Activation and Torque Deficits in ACL-Reconstructed Patients 4 Months Post-Operative

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This study compares knee extension and flexion torques and electromyographic (EMG) activity of normal and anterior cruciate ligament (ACL)-reconstructed knees during maximal unilateral isometric and isokinetic tests performed 4–5 months after ACL reconstruction. The subjects consisted of 3 age- and activity-matched groups of 6 subjects: a healthy control group (Ctrl) and 2 groups of patients, with Kenneth-Jones technique using autologous patellar-ligament graft (KJ group) and autologous graft from the semitendinosus tendon (ST group). When compared to the Ctrl group values, each patient group had significant bilateral extension torque deficits in isometric and at slow velocity concentric conditions. In the Op leg, this deficit was associated with lower quadriceps EMG activation. In all tests, bilateral hamstring co-activation level was lower in the patient groups. Flexion torque deficits were observed in the Op leg of both patient groups and in the Nop leg of the KJ group, with minor differences in either hamstring EMG activation or quadriceps co-activation. The inter-leg difference in extension and flexion torques were significant for both patient groups, but only in the isometric strength test, and with no difference in activation. These data demonstrate bilateral knee extension and flexion torque deficits a few months after ACL reconstruction. The observed deficit in quadriceps activation emphasizes the interest to perform combined EMG and strength testings quite early during the rehabilitation period.

Key Words: ACL, EMG, activation deficit, torque deficit, co-activation.

Key Points:
• Protective deficit in quadriceps activation partly explains the long-lasting recovery in extension torque production in ACL-reconstructed patients.
• An isometric strength test is useful in revealing deficits in maximal activation and torque production in such patients.
• Based on observed bilateral deficit, responses of the operated leg should be compared not only with the non-operated leg, but also with the legs of healthy control subjects.

Introduction
The anterior cruciate ligament (ACL) plays major mechanical and kinesthetic roles at the knee joint. Anatomic studies have clearly defined the composition, orientation, and attachment sites of the ACL (1). Mechanical studies with cadavers have demonstrated that the ACL ligament plays a primary role in restraining anterior tibial displacement with respect to the femur (2). In addition
to this function, the ACL also restrains internal knee rotation (3). The ACL tension varies depending on the type of movement (knee extension, flexion, and/or rotation), angular position (3), and intervention of active muscle forces (4, 5). Equilibrium under variable loading and geometric conditions of the joint would require synergism of all active and passive components. The literature reports of the existence of a complex but systematic sensory-motor synergy around the anatomical structures of the knee. In vivo mechanical studies with strain transducers have suggested that cruciate receptors may act as sensors of tibial position and movement (6). Histologic investigations have identified in the ACL two types of mechanoreceptors, similar to Ruffini corpuscles and Golgi tendon organs, that could respond to changes in ligament tension and provide the central nervous system with information on the position, velocity, and acceleration of the tibial motion (7). ACL rupture may thus be suggested to lead to a vicious circle in which the combined increased laxity and sensorial deficits would induce a functional instability, thus increasing the risk of additional lesions.

ACL injury is particularly high in contact sports that involve high acceleration and/or side-cutting movements, and more frequent in sportive females than in males (8). In spite of its surgical reconstruction, the ACL injury is generally followed by long-term weakness of especially the knee extensor muscles. Deficits of 30 to 50% in maximal knee extension torque have been reported 3 and 9 months after ACL reconstruction (9, 10) as well as long-lasting deficits of 25% and 15%, respectively, 1 year and 2 years post surgery (11). Muscle weakness has been mostly attributed to a loss of muscle mass caused by immobilization or limited use of the injured limb. Disuse atrophy does not seem, however, to fully account for the prolonged decrease in knee extension strength in acute disuse. According to Suter and Herzog (12), only little attention has been paid to the possible contribution of inhibition to the muscle weakness and associated long-term functional and structural changes of the knee joint after surgery. Using the interpolated twitch technique in ACL patients several months after the ligament rupture, these authors demonstrated knee extensor inhibition in the injured as well as in the contralateral limb. These bilateral activation deficits were 2 to 3 times higher than the 10% inhibition of the control group. On the other hand, Hurley et al. (13) reported that severe initial knee extensor inhibition (30–45%) might interfere with the rehabilitation process and compromise the functional recovery, whereas a small amount of initial inhibition seems to allow for strength gains early in the rehabilitation process.

Observation of long-lasting torque decrement, even after ACL-reconstruction, raises the problem of the protective neuromuscular adjustments that could delay or even limit the functional recovery. The present paper therefore makes an attempt to evaluate after a few months of recovery the effects of ACL injury and reconstruction on the neuromuscular function in commonly used isometric and isokinetic strength tests.

Materials and Methods

Subjects
A total of 18 subjects, ranging from 18 to 28 years, volunteered for the study. From these, the control group (C) included 6 healthy subjects (5 males and 1 female) with no previous records of any lower extremity problems. This group was matched, with respect to age and athletic level, with two patient groups. The first patient group (KJ) included 4 males and 2 females with reconstructed ACL by the Kenneth-Jones technique (autologous patellar-ligament graft). The
second patient group (ST) included 3 men and 3 women with reconstructed ACL from an autologous graft consisting of four loops of the semitendinosus tendon. Table 1 summarizes data on each group, including the mean times from accident to surgery and from surgery to testing. None of the patients had any pain, knee effusion, or exhibited any type of knee-joint damage besides the ACL rupture. None of the subjects had undergone isokinetic training or testing before participating in this study.

**Methods**

The testing protocol included a series of isometric and concentric knee extensions and flexions on a specific isokinetic dynamometer (Cybex Norm). The subjects sat on the dynamometer chair in a secured position. Special care was taken to align the dynamometer rotation axis with the estimated knee joint anatomical flexion-extension axis. In order to familiarize the patients with the tests and to make them more confident while testing the operated leg (Op), the non-operated leg (Nop) was systematically tested first. In addition, the tests were preceded by a warm-up and training periods with visual feedback to teach the patients to produce a fast rise in torque and to maintain the maximal effort over the entire range of motion.

In the isometric strength tests, two maximal trials were performed at the knee angles of 65° in extension and 40° in flexion (0° corresponding to complete knee extension). The subjects were verbally encouraged to produce maximal torque output as fast as possible and to maintain the maximal effort for 5 s. The trials were separated by a recovery period of 5 min. Four warm-up contractions were systematically performed with gradually increasing efforts before the maximal test. The isokinetic testing series included 3 maximal tests of 4 successive knee extension-flexion movements (at 60, 180, and 240° · s⁻¹). Each test was performed over a 95° range (5–100°) and was preceded by a familiarization over 3 repetitions at the predetermined velocity.

Torque and angular displacement were recorded in parallel with surface electromyographic (EMG) activity detected from 6 selected muscles. The overall recording was performed at a sampling frequency of 1000 Hz (ME3000P; MEGA systems, Finland), and transmitted via an optic fiber to a PC. Bipolar technique with disposable surface electrodes (Contrôle Graphique Médical) was used to record the EMG activity from (a) the major knee extensors: vastus medialis (VM), vastus lateralis (VL), and rectus femoris (RF); (b) the knee flexors: biceps femoris (BF), semitendinosus and semimembranosus (ST-SM); and (c) the lateral gastrocnemius (LG) of both legs. Each EMG signal was band-pass filtered (20–500 Hz) and pre-amplified nearby the site of the electrode.

**Data Analysis**

From the recorded maximal isometric performances, the trial presenting the highest peak torque was kept for further analysis. For the isokinetic strength tests, the two intermediate repetitions, which showed lesser variability than the other two, were selected for subsequent analysis. For each of the selected trials, the digitized EMG signals were rectified and afterwards low-pass filtered at 100 Hz in parallel with the torque signal. The subsequent part of the analysis was then specific to each mode of testing.

The specific analysis of the isometric strength performances examined the peak torque values during the first 2,500 ms from the start of the torque rise. The EMG activity of each muscle was
averaged for a period of 1 s (from −500 to +500 ms) around the peak torque. These averaged EMG values were then used to calculate mean EMG activation of the quadriceps femoris (VM, VL, RF) and hamstring (BF, ST-SM) muscle groups. The lateral gastrocnemius (LG) EMG activity varied greatly among subjects and was not analyzed any further. For the isokinetic performances, the torque and EMG signals were first averaged for the 2 successive repetitions of each selected velocity (Figure 1A). Mean torque and mean EMG activity of each muscle were then calculated for the respective mean extension and flexion phases. Mean torque was chosen instead of peak torque to provide more insight into the total contribution of the involved muscles. To estimate the relative co-activation level, the mean activity of each antagonist muscle was expressed in percentage of its agonist activity during the opposite phase of movement. The hamstring muscle group presented in some subjects a short initial burst of activity at the beginning of the extension movement that has been previously described by Solomonov et al. (1987) and Aagaard et al. (2000). This hamstring burst differed clearly from the rest of the antagonist EMG activity that was steady thereafter at a lower level throughout extension. The level of co-activation was calculated for the second half of the respective extension and flexion movements.

![Figure 1 — A. Average recording for a patient of 2 consecutive knee extension-flexion movements at 60° · s⁻¹ including knee torque, angular displacement, mean quadriceps femoris (QF), hamstrings (H), and lateral gastrocnemius (LG) rectified EMG activity. B. Mean value of each signal corresponding to 4 equal parts of the angular displacement in extension (from 100 to 5°) and in flexion (from 5 to 100°). Reference 100%: maximal Nop leg value. (Note: See color originals of this figure at <www.humankinetics.com/cjss>.)](image)

The subsequent part of the analysis (Figure 1B) consisted of calculating the mean value of each signal corresponding to 4 equal parts (∼24°) of the angular displacement in extension as well as
in flexion. These angular displacements (100–76, 76–52, 52–28, 28–5°) are indicated on Figure 1B by the corresponding mean knee joint positions of 88, 64, 40, and 16°. The maximal values obtained for each signal in the Nop leg were then used as 100% reference to allow comparison of the relative EMG and torque patterns between the Nop and the Op legs. The obtained individual values were then averaged for each group.

To calculate the relative differences in torque and activation between the two legs, the maximal torque and EMG values of the Op leg were then expressed as a percentage of the corresponding values measured in the Nop leg. In the control group, the left leg values were taken as 100% reference.

**Statistical Analysis**
Differences in absolute torque value and agonist EMG activation as well as in relative level of co-activation were examined by a three-way (Group x Leg x Velocity) analysis of variance (ANOVA) with repeated measures on the last two factors. When this analysis revealed significant differences, a HSD Tukey post hoc test was used to compare the experimental conditions. The level of statistical significance was set at $p = .05$, and the minimum effect intensity (EI) was set at 2%.

**Results**
Independently of the tested velocity, group effects, and leg effects, the results differed clearly in extension and flexion. These conditions therefore are presented separately. In the case of significant main effects and interactions, the latter are shown with statistical significance. When the absolute changes in torque and EMG activity were significant, they are often illustrated as corresponding relative changes for reasons of convenience.

**Maximal Knee Extension Tests**
Figure 2 presents the averaged values of extension torque, quadriceps EMG activity, and hamstring co-activation for each of the 4 testing conditions (0, 60, 180, and 240° · s$^{-1}$) and for the operated (Op) and non operated (Nop) legs of each patient group. As the Ctrl group presented only minor and non-significant inter-leg variations (0 ± 7% in torque, 2 ± 11% in quadriceps EMG and 0 ± 5% in hamstring co-activation), this group is represented in Figure 2 by the mean values only. However, for statistical inter-group comparisons, the Nop and Op legs of the patient groups were compared with the respective left and right legs of the Ctrl group.
Figure 2 — Patient group mean (±SD) values corresponding to the operated (Op) and non operated (Nop) extension leg torque, quadriceps AEMG activity, and relative hamstring co-activation level in each of the 4 tests (0, 60, 180, and 240° · s⁻¹). The control (Ctrl) group is represented by mean left and right leg values. Note: *p < .05 and ***p < .001 in the Group x Velocity comparison; ###p < .001 in the Group x Leg x Velocity comparison; £p < .05 and ££p < .01 in the Group x Leg comparison. (Note: See color originals of this figure at www.humankinetics.com/ejss.)

For the extension torque, the analysis revealed significant main effects of Velocity (p < .001), Leg (p < .001), and Group (p < .01), accompanied by significant two-way interactions between Group and Velocity (F_{6,45} = 12.48; p < .001; EI = 3.5%), Group and Leg (F_{2,15} = 6.90; p < .01; EI = 1.4%), Velocity and Leg (F_{3,45} = 109.39; p < .001; EI = 15%) as well as between Group and Leg and Velocity (F_{6,45} = 6.96; p < .001; EI = 1.9%).

For the Group x Velocity effect, post hoc analysis revealed that the patient groups differed from the Ctrl group during isometric testing (KJ, ST: p < .001) and at low (60° · s⁻¹) velocity (KJ: p < .001; ST: p < .05), while no differences were observed at the higher velocities. When expressed in relative values, the torque deficits of the patient groups averaged 34 ± 2% in isometric and 24
± 1% at 60°·s⁻¹. The Group x Leg effect resulted from the bilateral similarity of the Ctrl group, while each patient presented around 30% torque deficit in the Op leg (KJ, ST: p < .001) and 20% in the Nop leg (KJ, ST: p < .001) as compared to the Ctrl leg values. The Velocity x Leg effect reflected a significant 30 ± 10% inter-leg difference in the isometric testing condition (p < .001) but none in the other tests. More specifically, the interaction between the three factors revealed in the isometric condition and for the KJ patient group only, a significant torque deficit in the Op leg as compared to the Nop leg (p < .001). When compared to the unilateral Ctrl leg values, each patient group presented at least 30% lower torque values in the Op leg and 25% in the Nop leg. Post hoc analysis revealed for the Op leg significant torque deficits in isometric (KJ, ST: p < .001) and at 60°·s⁻¹ concentric velocity (KJ: p < .001; ST: p < .05). In the Nop leg, both patient groups had a significant torque deficit in isometric strength test (p < .001); the ST group presenting also deficits in the concentric tests at 60°·s⁻¹ (p < .05) and at 240°·s⁻¹ (p < .05).

In quadriceps EMG activation, the analysis revealed a significant Group and Leg interaction (F₂,₁₅ = 3.71; p < .05; EI = 16.6%) that reflected 20 ± 3% lower quadriceps activation in the Op leg of the patient groups (KJ: p < .05; ST: p < .01) as compared to the reference leg of the Ctrl group. The inter-leg difference did not reach the level of statistical significance.

For the hamstring co-activation, the analysis revealed a significant main effect of Group (F₂,₁₅ = 4.91; p < .05; EI = 79.1%). The relative co-activation level that averaged 27 ± 3% for both legs in the Ctrl group was significantly lower (p < .05) in the KJ and ST patient groups, with 16 ± 4% and 14 ± 3%, respectively.

**Maximal Knee Flexion Tests**

Similar to the results in extension, the Ctrl group presented only minor and non-significant differences between the left and the right leg values: 3 ± 5% in flexion torque, 6 ± 20% in hamstring AEMG activity and 1 ± 0.5% in quadriceps co-activation. For this reason, the Ctrl group is represented in Figure 3 by the mean leg values together with the respective Op and Nop leg values of each patient group. For the statistical comparisons, the Nop and Op legs of the patient groups are compared to the respective left and right legs of the Ctrl group.
For the flexion torque, the statistical analysis revealed significant main effects of Leg ($p < .01$) and Velocity ($p < .001$) accompanied by significant interactions, although of limited influence (< 2%) between Group and Leg ($F_{2,15} = 9.57; p < .01; EI = 1.7\%$) and Leg and Velocity ($F_{3,45} = 4.33; p < .01; EI = 0.6\%$). As compared to the Ctrl leg values, each patient group presented a significant flexion torque deficit of at least 20% in the Op leg (KJ, ST: $p < .001$), with a slight 7% but significant deficit ($p < .05$) in the Nop leg of the KJ group. The Leg x Velocity effect reflected significant inter-leg difference, but only in the isometric testing condition ($p < .001$).

For the hamstring AEMG activity, the analysis did not reveal any significant main or interaction effects. As shown in Figure 3, patient and Ctrl groups presented very similar and constant AEMG values among the 4 testing conditions.
For the quadriceps co-activation during the flexion movement, the analysis indicated significant main effects of Leg ($F_{1,15} = 5.16; p < .05; EI = 8.8\%$) and Velocity ($F_{3,45} = 5.87; p < .01; EI = 27.5\%$) but no interactions. With regard to the leg effect, its influence may be considered as negligible, as the inter-leg difference in co-activation corresponded to only 1%. The velocity effect revealed that, independent of the subject group, quadriceps co-activation was slightly lower (7.5\%) in the isometric as compared to dynamic tests. In the latter tests the co-activation level averaged 10\% at $60^\circ \cdot s^{-1}$ ($p < .01$) and 9.5\% at $180^\circ \cdot s^{-1}$ ($p < .05$).

**Discussion**

The major findings of the present study were as follows: despite the limited number of subjects, both ACL-reconstructed patient groups presented significant extension and flexion torque deficits in the Op leg as compared to Ctrl subjects with comparable anthropometric characteristics and athletic background. Independently of the type of surgery, the torque deficits were more prominent in extension than in flexion (32 vs. 22\%), and were larger in isometric and at slow isokinetic concentric velocity. The torque deficits were associated in the patient groups, with 20\% lower EMG of the quadriceps muscle group. Interestingly, the parallel analysis of Nop leg values revealed a mean 21\% extension torque deficit in the patient groups and 7\% flexion torque deficit in the KJ patient group, with no significant differences in the agonist EMG. On the other hand, the hamstring co-activation level was bilaterally and independent of the testing condition significantly lower in the KJ and ST patient groups than in the Ctrl group (16 and 14\% vs. 27\%). The inter-leg comparison revealed for both patient groups significant extension and flexion torque deficits in the Op leg, particularly in the isometric testing condition.

A first explanation for these findings may be that disuse and knee pain are more likely to affect primarily the extensor muscle chain, leading to quadriceps amyotrophy and associated performance reduction. It should be mentioned that disuse amyotrophy alone does not account for the prolonged decrease in knee extension strength observed in acute disuse (16) and chronic ACL-tear (17). As emphasized by recent studies on ACL patients (12, 18), more attention should be paid to the additional contribution of inhibition to the measured muscle weakness and associated long-term functional and structural changes of the knee joint after surgery. With regard to the potential role of deficit in activation of the knee extensors of the operated leg, the present EMG analysis revealed a significantly lower quadriceps activation in both patient groups as compared to the Ctrl group. As illustrated in Figure 1 for one KJ patient at $60^\circ \cdot s^{-1}$, this activation deficit was not position-specific but covered the entire range of motion, resulting in a slow rate and reduced maximal torque development. Similarly, incomplete voluntary activation of knee extensor muscles has been observed in patients after ACL rupture (12, 13, 18) and ACL reconstruction (12) as well as after meniscectomy (19), anterior knee pain (20), experimental knee effusion (21), and extensive knee injury (22, 23). In the latter study of Hurley et al. (23), it was shown that severe initial knee extensor inhibition (30–45\%) may interfere with the rehabilitation process and compromise the functional recovery, whereas a small amount of initial inhibition seems to allow for strength gains early in the rehabilitation process. This emphasizes the interest to perform quite early in the recovering period combined EMG and torque tests in order to detect potential inhibition that could delay the functional recovery. On the other hand, the underlying neurophysiological mechanisms of the observed activation deficits are not yet fully understood.
Intervention of several protective reflexes induced primarily by ACL strain (14, 28), but also by stretch of joint capsule and associated muscle-tendon complex (4, 14, 28, 38), may reduce the anterior pulling force on the tibia and knee joint, via the extensor muscle inhibition and hamstring activation. In case of ACL-deficiency, Beard et al. (29) reported an increased latency of the protective reflexes that persisted after ACL-reconstruction. Interestingly, evidence of reinnervation of free patellar autograft used for ACL-reconstruction has been recently reported in dogs by Barrack et al. (30). These authors suggested that a successful reconstruction includes a tendon graft reinnervation by mechanoreceptors, allowing an afferent feedback to stabilize the knee. It is noticeable that such an effective graft reinnervation, observed in 50% of the animals, took place at least 6 months post-surgery.

The variability in the relative content of mechanoreceptors and free nerve endings are large among different anatomical structures. It is therefore important to consider the delays inherent to the different afferent pathways. As shown in cat studies (31), nociceptors are innervated by type IV (C) afferent fibers with a slow conduction velocity (<1 m · s⁻¹), whereas mechanoreceptors are innervated by type III (A delta) afferent fibers with faster conduction velocity (2.5 to 20 m · s⁻¹). Nociceptor-induced reflex activation of the hamstring muscle group does not appear to be fast enough to counteract the anterior draw movement of the tibia during rapid leg extension. In addition, strain-induced reflex inhibition does not explain the present observation of EMG deficits that lasted in most subjects from the initiation till the end of contraction, both in isometric and isokinetic conditions. On the other hand, studies based on healthy subjects have revealed reflex inhibition of agonistic muscle activation in situations of high force production (24, 25), with a trend to be overcome in trained subjects (26). In the present study, however, the observed deficit in quadriceps activation was not more accentuated in the isometric strength test, which allows for the production of larger torques, than in the other testing conditions (Figure 2). This may be explained partly by the fact that the isometric extension test was performed at 65° of knee flexion, a position in which quadriceps contraction has been reported to produce no anterior or posterior tibial translation (27).

Among other stimuli that could sensitize free nerve endings, chemicals released during acute inflammation have been reported in cats to lead to hyper-excitability of spinal neurons for the afferent input from the inflamed knee as well as for the input from regions of the contralateral leg (31, 32). This effect is counteracted by progressive enhancement of the tonic descending inhibition (33, 34). In humans, both voluntary and reflex EMG inhibitions have been reported to last over a few days after eccentric exercise induced ultrastructural muscle damage (35). It is thus suggested that polymodal group III and IV muscle afferent fibers might also be involved and lead to a pre-synaptic inhibition of alpha motoneurons in case of knee effusion. This was not, however, the case in the present study as none of the patients experienced any knee pain and/or effusion. Finally, considering the protective antagonistic hamstring effect against the anterior tibial load (4, 14, 36), much emphasis has been put in rehabilitation programs on the hamstring strengthening after ACL rupture (37–39). It should be mentioned, however, that Bencke et al. (40) were unable to demonstrate a significant effect of a prophylactic training program on healthy athletes. In the present study, the hamstring co-activation of both patient groups was about 10% lower than in the Ctrl group in all tested conditions but remained in the 15–35% range reported in the literature on isokinetic strength tests (15). It is suggested, however, that due to the automatic braking of the leg lever arm by the dynamometer, isokinetic strength tests may
not be considered as the proper way to evaluate the exact co-activation level of natural lower limb movements.

A second hypothesis, based on the so-called “quadriceps avoidance gait pattern,” would be the expected neuromuscular disuse effect that would be learnt before the surgery when the knee was unstable (41). As an acute reconstruction is rarely performed, many patients walk without an intact ACL for some weeks or months before the surgery. This is expected to lead also to a progressive deficiency in locomotor type actions, thus favoring the occurrence of bilateral functional deficits. In support of this hypothesis, our results revealed extension and flexion torque deficits in the Op leg as well as in the Nop leg of both patient groups as compared to an age- and activity-matched Ctrl group. Similarly, Urbach et al. (18) reported moderate (~15%) but significant bilateral activation deficit in isometric strength test in unilateral ACL deficient patients as compared to a matched control group. These authors suggested that stabilization of the knee joint by ACL-reconstruction might eliminate voluntary activation deficits. This hypothesis is not supported, however, by the present activation deficits observed in both isometric and isokinetic strength tests of the Op leg 4 months post ACL-reconstruction (Figure 2). Similarly, Hurley et al. (23) reported close to 20% bilateral inhibition of the knee extensors 22 months post ACL-surgery. In both of these studies as well as in the present results (Figures 2 & 3), the inter-leg functional differences were found to be only minor. Based on the observed voluntary-activation deficit in the uninjured side, Urbach et al. (18) and Hurley et al. (23) suggested that functional tests might underestimate the exact deficit when the uninjured limb serves as reference. Although the present study did not reveal any significant activation deficit in the Nop leg of the patient groups, the observed large bilateral torque deficits would suggest also that functional tests might not be valid when the uninjured limb provides reference values. Supporting the interest of a functional comparison to normal subjects, De Vita et al. (42) reported normal walking kinematic patterns, although altered joint torque and power patterns, 6 months post ACL-reconstruction and accelerated rehabilitation. In activities such as walking, ramp descending, stair ascending and descending, running, and cross-cutting, Ciccotti et al. (43) reported greater knee extensor and flexor EMG activity in ACL-deficient patients as compared to those of uninjured subjects, whereas ACL-reconstructed subjects (2 to 3 years post-surgery) generally produced EMG profiles that were statistically similar to the normal subjects. Referring to Hurley et al. (13), it is suggested that the specific activation deficit in the knee extension muscles of the Op leg might compromise for a longer time period the functional recovery of the Op leg as compared to the other. Inter-leg differences in agonistic muscle inhibition is thus expected to interfere in the respective functional recovery of each leg. The present findings emphasize the fact that EMG analysis may reveal persisting neuromuscular deficits that could not be detected by kinetic or kinematic parameters alone.

**Conclusion**
The present study revealed bilateral extension and flexion torque deficits in ACL-reconstructed patient groups. This would suggest potential underestimation of the actual torque deficits in the Op leg when the analysis is based on inter-leg torque comparison only. On the other hand, the EMG analysis suggests that protective activation mechanisms might occur and would result in slower functional recovery, especially in the extensor muscle chain. In order to detect and counteract such a vicious circle, it is recommended that the strength and EMG of agonist and
antagonist muscles be measured, especially during forceful actions, around 6 months post-surgery.

References
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