Quadriceps Activation During Knee Extension Exercises in Patients With ACL Pathologies

Esther Suter, Walter Herzog, and Robert Bray

This study assessed muscle inhibition in patients with chronic anterior cruciate ligament (ACL) deficiency or ACL reconstruction. A series of protocols were tested for their effectiveness in increasing activity of the individual knee extensor muscles and decreasing muscle inhibition of the whole quadriceps group. Quadriceps muscle inhibition was measured by superimposing an electrical twitch onto the quadriceps muscle during a maximal voluntary knee extension. The level of activation of the individual knee extensor and knee flexor muscles was assessed via electromyography (EMG). Patients with ACL pathologies showed strength deficits and muscle inhibition in the knee extensors of the involved leg and the contralateral leg. Muscle inhibition was statistically significantly greater in ACL-deficient patients compared to ACL-reconstructed patients. When a knee extension was performed in combination with a hip extension, there was a significant increase, \( p < 0.05 \), in activation of the vastus medialis and vastus lateralis muscles compared to isolated knee extension. The use of an anti-shear device, designed to help stabilize the ACL-deficient knee, resulted in increased inhibition in the quadriceps muscle. Furthermore, a relatively more complete activation of the vasti compared to the rectus femoris was achieved during a fatiguing isometric contraction. Based on the results of this study, it is concluded that performing knee extension in combination with hip extension, or performing fatiguing knee extensor contractions, may be more effective in fully activating the vasti muscles than an isolated knee extensor contraction. Training interventions are needed to establish whether these exercise protocols are more effective than traditional rehabilitation approaches in decreasing muscle inhibition and achieving better functional recovery, including equal muscle strength in the injured and the contralateral leg.

Key Words: ACL deficiency, ACL reconstruction, muscle inhibition, electromyographic activity, exercise protocols

Introduction

Structural and functional recovery after anterior cruciate ligament (ACL) injury has been researched in great detail. The issues most commonly investigated are muscle
strength of the involved and the noninvolved limb (LoPresti, Kirkendall, Street, & Dudley, 1988; Lorentzon, Elmquist, Sjöström, Fagerlund, & Fugl-Meyer, 1989; Natri, Järvinen, Latvala, & Kannus, 1996), muscle activation patterns (Elmquist, Lorentzon, Johansson, & Fugl-Meyer, 1988; McNair & Wood, 1993), changes in muscle mass and fiber type distribution (Gerber, Hoppeler, Claassen, et al., 1985; LoPresti et al., 1988; Lorentzon et al., 1989), joint proprioception (Ochi, Iwasa, Uchio, Adachi, & Sunnen, 1999; Pap, Machner, Nebelung, & Awiszus, 1999), gait patterns (Devita, Hortobagyi, & Barrier, 1998), and degenerative aspects of joint structures (Gillquist & Messner, 1999). There is general agreement that rupture of the ACL constitutes a major insult to the integrity of the knee and results in functional adaptations to cope with pain and joint instability.

Standard rehabilitation programs after joint injury are mainly concerned with the restoration of muscle strength, range of motion, and joint proprioception. Despite these efforts, deficits in quadriceps strength are often observed long after the rehabilitation process has been completed (LoPresti et al., 1988; Lorentzon et al., 1989; Natri et al., 1996). In particular, weakness of the vastus medialis seems to persist. Exercise protocols to specifically strengthen this muscle have been explored (Karst & Jewitt, 1993; Laprade, Culham, & Brouwer, 1998; Salzman, Torburn, & Perry, 1993; Zakaria, Harburn, & Kramer, 1997). However, it appears that none of these protocols substantiated the notion that vastus medialis can be selectively activated.

Few studies have addressed the problem of muscle inhibition in patients with ACL pathologies (Hurley, Jones, Wilson, & Newham, 1992; Urbach, Nebelung, Wiler, & Awiszus, 1999). Muscle inhibition is defined as the inability to fully activate all motor units of a muscle or a muscle group during a maximal voluntary effort. The mechanisms causing the decreased motoneuron activation are not entirely understood. It is thought that sensory stimuli reflexively inhibit α-motoneurons and consequently impede voluntary muscle activation (Young, 1993). Pain and swelling in the knee joint have been shown to contribute to muscle inhibition, although muscle inhibition has been observed in the absence of any obvious symptoms (Young, 1993).

Muscle inhibition is generally measured using the interpolated twitch technique, which requires applying an electrical stimulation to the femoral nerve during a maximal voluntary knee extensor contraction (Belanger & McComas, 1981; Hurley et al., 1992; Suter & Herzog, 1997). In case of muscle inhibition, the electrical stimulation will increase the knee extensor moment. Mild to moderate muscle inhibition has been found in patients with isolated ACL rupture (Hurley et al., 1992; Urbach et al., 1999), and muscle inhibition changed little following standard rehabilitation (Hurley et al., 1992).

We found substantial muscle inhibition and knee extensor weakness in patients with anterior knee pain (Suter, Herzog, & Bray, 1998; Suter, Herzog, DeSouza, & Bray, 1998). In this patient population, medical and surgical interventions improved subjective symptoms, but muscle inhibition and strength deficits remained (Suter et al.). From these results, it is hypothesized that persistent muscle inhibition may be responsible for the long-term strength deficits measured in ACL-deficient patients, which have traditionally been attributed to muscle atrophy. Furthermore, there is recent evidence that muscle inhibition may affect force production during normal locomotion: experimentally induced knee effusion was found to cause muscle inhibition and gait alterations such as reduced knee extensor torque (Torry, Decker, Viola, O’Connor, & Steadman, 2000). The adaptations were similar to the ones frequently observed in ACL-deficient patients (Berchuck, Andriacchi, Bach, & Reider, 1990; Devita et al., 1998).
It has been suggested that muscle inhibition must be overcome before full functional recovery can be achieved (Hurley et al., 1992; Lorentzon et al., 1989). However, little research has been conducted to investigate how muscle inhibition may be reduced or eliminated during the rehabilitation process. Standard rehabilitation protocols appear to be ineffective in eliminating muscle inhibition.

The present study was aimed at assessing muscle inhibition in patients with chronic ACL deficiency or ACL reconstruction. A series of protocols were tested for their effectiveness in increasing activation of the individual knee extensors and decreasing muscle inhibition of the quadriceps group.

The following hypotheses were put forward: Patients with chronic ACL pathologies show a significant amount of muscle inhibition in the quadriceps group. Furthermore, activation level of the knee extensors will increase compared to an isometric knee extension (a) when a knee extension is performed in combination with a hip extension or a hip flexion, (b) when an anti-shear device is used during an isometric knee extension, and (c) during a fatigue protocol, near the end of a sustained submaximal knee extension. As activation of the knee extensor muscles increases, muscle inhibition of the quadriceps is expected to decrease.

### Methods

Twenty-four patients with ACL deficiency and 22 with ACL-reconstruction were recruited through the database of the local sport medicine clinic. The patients were tested on average 44 months ($SD = 66$ months) after ligament rupture (ACL-deficient) and 22 months ($SD = 40$ months) after ligament reconstruction (ACL-reconstructed). Patients gave written informed consent to participate. The study protocol was approved by the Conjoint Medical Ethics Committee of the University of Calgary.

Muscle inhibition was assessed using the interpolated twitch technique (Belanger & McComas, 1981; Suter & Herzog, 1997). An electrical twitch was applied to the femoral nerve approximately 1 second after the subjects reached the force plateau during maximal isometric knee extensions. If muscle activation is incomplete, the electrical stimulation produces an increase in torque. The magnitude of this interpolated twitch torque (ITT) is representative of the amount of muscle inhibition.

<table>
<thead>
<tr>
<th>Table 1 Descriptive Data (means ± SD) for the Anterior Cruciate Ligament (ACL)-Deficient and the ACL-Reconstructed Patients</th>
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<tr>
<td><strong>ACL Deficient</strong> ($n = 24$: 18 M, 6 F)</td>
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<td>Age (yrs)</td>
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*Time in months.
Our setup allows for analysis of forces less than 0.5% of the total voluntary force. A Grass S88 stimulator in combination with an isolation unit approved for human use (Quincy, MA) was used for nerve stimulation. Carbon-impregnated rubber electrodes (4.5*10 cm) were thinly coated with a conductive gel and secured to the thigh with adhesive tape. The stimulating electrode was placed over the femoral nerve just distal to the inguinal ligament; the second electrode was placed over the distal portion of the quadriceps muscle. Double square-wave pulses of 240V (maximum), 0.8 ms duration, and 8 ms interpulse interval were used for stimulation. Prior to testing, electrical stimuli of increasing voltage up to 240V were applied to the nerve to familiarize the participants with the stimulation procedure and to ensure maximal nerve stimulation.

The ITT was compared to the torque produced by the relaxed quadriceps muscle when stimulated by an identical electrical twitch as that used during the voluntary contraction, referred to as resting twitch torque (RTT). Muscle inhibition was calculated as \((\text{ITT/RTT})*100\).

Isometric knee extensor moments were measured using a Cybex dynamometer (Cybex Norm, Testing & Rehabilitation System, Lumex Inc., New York). Participants were seated with a knee angle of 30° from full extension and a hip angle of 85°. The lateral epicondyle of the femur was aligned with the axis of rotation of the dynamometer and the thighs were firmly secured with straps. The participants were asked to slowly build up force to reach maximum levels and then hold the maximum contractions for 3 or 4 s. The knee extensor moment produced was displayed on-line for visual feedback.

Electromyographic (EMG) activity of the vastus lateralis, vastus medialis, rectus femoris, semimembranosus, and biceps femoris was recorded. Bipolar Ag-AgCl surface electrodes of 1 cm active diameter were placed on the belly of the respective muscles. Electrodes were placed along the estimated direction of the muscle fibers. The inter-electrode distance was 3 cm. A common ground electrode was placed on the patella. Before the electrodes were attached, the skin was shaved and cleaned according to standard procedures and skin resistance was kept below 5kΩ. EMG signals were passed through an amplifier located 10 cm from the electrodes, and a bandpass filter with cutoff frequencies of 10 Hz and 700 Hz was used.

Knee extensor moments and EMG signals were sampled at 2000 Hz using an analog-to-digital board with a resolution of 12 bits, and stored on an IPC 486DX for further signal analysis.

Participants underwent three trials of maximal isometric knee extensions at 30° from full extension using four protocols: (a) isolated knee extension, (b) knee extension in combination with hip extension, (c) knee extension in combination with hip flexion, and (d) knee extension using an anti-shear device. The isolated knee extension was used as the reference contraction against which the three other conditions were compared. For the knee extension/hip extension protocol, participants were instructed to focus on extending the hip while maximally contracting the knee extensor muscles. For the knee extension/hip flexion protocol, they were asked to focus on producing a flexor force in the hip while maximally contracting the knee extensors.

The anti-shear device consisted of a dual pad attachment with the second pad positioned at the proximal end of the tibia. It is thought that this proximal pad prevents anterior translation of the tibia relative to the femur during knee extension, and therefore reduces shear stress in the knee (Johnson, 1982). In contrast, the standard device consisted of a single pad at the distal end of the tibia. For all protocols, the hip angle did not change since the participants were restrained at the hip and shoulder. They were verbally encouraged during all trials to facilitate maximal efforts.
Prior to testing, the protocol was carefully explained to the participants and submaximal contractions were performed to familiarize them with the procedures. During the contractions, maximal knee extensor moments, knee extensor inhibition using the interpolated twitch technique, and EMG activity of the vastus lateralis, vastus medialis, rectus femoris, semimembranosus, and biceps femoris were measured. All four conditions and both legs were tested in random order. A 2-min rest was enforced between contractions to avoid fatigue.

The second part of the study consisted of a fatigue test. Participants sustained a submaximal isometric contraction of 75% maximal voluntary effort (MVC) until exhaustion. They performed a maximal isometric knee extension, and the peak force was marked on an oscilloscope. Then a target line was set at 75% MVC for feedback to the participant. This line had to be matched by a second line, which moved as a result of the individual’s contractions. The test was terminated when the force level could no longer be maintained, i.e., the two lines no longer matched. EMGs of vastus lateralis, vastus medialis, rectus femoris, semimembranosus, and biceps femoris, and knee extensor moments were measured for 2-s periods every 5 seconds during the sustained contraction. An electrical twitch was applied at the end of every 2-s recording interval in order to assess muscle inhibition.

The following statistics were performed: Muscle inhibition was calculated as (ITT/RTT) * 100%. From the EMG data recorded during the maximal knee extensions, 1-s segments were used to calculate the root mean square (RMS) values of the vastus lateralis, vastus medialis, rectus femoris, semimembranosus, and biceps femoris. The segments were taken during the force plateau just before applying the interpolated twitch. The EMG RMS values of the knee extension test were taken as 100% and the RMS for the other three conditions were expressed as a percentage of the knee extension values. The best trial in terms of knee extensor moment for each condition was used for statistical analysis.

ANOVA was used to compare values for knee extensor moments, muscle inhibition, and EMG RMS for both legs of the ACL-deficient group and ACL-reconstructed group for all four conditions. Since ANOVAs revealed significant effects for pathology (i.e., ACL deficient vs. ACL reconstructed) and pathology × condition interactions for some of the variables, analyses of muscle inhibition, knee extensor moments, and EMG RMS were conducted separately for the ACL-deficient group and the ACL-reconstructed group.

For the fatigue test, 1-s segments taken from the middle section of the 2-s recording intervals were used for statistical analyses. Values for muscle inhibition, knee extensor moment, and EMG RMS were averaged for the first, middle, and last third of the fatigue test. An ANOVA for repeated measures was used to test for changes in muscle inhibition, knee extensor moments, and EMG RMS over time. A significance level of $\alpha = .05$ was employed for all tests.

**Results**

ACL-deficient and ACL-reconstructed patients showed muscle inhibition in both legs during the maximal knee extensions (Figure 1), which was significantly, $p < 0.05$, greater than in normal controls (Suter & Herzog, 1997). Muscle inhibition in the ACL-deficient group was significantly greater compared to the ACL-reconstructed group. The involved legs in both groups had greater muscle inhibition (Figure 1) and less knee extensor moments (Figure 2) than the contralateral legs.
Figure 1 — Muscle inhibition (mean ± SD) at baseline for involved legs and noninvolved legs of ACL-deficient and ACL-reconstructed patients. *Significant difference between ACL-deficient and ACL-reconstructed group. Horizontal line indicates mean muscle inhibition values found in a healthy population (Suter & Herzog, 1997).

Figure 2 — Knee extensor moments (mean ± SD) at baseline for involved and contralateral legs of ACL-deficient and ACL-reconstructed patients. *Significant difference between involved and contralateral legs.
Figure 3 — Muscle activation (mean ± SD) in the ACL-deficient (top) and ACL-reconstructed (bottom) patients during different exercise protocols. Values are EMG root mean square (RMS) normalized to knee extension protocol. *Statistically significant differences compared to knee extension protocol, indicated as horizontal line.
In the ACL-deficient patients, the knee extension/hip extension protocol resulted in statistically significantly increased EMG RMS values for the vastus lateralis and vastus medialis (Figure 3, top) compared to the EMG RMS value of the isolated knee extension (indicated as black line). Also, EMG RMS values for the semimembranosus and the biceps femoris for the knee extension/hip extension protocol was significantly greater than for the knee extension protocol. The knee extension/hip flexion protocol and the anti-shear device protocol did not result in any statistically significant changes in EMG activity. In the ACL-reconstructed patients, the only statistically significant changes were a reduction in vastus medialis activity for the knee extension/hip flexion and anti-shear device conditions, and an increase in semimembranosus activity during the knee extension/hip extension protocol compared to the knee extension condition (Figure 3, bottom).

None of the protocols led to a statistically significant reduction in muscle inhibition in the ACL-deficient (Figure 4, top) or the ACL-reconstructed (Figure 4, bottom) group. In contrast, the anti-shear device resulted in a statistically significantly greater muscle inhibition in both groups. With the exception of the lower knee extensor moments for the ACL-reconstructed group when performing the knee extension/hip extension protocol, knee extensor moments did not change significantly.

Muscle inhibition and knee extensor moments showed a statistically significant decrease throughout the sustained submaximal contraction (Figure 5, top). The values are calculated for ACL-deficient and ACL-reconstructed patients combined and represent means and standard deviations for the early, middle, and late stages of the sustained contraction. The holding time during the fatigue test varied across participants from 16 to 144 seconds in the injured leg, and from 20 to 124 seconds in the contralateral leg.

EMG RMS values increased statistically from the first third to the second third of the submaximal contraction, then remained constant from the middle third to the end of the test (Figure 5, middle). The increase in EMG RMS was statistically significant for all three knee extensors of the involved leg, and for the vasti muscles of the contralateral leg. The vasti muscles showed a proportionally greater increase in activation than the rectus femoris. Antagonist muscle activity increased significantly toward the end of the fatigue test (Figure 5, bottom).

**Discussion**

Chronic quadriceps weakness is a well recognized problem after ACL injuries (Gerber et al., 1985; Gillquist & Messner, 1999; LoPresti et al., 1988; Lorentzon et al., 1989; Natri et al., 1996). Nonoptimal muscular activation patterns are thought to partially account for the strength deficits (Elmquist et al., 1988; Lorentzon et al., 1989); however, systematic investigations of the relationship between strength deficits and muscle activation are lacking. The goal of the present study was twofold: (a) to assess muscle inhibition in chronic ACL-deficient and ACL-reconstructed patients, and (b) to test whether specific exercise protocols would increase knee extensor activation and therefore decrease or eliminate muscle inhibition in the quadriceps muscles.

ACL-deficient and ACL-reconstructed patients showed a substantial amount of knee extensor inhibition that was significantly higher compared to healthy subjects (Suter & Herzog, 1997). The finding of bilateral knee extensor inhibition concurs with our observations from anterior knee pain patients (Suter, Herzog, & Bray, 1998; Suter, Herzog, DeSouza, & Bray, 1998), and indicates that a unilateral joint injury may affect the functional capacity of the noninjured, contralateral leg. The greater average muscle inhibition in the involved leg compared to the contralateral leg was associated with a marked strength deficit in the involved leg.
Persistent muscle weakness in the involved leg after ACL injury has been observed in previous studies, with strength deficits ranging from 11 to 29% between the injured and contralateral legs (LoPresti et al., 1988; Lorentzon et al., 1989; Natri et al., 1996). Differences in muscle mass and fiber type composition between the injured and contralateral legs could not fully explain the strength deficit in these studies. Our results confirm that maximal activation is impaired in persons with chronic ACL pathologies, and they further support the notion that part of the muscle weakness observed after joint injury may be due to muscle inhibition.

The reasons for the significantly higher amount of muscle inhibition in the ACL-deficient group compared to the ACL-reconstructed group can only be speculated
Figure 5 — (top): Knee extensor moments and muscle inhibition during early, middle, and late stages of fatigue test. Knee extensor moments and muscle inhibition were calculated as % of maximal knee extensor contraction. *Statistically significant decrease in knee extensor moment and muscle inhibition from early to late stage.

—(middle): EMG root mean square (RMS) values for vastus lateralis, vastus medialis, and rectus femoris during early, middle, and late stages of fatigue test, calculated as % of maximal knee extensor contraction. *Statistically significant increase in EMG RMS from early to middle stage for vastus lateralis and vastus medialis. # Statistically significant increase in EMG RMS from early to middle stage for all 3 knee extensors. & Statistically significant increase in EMG RMS from middle to late stage for both knee flexors.

—(bottom): EMG RMS values for semimembranosus and biceps femoris during early, middle, and late stages of fatigue test, calculated as % of maximal knee extensor contraction. *Statistically significant increase in EMG RMS from early to middle stage for both knee flexors. +Statistically significant increase in EMG RMS from middle to late stage for biceps femoris. &Statistically significant increase in EMG RMS from middle to late stage for both knee flexors.

Note: Data in all three sections are means (± SD) for ACL-deficient and ACL-reconstructed patients combined.
Quadriceps Activation

upon. In the ACL-deficient knee, episodes of “giving way” may lead to reinjury and joint swelling which have been associated with increased muscle inhibition (Young, 1993). Also, in ACL-deficient patients, consistent deficits in joint position sense and motion have been found (Ochi et al., 1999; Pap et al., 1999). The sensory system of the ACL is thought to contribute significantly to the functional stability of the knee joint (Johansson, Sjölander, & Sojka, 1991). Somatosensory evoked potentials were found to be smaller or absent in the ACL remnants compared to the reconstructed or intact ACL (Ochi et al., 1999), indicating less sensory function in the ACL remnants compared to the reconstructed or intact ACL.

It has been demonstrated that sensory function recovers after ACL reconstruction, while in ACL-deficient patients the sensory deficits remained (Ochi et al., 1999). Therefore, the ACL not only serves as a mechanical stabilizer of the knee joint but also appears to have feedback function to protect the joint from abnormal mechanical stress (Johansson et al., 1991; Ochi et al., 1999). A lack of appropriate sensory feedback combined with joint instability may result in repetitive minor joint injury (Gillquist & Messner, 1999) and account for the larger amount of muscle inhibition in ACL-deficient patients compared to ACL-reconstructed patients.

The long-term consequences of persistent muscle inhibition and strength deficits are not known (Suter & Herzog, 2000). It is possible that deficits in knee extensor strength result in altered joint loading, which in turn has been associated with increased incidence of osteoarthritis in animal models (Brandt, 1997). The results of a recent prospective study suggest that quadriceps weakness may predispose the joint to premature degeneration (Slemenda, Heilman, Brandt, et al., 1998).

Adaptations in gait have been observed in ACL-deficient and ACL-reconstructed subjects. Reduced knee extensor torque and excessive hip extension torque have been measured in some (Berchuck et al., 1990; Devita et al., 1998), but not all (Roberts, Rash, Honaker, Wachowiak, & Shaw, 1999) patients with ACL pathologies. It has been argued that this altered gait pattern might be a result of reduced knee extensor and increased hamstring activity during walking (Berchuck et al., 1990; Devita et al., 1998).

In an experimental study, applying saline injections into the knee joint of healthy subjects induced a gait pattern similar to the one observed in patients with ACL pathologies (Torry et al., 2000). In particular, there was a decrease in torque and work produced at the knee. EMG results confirmed a significantly decreased activity of the quadriceps muscles, with vastus medialis showing the largest decrease (Torry et al., 2000).

Joint distension has been shown previously to produce inhibition of the quadriceps motoneurons in a dose dependent manner (Iles, Stokes, & Young, 1990). But the association between muscle inhibition in the quadriceps and alterations in gait pattern is new. In a study looking at quadriceps function and gait patterns, paralysis of the quadriceps muscle evoked a drastic increase in heel strike transient (Jefferson, Radin, & O’Connor, 1990). Repetitive impulse loading, such as occurs during heel strike, has been related to the etiology of osteoarthritis (Jefferson et al., 1990).

In summary, it appears that muscle inhibition persists for extended time periods in patients with ACL pathologies. Muscle inhibition may be responsible for persistent knee extensor weakness and alterations in kinetic and kinematic components during normal walking, which may be associated with premature degeneration of the knee joint.

The second part of this study was aimed at testing a series of exercises that were thought to facilitate excitatory pathways of the knee extensors (Fujiwara & Basmajian, 1975). For ACL-deficient patients, performing a knee extension in combination with a hip extension did increase the activity of the vastus medialis and lateralis by approxi-
mately 13% and 11%, respectively, compared to an isolated knee extension. The lack of a significant increase in knee extensor moments, which would have been expected based on the increased activity of the knee extensor group, may be explained by the increased activation level of the hamstring muscles observed during the hip extension protocol.

There is a general consensus that strengthening of the quadriceps muscles, specifically the vastus medialis, should be a primary focus after knee pathology (Karst & Jewett, 1993; Laprade et al., 1998; Salzman et al., 1993; Zakaria et al., 1997). However, there is disagreement as to whether individual components of the quadriceps muscle can be activated preferentially. A number of studies focused on selectively increasing the activity of the vastus medialis by combining quadriceps contraction with hip adduction (Laprade et al., 1998; Zakaria et al., 1997), internal and external hip rotation (Karst & Jewett, 1993), or using different combinations of hip and knee angle (Salzman et al., 1993). In general, these studies did not find a significant increase in the ratio of vastus medialis to vastus lateralis recruitment, although differences in the total amount of activation were observed for specific exercise conditions.

Our results tend to support these findings, as vastus medialis and vastus lateralis showed a similar increase in activity (13% vs. 11%) when knee extension was combined with hip extension. Although it may not be possible to isolate vastus medialis activation from the other knee extensor muscles, performing a knee extension in combination with a hip extension may offer a more effective strengthening of the whole quadriceps group than a standard knee extension protocol. It remains to be tested in an intervention program whether an exercise protocol that combines knee extension with hip extension will help to improve knee extensor activation and reduce muscle inhibition, and lead to a more successful recovery of muscle strength.

In the ACL-reconstructed patients, none of the protocols turned out to be effective in increasing knee extensor activity. It is speculated that the better muscle function, reflected by the significantly lower muscle inhibition in these patients compared to the ACL-deficient group, may have partially accounted for the lack of increase in EMG activity. It may be that the knee extension/hip extension protocol is only effective in increasing knee extensor activation when the ability to activate the muscle is severely limited, such as observed in the ACL-deficient group.

A surprising result was found when the knee extensions were performed with a Johnson anti-shear device. When using this device, which has a second, proximal tibial pad, muscle inhibition in both legs was statistically significantly greater compared to tests that employed the standard attachment with only a single pad at the distal end of the tibia. This result is contrary to what we expected. Our hypothesis was based on the findings of Solomonow, Baratta, Zhou, et al. (1987), who observed a marked anterior translation of the tibia relative to the femur during isokinetic knee extensor contractions in ACL-deficient patients. In that study, the tibial translation occurred at about 45° from full extension and was accompanied by a drop in quadriceps EMG, an increase in hamstring EMG, and a simultaneous decrease in force, suggesting a reflexive inhibition of the quadriceps induced by the tibial translation. By using an anti-shear device with a second pad at the proximal end of the tibia, shear forces in the knee are assumed to be reduced and translation of the tibia is thought to be prevented (Johnson, 1982).

We expected this more stable position to result in decreased muscle inhibition, particularly at the 30° knee angle tested. Studies comparing results from the single pad and dual pad device only focused on moment output and did not consider muscle activation. Li, Chan, and Hsu et al. (1993) documented significantly lower knee extensor moments during knee extension when the dual pad device was used.
This is in line with the findings from Nisell and co-workers (Nisell, Ericson, Nemeth, & Ekholm, 1989), who found lower knee extensor moments for flexion angles of less than 65º when a single pad was moved from the distal end of the tibia to the proximal end. Nisell et al. offered two possible explanations for this finding. The first was of a biomechanical nature and related to a possible shortening of the patellar tendon moment arm due to the increased force of the resistance pad when the pad is moved proximally. The second explanation suggested that the greater resistance pad forces may induce subliminal pain or other inhibiting influences from the cutaneous and/or periosteal mechano-receptor afferents, which may in turn reflexively inhibit the activation of the extensor motoneurons (Nisell et al., 1989). Since muscle activation was not assessed in that study, no direct confirmation of this hypothesis was provided.

The large increase in muscle inhibition when using the proximal pad in our protocol would tend to support the second hypothesis. However, the increase in muscle inhibition during the anti-shear device protocol was not accompanied by a decrease in knee extensor moment. This result suggests that the contraction may have been performed in a mechanically more advantageous position, which gave similar knee extensor moments for less knee extensor force. From a clinical point of view, the increased muscle inhibition when using the anti-shear device may be important. If the primary purpose of the exercise is to provide maximal safety and stability during the exercise, the anti-shear device may be a good choice for patients with ACL pathologies. If the goal is to maximally activate the quadriceps muscles during knee extension exercises, the proximal pad may prevent an optimal training effect.

The last part of this study was aimed at overcoming muscle inhibition in the knee extensors using a submaximal fatigue protocol. It was speculated that during the end-stages of the test, when force production starts to fail, motor units that are inhibited during a maximal contraction might be fully activated in order to maintain the required force level. This increased motor unit activation should then be reflected by a decrease in the interpolated twitch torque.

EMG RMS values of all three knee extensor muscles showed the expected increase in amplitude, as the contraction was sustained. Interestingly, the vastus medialis and vastus lateralis of the injured legs showed the largest increase in activation. The rectus femoris, particularly in the contralateral leg, showed a smaller change in activation than the vasti muscles throughout the contraction. The differences in muscle activation indicate that the relative contribution of the knee extensors to the total knee extensor moment may change throughout a fatiguing task. The vasti seem to make a larger relative contribution to the force production during the later stages of the contraction than during the early stages.

It has been hypothesized that the knee extensors, based on physiological parameters such as fiber type distribution, may have different fatigue rates, which could be used to selectively strengthen a particular muscle group (Grabiner, Koh, & Miller, 1991). However, the findings in normal healthy subjects did not support this hypothesis. The results of the present study indicate that the situation in a dysfunctional knee extensor group may be different than in an intact knee joint, and that a fatiguing exercise may indeed be more effective in selectively strengthening the vasti muscles.

Despite the increase in muscle activation during the sustained contraction, a substantial interpolated twitch torque could be evoked until the very end. The interpolated twitch at the end of the fatigue test was higher than the one measured during the maximal voluntary contractions. This suggests there was muscle inhibition at the end of the task, and that complete activation of the knee extensors was not achieved in
these patients with severe muscle inhibition. This finding may be confounded by lack of motivation on the part of the study population, resulting in submaximal efforts as fatigue sets in. For untrained, nonathletic subjects, an all-out fatigue test constitutes an unusual and strenuous task, and true maximal efforts are hard to achieve despite verbal encouragement and visual feedback to them. In a motivated athletic population, submaximal sustained contractions may be more effective in fully activating inhibited knee extensor muscles.

In summary, the present study confirmed that patients with ACL pathologies show substantial functional deficiencies, such as weakness and inhibition of the knee extensors in the involved and the contralateral legs. Muscle inhibition was significantly greater in the ACL-deficient patients than in the ACL-reconstructed patients. By performing knee extension in combination with hip extension, a significantly increased activation of the vastus medialis and vastus lateralis muscles occurred compared to an isolated knee extension in the ACL-deficient group. The use of an anti-shear device, designed to provide stability to the ACL-deficient knee, did not improve muscle activation but resulted in increased muscle inhibition. Furthermore, a relatively more complete activation of the vasti muscles compared to the rectus femoris was achieved during a submaximal sustained isometric contraction.

These results suggest that knee extensions performed in combination with hip extensions or fatiguing contractions may be more appropriate than knee extensor contractions alone when optimal knee extensor activation is the primary goal. This suggestion should be tested in a future intervention study. By increasing activation of the individual knee extensor muscles, muscle inhibition of the whole extensor group should be reduced or eliminated over time. As a result, full functional recovery, including equal muscle strength in the injured and the contralateral leg, may be achieved.

References


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