal sports performance, exposing them to increased possibility of knee re-injury. In this review, our recommendations point towards further experimental work, in order to assist in the development of new surgical procedures (like DB ACL reconstruction) that could possibly replicate more closely the natural ACL anatomy and prevent future knee pathology.

Conflict of interest statement

There is no conflict of interest.

References

- [1] Amis A, Bull AMJ, Lie DT. Biomechanics of rotational instability and anatomic anterior cruciate ligament reconstruction. *Oper Tech Orthop* 2005;15:29–35.
- [2] Petersen W, Zantop T. Anatomy of the anterior cruciate ligament with regard to its two bundles. *Clin Orthop Relat Res* 2007;454:35–47.
- [3] Colombet P, Robinson J, Christel P, Franceschi JP, Djian P, Bellier G, et al. Morphology of anterior cruciate ligament attachments for a natomic reconstruction: a cadaveric dissection and radiographic study. *Arthroscopy* 2006;22(9):984–92.
- [4] Zantop T, Herbolt M, Raschke MJ, Fu FH, Petersen W. The role of the anteromedial and posterolateral bundles of the anterior cruciate ligament in anterior tibial translation and internal rotation. *Am J Sports Med* 2007;35(2):223–7.
- [5] Girgis FG, Marshall JL, Monajem A. The cruciate ligaments of the knee joint: anatomical, functional and experimental analysis. *Clin Orthop Relat Res* 1975;106:216–31.
- [6] Woo SL, Kanamori A, Zeminski J, Yagi M, Papageorgiou C, Fu FH. 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* 2002;84:907–14.
- [7] Paessler HH, Michel D. How new is the Lachman test? *Am J Sports Med* 1992;20:95–8.
- [8] Noulis G. Entorse du genou. Thèse n° 142. Fac Med Paris 1875:1–53.
- [9] Arendt E, Agel J, Dick RW. Anterior cruciate ligament injury patterns among collegiate men and women. *J Athl Train* 1999;34(2):86–92.
- [10] Boden BP, Dean GS, Feagin Jr JA, Garrett Jr WE. Mechanisms of anterior cruciate ligament injury. *Orthopedics* 2000;23(6):573–8.
- [11] Cochrane JL, Lloyd DG, Butfield A, Seward H, McGivern J. Characteristics of anterior cruciate ligament injuries in Australian Football. *J Sci Med Sport* 2007;10(2):96–104.
- [12] Griffin LY, Albohm MJ, Arendt EA, et al. Understanding and preventing non-contact anterior cruciate ligament injuries: a review of the Hunt Valley II Meeting. *Am J Sports Med* 2005;34(9):1512–23.
- [13] Cerulli G, Benoit DL, Lamontagne M, Caraffa A, Liti A. In vivo anterior cruciate ligament strain behaviour during a rapid deceleration movement: case report. *Knee Surg Sports Traumatol Arthrosc* 2003;11(5):307–11.
- [14] Dempsey AR, Lloyd DG, Elliott BC, Steele JR, Munro BJ. Changing sidestep cutting technique reduces knee valgus loading. *Am J Sports Med* 2009;37(11):2194–200.
- [15] Caraffa A, Cerulli G, Proietti M, Aisa G, Rizzo A. Prevention of anterior cruciate ligament injuries in soccer: a prospective controlled study of proprioceptive training. *Knee Surg Sports Traumatol Arthrosc* 1996;4(1):19–21.
- [16] Hamel SL, Oakes DA, Markolf KL. Injury to the anterior cruciate ligament during alpine skiing: a biomechanical analysis of tibial torque and knee flexion angle. *Am J Sports Med* 2002;30(4):537–40.
- [17] Markolf KL, Burchfield DM, Shapiro MM, Shepard MF, Finerman GA, Slauterbeck JL. Combined knee loading states that generate high anterior cruciate ligament forces. *J Orthop Res* 1995;13(6):930–5.

- [18] Hewett TE, Myer GD, Kevin R, Ford KR. Prevention of Anterior Cruciate Ligament Injuries. *Curr Womens Health Rep* 2001;1:218–24.
- [19] Hewett TE, Stroupe AL, Nance TA, Noyes FR. Plyometric training in female athletes. Decreased impact forces and increased hamstring torques. *Am J Sports Med* 1996;24:765–73.
- [20] Hewett TE, Riccobene JV, Lindenfeld TN, et al. The effect of neuromuscular training on the incidence of knee injury in female athletes: a prospective study. *Am J Sports Med* 1999;27:699–706.
- [21] Knapik JJ, Bauman CL, Jones BH, et al. Preseason strength and flexibility imbalances associated with athletic injuries in female collegiate athletes. *Am J Sports Med* 1991;19:76–81.
- [22] Alentorn-Geli E, Myer GD, Silvers HJ, Samitier ZG, Romero D, Lázaro-Haro C, et al. Prevention of non-contact anterior cruciate ligament injuries in soccer players. Part 2. A review of prevention programs aimed to modify risk factors and to reduce injury rates. *Knee Surg Sports Traumatol Arthrosc* 2009;17:859–79.
- [23] Renstrom P, Ljungqvist A, Arendt E, Beynon B, Fukubayashi T, Garrett W, et al. Non-contact ACL injuries in female athletes: an International Olympic Committee current concepts statement. *Br J Sports Med* 2008;42:394–412.
- [24] Wilkerson GB, Colston MA, Short NI, Neal KL, Hoewischer PE, Pixley JJ. Neuromuscular changes in female collegiate athletes resulting from a plyometric jump-training program. *J Athl Train* 2004;39:17–23.
- [25] Chimera NJ, Swanik KA, Swanik CB, Straub SJ. Effects of plyometric training on muscle-activation strategies and performance in female athletes. *J Athl Train* 2004;39:24–31.
- [26] Pujol N, Blanchi MP, Chablat P. The incidence of anterior cruciate ligament injuries among competitive Alpine skiers: a 25-year investigation. *Am J Sports Med* 2007;35(7):1070–4.
- [27] Stergiou N, Ristanis S, Moraiti C, Georgoulis AD. Tibial rotation in anterior cruciate ligament (ACL)-deficient and ACL-reconstructed knees: a theoretical proposition for the development of osteoarthritis. *Sports Med* 2007;37(7):601–13.
- [28] Kessler MA, Behrend H, Henz S, Stutz G, Rukavina A, Kuster MS. Function, osteoarthritis and activity after ACL-rupture: 11 years follow-up results of conservative versus reconstructive treatment. *Knee Surg Sports Traumatol Arthrosc* 2008;16(5):442–8.
- [29] Feagin JA, Curl WW. Isolated tear of the anterior cruciate ligament: 5-year follow-up study. *Am J Sports Med* 1976;4:95–100.
- [30] Noyes FR, Butler DL, Grood ES, Zernicke RF, Hefzy MS. Biomechanical analysis of human ligament grafts used in knee-ligament repairs and reconstructions. *J Bone Joint Surg Am* 1984;66:344–52.
- [31] Jones RS, Nawana NS, Pearcy MJ, Learmonth DJ, Bickstaff DR, Costi JJ, et al. Mechanical properties of the human anterior cruciate ligament. *Clin Biomech* 1995;10(7):339–44.
- [32] Paschos NK, Gartzonikas D, Barkoula N-M, Moraiti C, Paipetis A, Matikas TE, et al. Cadaveric study of anterior cruciate ligament failure patterns under uniaxial tension along the ligament. *Arthroscopy* 2010;26(7):957–67.
- [33] Sonnerby-Cottet B, Lavoie F, Ogassawara R, Scussiato RG, Kidder JF, Chablat P. Selective anteromedial bundle reconstruction in partial ACL tears: a series of 36 patients with mean 24 months follow-up. *Knee Surg Sports Traumatol Arthrosc* 2010;18(1):47–51.
- [34] Siebold R, Fu FH. Assessment and augmentation of symptomatic anteromedial or posterolateral bundle tears of the anterior cruciate ligament. *Arthroscopy* 2008;24(11):1289–98.
- [35] Spindler KP, Schils JP, Bergfeld JA, et al. Prospective study of osseous, articular, and meniscal lesions in recent anterior cruciate ligament tears by magnetic resonance imaging and arthroscopy. *Am J Sports Med* 1993;21:551–7.
- [36] Viskontas DG, Giuffre BM, Duggal N, Graham D, Parker D, Coolican M. Bone bruises associated with ACL rupture: correlation with injury mechanism. *Am J Sports Med* 2008;36:927–33.
- [37] Moisala AS, Järvelä T, Harilainen A, Sandelin J, Kannus P, Järvinen M. The effect of graft placement on the clinical outcome of the anterior cruciate ligament reconstruction: a prospective study. *Knee Surg Sports Traumatol Arthrosc* 2007;15(7):879–87.
- [38] Ochi M, Adachi N, Uchio Y, Deie M, Kumahashi N, Ishikawa M, et al. A minimum 2-year follow-up after selective anteromedial or posterolateral bundle anterior cruciate ligament reconstruction. *Arthroscopy* 2009;25(2):117–22.
- [39] Biau DJ, Katsahian S, Kartus J, Harilainen A, Feller JA, Sajovic M, et al. Patellar tendon versus hamstring tendon autografts for reconstructing the anterior cruciate ligament: a meta-analysis based on individual patient data. *Am J Sports Med* 2009;37(12):2470–8.
- [40] Branch TP, Browne JE, Campbell JD, Siebold R, Freedberg HI, Arendt EA, et al. Rotational laxity greater in patients with contralateral anterior cruciate ligament injury than healthy volunteers. *Knee Surg Sports Traumatol Arthrosc* 2010;18(10):1379–84.
- [41] 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* 2003;31(1):75–9.
- [42] 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* 2003;11(6):360–5.
- [43] Ristanis S, Stergiou N, Patras K, et al. Excessive tibial rotation during high demanding activities is not restored by ACL reconstruction. *Arthroscopy* 2005;21(11):1323–9.
- [44] Ristanis S, Stergiou N, Patras K, et al. Follow-up evaluation 2 years after ACL reconstruction with a BPTB graft shows that excessive tibial rotation persists. *Clin J Sports Med* 2006;16(2):111–6.
- [45] Ristanis S, Stergiou N, Siarava E, Ntoulia A, Mitsionis G, Georgoulis AD. Effect of femoral tunnel placement for reconstruction of the anterior cruciate ligament on tibial rotation. *J Bone Joint Surg Am* 2009;91(9):2151–8.
- [46] Chouliaras V, Ristanis S, Moraiti C, Stergiou N, Georgoulis AD. Effectiveness of reconstruction of the anterior cruciate ligament with quadrupled hamstrings and bone-patellar tendon-bone autografts: an in vivo study comparing tibial internal-external rotation. *Am J Sports Med* 2007;35(2):189–96.
- [47] Georgoulis AD, Ristanis S, Chouliaras V, Moraiti C, Stergiou N. Tibial rotation is not restored after ACL reconstruction with a hamstring graft. *Clin Orthop Relat Res* 2007;454:89–94.
- [48] Rowden N, Sher D, Rogers G, et al. Anterior cruciate ligament graft fixation. Initial comparison of patellar tendon and semitendinosus autografts in young fresh cadavers. *Am J Sports Med* 1997;25:472–8.
- [49] 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* 2002;30(3):361–7.
- [50] Logan CM, Williams A, Lavelle J, et al. Tibiofemoral kinematics following successful ACL reconstruction using dynamic multiple resonance imaging. *Am J Sports Med* 2004;32(4):984–92.
- [51] McLean SG, Huang X, Su A, Van Den Bogert AJ. Sagittal plane biomechanics cannot injure the ACL during sidestep cutting. *Clin Biomech* 2004;19(8):828–38.

- [52] Lohmander LS, Ostergren A, Englund M, Roos H. High prevalence of knee osteoarthritis, pain, and functional limitations in female soccer players twelve years after anterior cruciate ligament injury. *Arthritis Rheum* 2004;50(10):3145–52.
- [53] Andriacchi TP, Briant PL, Bevil SL, Koo S. Rotational changes at the knee after ACL injury cause cartilage thinning. *Clin Orthop Relat Res* 2006;442:39–44.
- [54] Hantes ME, Liantis AK, Basdekis GK, Karantanas AH, Christel P, Malizos KN. Evaluation of the bone bridge between the bone tunnels after anatomic double-bundle anterior cruciate ligament reconstruction: a multidetector computed tomography study. *Am J Sports Med* 2010;38(8):1618–25.
- [55] Yagi M, Kuroda R, Nagamune K, Yoshiya S, Kurosaka M. Double bundle ACL reconstruction can improve rotational stability. *Clin Orthop Relat Res* 2007;454:100–7.
- [56] Zantop T, Wellmann M, Fu FH, Petersen W. Tunnel positioning of anteromedial and posterolateral bundles in anatomical anterior cruciate ligament reconstructions: anatomic and radiographic findings. *Am J Sports Med* 2008;36:65–72.
- [57] Zantop T, Petersen W, Sekiya JK, Musahl V, Fu FH. Anterior cruciate ligament anatomy and function relating to anatomical reconstruction. *Knee Surg Sports Traumatol Arthrosc* 2006;14:982–92.
- [58] Colombet PD, Robinson JR. Computer assisted anatomic, double-bundle anterior cruciate ligament reconstruction. *Arthroscopy* 2008;24(10):1152–60.
- [59] Christel P, Sahasrabudhe A, Basdekis G. Anatomic double-bundle anterior cruciate ligament reconstruction with anatomic aimers. *Arthroscopy* 2008;24(10):1146–51.
- [60] Stergiou N, Buzzi UH, Kurz MJ, et al. Nonlinear tools in human movement. In: Stergiou N, editor. *Innovative analyses of human movement*. Champaign, IL: Human Kinetics Publ; 2004.
- [61] Hausdorff JM, Peng CK, Ladin Z, Wei JY, Goldberger AL. Is walking a random walk? Evidence for long-range correlations in stride interval of human gait. *J Appl Physiol* 1995;78(1):349–58.
- [62] Kaplan DT, Furman MI, Pincus SM, et al. Aging and the complexity of cardiovascular dynamics. *Biophys J* 1991;59:945–9.
- [63] Le Van Quyen M, Martinerie J, Navarro V, Boon P, D'Have M, Adam C, et al. Anticipation of epileptic seizures from standard EEG recordings. *Lancet* 2001;357:183–8.
- [64] Veldman RG, Frolich M, Pincus SM, Veldhuis JD, Roelfsema F. Growth hormone and prolactin are secreted more irregularly in patients with Cushing's disease. *Clin Endocrinol* 2000;52(5):625.
- [65] Lipsitz LA, Goldberger AL. Loss of 'complexity' and aging. Potential applications of fractals and chaos theory to senescence. *JAMA* 1992;267(13):1806–9.
- [66] Stergiou N, Harbourne RT, Cavanaugh JT. Optimal movement variability: a new theoretical perspective for neurologic physical therapy. *J Neurol Phys Ther* 2006;30(3):120–9.
- [67] Stergiou N, Moraiti C, Giakas G, Ristanis S, Georgoulis AD. The effect of the walking speed on the stability of the anterior cruciate ligament deficient knee. *Clin Biomech (Bristol, Avon)* 2004;19(9):957–63.
- [68] Georgoulis AD, Moraiti C, Ristanis S, Stergiou N. A novel approach to measure variability in the anterior cruciate ligament deficient knee during walking: the use of the approximate entropy in orthopaedics. *J Clin Monit Comput* 2006;20(1):11–8.
- [69] Moraiti C, Stergiou N, Ristanis S, Georgoulis AD. ACL deficiency affects stride-to-stride variability as measured using nonlinear methodology. *Knee Surg Sports Traumatol Arthrosc* 2007;15(12):1406–13.
- [70] Schultz RA, Miller DC, Kerr CS, Micheli L. Mechanoreceptors in human cruciate ligaments. A histological study. *J Bone Joint Surg Am* 1984;66:1072–6.
- [71] Schutte MJ, Dabezies EJ, Zimny ML, Happel LT. Neural anatomy of the human anterior cruciate ligament. *J Bone Joint Surg Am* 1987;69:243–7.
- [72] Courtney C, Rine RM, Kroll P. Central somatosensory changes and altered muscle synergies in subjects with anterior cruciate ligament deficiency. *Gait Posture* 2005;25:69–74.
- [73] Di Fabio RP, Graf B, Badke MB, Breunig A, Jensen K. Effect of knee joint laxity on long-loop postural reflexes: evidence for a human capsular-hamstring reflex. *Exp Brain Res* 1992;90:189–200.
- [74] Georgoulis AD, Pappa L, Moebius U, Malamou-Mitsi V, Pappa S, Papageorgiou CO, et al. The presence of proprioceptive mechanoreceptors in the remnants of the ruptured ACL as a possible source of re-innervation of the ACL autograft. *Knee Surg Sports Traumatol Arthrosc* 2001;9:364–8.
- [75] Moraiti CO, Stergiou N, Ristanis S, Vasiliadis HS, Patras K, Lee C, et al. The effect of anterior cruciate ligament reconstruction on stride-to-stride variability. *Arthroscopy* 2009;25(7):742–9.
- [76] Moraiti CO, Stergiou N, Vasiliadis VS, Mosis E, Georgoulis AD. Anterior cruciate ligament reconstruction results in alterations in gait variability. *Gait Posture* 2010;32(2):169–75.
- [77] Hiemstra LA, Webber S, MacDonald PB, Kriellars DJ. Hamstrings and quadriceps strength balance in normal and hamstring anterior cruciate ligament-reconstructed subjects. *Clin J Sports Med* 2004;14:274–80.
- [78] Nakamura N, Horibe S, Sasaki S, et al. Evaluation of active knee flexion and hamstring strength after anterior cruciate ligament reconstruction using hamstring tendons. *Arthroscopy* 2002;18:598–602.
- [79] Hiemstra LA, Webber S, MacDonald PB, Kriellaars DJ. Contralateral limb strength deficits after anterior cruciate ligament reconstruction using a hamstring tendon graft. *Clin Biomech* 2007;22:543–50.
- [80] Myer GD, Paterno MV, Ford KR, Hewett TE. Neuromuscular training techniques to target deficits before return to sport after anterior cruciate ligament reconstruction. Review. *J Strength Cond Res* 2008;22:987–1014.
- [81] Winter DA, Pluck N, Yang JF. Backward walking: a simple reversal of forward walking? *J Mot Behav* 1989;21:291–305.
- [82] Grasso R, Bianchi L, Lacquaniti F. Motor patterns for human gait: backward versus forward locomotion. *J Neurophysiol* 1998;8:1868–85.
- [83] Hooper TL, Dunn DM, Props JE, Bruce BA, Sawyer SF, Daniel JA. The effects of graded forward and backward walking on heart rate and oxygen consumption. *J Orthop Sports Phys Ther* 2004;34:65–71.
- [84] Keays SL, Bullock-Saxton J, Keays AC. Strength and function before and after anterior cruciate ligament reconstruction. *Clin Orthop* 2000;373:174–83.
- [85] Mattacola CG, Perrin DH, Gansneder BM, Gieck JH, Saliba EN, McCue FC. Strength, functional outcome, and postural stability after anterior cruciate ligament reconstruction. *J Athl Train* 2002;37:262–8.
- [86] Ageberg E, Thomeé R, Neeter C, Silbernagel KG, Roos EM. Muscle strength and functional performance in patients with anterior cruciate ligament injury treated with training and surgical reconstruction or training only: a two- to five-year follow-up. *Arthritis Rheum* 2008;50(12):1773–9.
- [87] Almekinders LC, Oman J. Isokinetic muscle testing: is it clinically useful? *J Am Acad Orthop Surg* 1994;2:221–5.
- [88] Pua YH, Bryant AL, Steele JR, Newton RU, Wrigley TV. Isokinetic dynamometry in anterior cruciate ligament injury and reconstruction. *Ann Acad Med Singapore* 2008;37(4):330–40.
- [89] Tsepis E, Vagenas G, Giakas G, Georgoulis A. Hamstring weakness as an indicator of poor knee function in ACL-deficient patients. *Knee Surg Sports Traumatol Arthrosc* 2004;12(1):22–9.

- [90] Eitzen I, Eitzen TJ, Holm I, Snyder-Mackler L, Risberg MA. Anterior cruciate ligament-deficient potential copers and noncopers reveal different isokinetic quadriceps strength profiles in the early stage after injury. *Am J Sports Med* 2010;38(3):586–93.
- [91] Tsepis E, Vagenas G, Ristanis S, Georgoulis AD. Thigh muscle weakness in ACL-deficient knees persists without structured rehabilitation. *Clin Orthop Relat Res* 2006;450:211–8.
- [92] Konishi Y, Fukubayashi T, Takeshita D. Mechanism of quadriceps femoris muscle weakness in patients with anterior cruciate ligament reconstruction. *Scand J Med Sci Sports* 2002;12(6):371–5.
- [93] Wexler G, Hurwitz D, Bush-Joseph CA, Andriacchi T, Bernard Jr RB. Functional gait adaptations in patients with anterior cruciate ligament deficiency over time. *Clin Orthop Relat Res* 1998;348:166–75.
- [94] Papadonikolakis A, Cooper L, Stergiou N, Georgoulis AD, Soucacos PN. Compensatory mechanisms in anterior cruciate ligament deficiency. *Knee Surg Sports Traumatol Arthrosc* 2003;11(4):235–43 [Epub 2003 Apr 17].
- [95] Krogsgaard MR, Dyhre-Poulsen P, Fischer-Rasmussen T. Cruciate ligament reflexes. *J Electromyogr Kinesiol* 2002;12(3):177–82.
- [96] Myer GD, Paterno MV, Ford KR, Quatman CE, Hewett TE. Rehabilitation after anterior cruciate ligament reconstruction: criteria-based progression through the return-to-sport phase. *J Orthop Sports Phys Ther* 2006;36(6):385–402.
- [97] Dauty M, Tortellier L, Rochcongar P. Isokinetic and anterior cruciate ligament reconstruction with hamstrings or patella tendon graft: analysis of literature. *Int J Sports Med* 2005;26(7):599–606.
- [98] Van Grinsven S, van Cingel RE, Holla CJ, van Loon CJ. Evidence-based rehabilitation following anterior cruciate ligament reconstruction. *Knee Surg Sports Traumatol Arthrosc* 2010;18(8):1128–44.
- [99] Eitzen I, Holm I, Risberg MA. Preoperative quadriceps strength is a significant predictor of knee function two years after anterior cruciate ligament reconstruction. *Br J Sports Med* 2009;43(5):371–6.
- [100] Petersen W, Laprell H. Combined injuries of the medial collateral ligament and the anterior cruciate ligament. Early ACL reconstruction versus late ACL reconstruction. *Arch Orthop Trauma Surg* 1999;119(5–6):258–62.
- [101] Andersson D, Samuelsson K, Karlsson J. Treatment of anterior cruciate ligament injuries with special reference to surgical technique and rehabilitation: an assessment of randomized controlled trials. *Arthroscopy* 2009;25(6):653–85.
- [102] Cavanagh PR, Komi PV. Electromechanical delay in human skeletal muscle under concentric and eccentric contractions. *Eur J Appl Physiol Occup Physiol* 1979;42(3):159–63.
- [103] Vos EJ, Harlaar J, van Ingen Schenau GJ. Electromechanical delay during knee extensor contractions. *Med Sci Sports Exerc* 1991;23(10):1187–93.
- [104] Georgoulis AD, Ristanis S, Papadonikolakis A, Tsepis E, Moebius U, Moraiti C, et al. Electromechanical delay of the knee extensor muscles is not altered after harvesting the patellar tendon as a graft for ACL reconstruction: implications for sports performance. *Knee Surg Sports Traumatol Arthrosc* 2005;13(6):437–43.
- [105] Ristanis S, Tsepis E, Giotis D, Stergiou N, Cerulli G, Georgoulis AD. Electromechanical delay of the knee flexor muscles is impaired after harvesting hamstring tendons for anterior cruciate ligament reconstruction. *Am J Sports Med* 2009;37(11):2179–86.
- [106] Limbird TJ, Shiavi R, Frazer M, Borra H. EMG profiles of knee joint musculature during walking: changes induced by anterior cruciate ligament deficiency. *J Orthop Res* 1988;6:630–8.
- [107] Kalund S, Sinkjaer T, Arendt-Nielsen L, Simonsen O. Altered timing of hamstring muscle action in anterior cruciate ligament deficient patients. *Am J Sports Med* 1990;18:245–8.
- [108] Knoll Z, Kiss RM, Kocsis L. Gait adaptation in ACL deficient subjects before and after anterior cruciate ligament reconstruction surgery. *J Electromyogr Kinesiol* 2004;14:287–94.
- [109] 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;17:56–63.
- [110] Alentorn-Geli E, Myer GD, Silvers HJ, Samitier G, Romero D, Lázaro-Haro C, et al. Prevention of non-contact anterior cruciate ligament injuries in soccer players. Part 1. Mechanisms of injury and underlying risk factors. *Knee Surg Sports Traumatol Arthrosc* 2009;17:705–29.
- [111] Patras K, Ziogas G, Ristanis S, Tsepis E, Stergiou N, Georgoulis AD. High intensity running results in an impaired neuromuscular response in ACL reconstructed individuals. *Knee Surg Sports Traumatol Arthrosc* 2009;17:977–84.
- [112] Patras K, Ziogas G, Ristanis S, Tsepis E, Stergiou N, Georgoulis AD. ACL reconstructed patients with a BPTB graft present an impaired vastus lateralis neuromuscular response during high intensity running. *J Sci Med Sport* 2010 [Epub ahead of print].