Human Movement Science 44 (2015) 234–245

Contents lists available at ScienceDirect

Human Movement Science

journal homepage: www.elsevier.com/locate/humov

Lower extremity muscle activation onset times during the transition from double-leg stance to single-leg stance in anterior cruciate ligament injured subjects

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ARTICLE INFO

Article history:
Received 15 April 2015
Revised 16 September 2015
Accepted 17 September 2015

Keywords:
Anterior cruciate ligament injury
Neuromuscular control
Lower extremity
Double-leg stance
Single-leg stance

ABSTRACT

The goal of this study was to evaluate muscle activation onset times (MAOT) of both legs during a transition task from double-leg stance (DLS) to single-leg stance (SLS) in anterior cruciate ligament injured (ACLI) ($n = 15$) and non-injured control subjects ($n = 15$) with eyes open and eyes closed. Significantly delayed MAOT were found in the ACLI group compared to the control group for vastus lateralis, vastus medialis obliquus, hamstrings medial, hamstrings lateral, tibialis anterior, peroneus longus and gastrocnemius in both vision conditions, for gluteus maximus and gluteus medius with eyes open and for tensor fascia latae with eyes closed. Within the ACLI group, delayed MAOT of tibialis anterior with eyes open and gastrocnemius with eyes closed were found in the injured leg compared to the non-injured leg. All other muscles were not significantly different between legs. In conclusion, the ACLI group showed delayed MAOT not only around the knee, but also at the hip and ankle muscles compared to the non-injured control group. No differences between both legs of the ACLI group were found, except for tibialis anterior and gastrocnemius. These findings indirectly support including central nervous system re-education training to target the underlying mechanisms of these altered MAOT after ACL injury.

1. Introduction

Tears of the anterior cruciate ligament (ACL) continue to be one of the most devastating sports injuries of the lower extremity, leading to a long and often difficult rehabilitation. The development of adequate dynamic knee joint stability after ACL injury is largely dependent on an optimal functioning of the neuromuscular system and is of crucial importance for rehabilitation (Williams, Chmielewski, Rudolph, Buchanan, & Snyder-Mackler, 2001). To optimize rehabilitation approaches

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http://dx.doi.org/10.1016/j.humov.2015.09.007
0167-9457/

and improve clinical outcomes after ACL injury, it is therefore essential to enhance our understanding of neuromuscular control deficits after ACL injury (Wikstrom, Tillman, Chmielewski, & Borsa, 2006; Williams et al., 2001).

Most of the previous literature measuring muscle activation patterns following ACL injury focused on the performance of muscles surrounding the injured knee joint (hamstrings, quadriceps and gastrocnemius) during a variety of experimental tasks (Ingersoll, Grindstaff, Pietrosimone, & Hart, 2008). An adaptive motor pattern that involves an increased hamstrings activity (Aalbersberg, Kingma, & van Dieen, 2009; Boerboom et al., 2001; Branch, Hunter, & Donath, 1989; Ciccotti, Kerlan, Perry, & Pink, 1994; Houck, Wilding, Gupta, De Haven, & Maloney, 2007; Swanik, Lephart, Giraldo, DeMont, & Fu, 1999; Swanik, Lephart, Swanik, Stone, & Fu, 2004) and decreased quadriceps activity (Branch et al., 1989; Gauffin & Tropp, 1992; Houck et al., 2007; Lynch, Logerstedt, Axe, & Snyder-Mackler, 2012; Swanik et al., 1999) has been observed in ACL injured (ACLI) subjects. This neuromuscular strategy may be the result of a series of complex neurophysiological alterations after ACL injury with the purpose to prevent excessive loads around the injured knee (Branch et al., 1989; Pietrosimone, McLeod, & Lepley, 2012; Swanik et al., 1999). However, a substantial variety between individual muscle activation patterns may exist, which can be related to the functional status after injury (Boerboom et al., 2001; Bryant, Newton, & Steele, 2009; Chmielewski, Rudolph, & Snyder-Mackler, 2002; Houck et al., 2007; Rudolph, Axe, Buchanan, Scholz, & Snyder-Mackler, 2001). Apart from the amount of activity of specific muscles, a precisely timed and coordinated muscle activation is also imperative to develop and maintain optimal joint stability during functional activities (Comerford & Mottram, 2001; Hewett, Zazulak, Myer, & Ford, 2005; Wikstrom et al., 2006) and avoid the development of maladaptive movement patterns which may predispose the ACLI subjects to an early onset of osteoarthritis and functional limitations (Kaplan, 2011; Trulsson, Miller, Hansson, Gummesson, & Garwicz, 2015), even when an ACL reconstruction is performed (Goerger et al., 2015). A delayed muscle activation of the hamstrings (Beard, Kyberd, Fergusson, & Dodd, 1993; Wojtys & Huston, 1994), quadriceps and gastrocnemius (Wojtys & Huston, 1994) in response to the application of an anterior tibial force was found in ACLI subjects. In contrast, other studies reported an earlier hamstring (Kalund, Sinkjaer, Arendt-Nielsen, & Simonsen, 1990; Sinkjaer & Arendt-Nielsen, 1991), quadriceps (Sinkjaer & Arendt-Nielsen, 1991) and gastrocnemius (Lindstrom, Fellander-Tsai, Wredmark, & Henriksson, 2009; Sinkjaer & Arendt-Nielsen, 1991) activation during walking after ACL injury, or no significant differences in timing of muscle activation during a single-leg hop for distance (Bryant et al., 2009). Klyne, Keays, Bullock-Saxton, and Newcombe (2012) reported a prolonged activity of medial gastrocnemius during a single-leg hop test in ACLI subjects. The variety of experimental tasks, methodologies and time since injury in these previous studies may have contributed to this lack of consistent evidence concerning the timing of muscle activation after ACL injury. Furthermore, the effects on muscle activation patterns of the non-injured leg remain largely unknown. Bilateral neuromuscular quadriceps deficits after unilateral ACL injury have been reported (Urbach, Nebelung, Weiler, & Awiszus, 1999), which may be attributed to alterations in the organization of the central nervous system (CNS) after ACL injury (Baumeister, Reinecke, Schubert, & Weiss, 2011; Baumeister, Reinecke, & Weiss, 2008; Grooms, Appelbaum, & Onate, 2015; Kapreli et al., 2009; Needle et al., 2014).

Muscle activation patterns around the proximal (hip) and distal (ankle) lower extremity joints after ACL injury are less studied, despite the suggestions that alterations in neuromuscular control may occur at different joints of the lower extremity after peripheral joint injury (Riemann & Lephart, 2002b). In subjects with chronic ankle instability for example, it has been shown that muscle activation onset times were delayed not only at the ankle but also at the hip and hamstring muscles during the transition from double-leg stance (DLS) to single-leg stance (SLS) compared to non-injured control subjects (Van Deun et al., 2007). During the transition from DLS to SLS, lower limb posture of the upcoming stance limb needs to be controlled by muscles functioning in the frontal and transversal plane, and not exclusively by muscles functioning in the sagittal plane, which is consistent with the multidirectional function of the ACL (Quatman et al., 2014). The stabilizing muscles of the hip, including gluteus maximus and gluteus medius, play an important role in controlling lower extremity and pelvic postures and movements during functional activities especially in the frontal and transversal plane (Ford et al., 2015; Willson, Ireland, & Davis, 2006; Zazulak et al., 2005), but the changes that may occur in these muscles following ACL injury remain unclear. With respect to the ankle muscles, an increase in the duration of tibialis anterior activity during gait (Lindstrom et al., 2009) and an increased tibialis anterior activity during a variety of functional activities (Ciccotti et al., 1994) were previously reported in ACLI subjects, possibly as a compensatory mechanism in the transversal plane by decreasing foot pronation and subsequent tibial internal rotation (Ciccotti et al., 1994).

Based on these earlier mentioned shortcomings and mixed findings in literature, there is a need to investigate neuromuscular control deficits within the lower extremity of both the injured and non-injured leg after ACL injury. Therefore, the goal of this study is to investigate lower extremity muscle activation onset times after ACL injury during a transition task from DLS to SLS during eyes open and eyes closed conditions. Our first hypothesis was that the ACLI group would show delayed muscle activation onset times compared to a non-injured control group, not only in muscles surrounding the knee, but also at the hip and ankle. Second, we hypothesized that these differences would become more apparent during the eyes closed condition, as subjects with an ACL injury may become more dependent on visual information to perform postural control tasks as a result of the altered proprioceptive information coming from the knee after ACL injury (Dingenen et al., 2015a; Grooms et al., 2015). Third, we hypothesized that no significant differences between the injured and non-injured leg of the ACLI group would be found, based on the earlier mentioned possible alterations in the CNS and the assumption that muscle activation patterns during voluntary postural control tasks are centrally mediated (Bouisset & Do, 2008).

2. Materials and methods

2.1. Subjects

The same subjects were tested as in a previous study of our group (Dingenen et al., 2015a). Thirty subjects participated in this study. Before participating in the study, all subjects read and signed an informed consent form, which was approved by the local ethical committee. The ACLI group (n = 15) included subjects with (1) a unilateral complete ACL injury confirmed by magnetic resonance imaging exams and (2) a passive knee range of motion of at least 120° (Dingenen et al., 2015a). Exclusion criteria were (1) a history of previous lower extremity or low back surgery, (2) reporting severe or extreme knee pain on the Knee Injury and Osteoarthritis Outcome Score (KOOS) questionnaire, (3) moderate or severe knee joint effusion at the time of data collection (International Knee Documentation Committee (IKDC) grade C or D), (4) meniscal injuries exceeding 1/3 of the meniscus, (5) other complete ligamentous knee injuries, (6) reporting a subjective feeling of "giving way" at the ankle (ankle instability), (7) knee osteoarthritis (Kellgren–Lawrence > grade 1) and (8) reporting ankle, hip or low back pain (Dingenen et al., 2015a). The time after ACL injury was 1.4 ± 0.7 months (range: 0.4–2.7 months). All tests were done immediately before the planned ACL reconstruction. No structured rehabilitation was performed before the measurements. From all ACL injuries, 13 were caused by a non-contact injury. Four subjects were injured on the dominant and 11 on the non-dominant leg. The dominant leg was defined as the preferred leg to kick a ball. The control group (n = 15) included subjects with no history of ankle, knee, hip or low back injury (Dingenen, Staes, & Janssens, 2013; Dingenen et al., 2015a). Subjects younger than 18 and older than 55 years old, and with the following conditions were also excluded: Parkinson, multiple sclerosis, cerebrovascular accident, peripheral neuropathies, circulation disorders, serious joint disorders (rheuma, osteoarthritis, etc.) (Dingenen et al., 2015a).

Subjective knee function was assessed with the IKDC Subjective Knee Form (Irrgang et al., 2001) and KOOS questionnaire (Roos & Lohmander, 2003; Roos, Roos, Lohmander, Ekdahl, & Beynnon, 1998). Both the IKDC Subjective Knee Form (Haverkamp et al., 2006) and KOOS (de Groot, Favejee, Reijman, Verhaar, & Terwee, 2008) were translated and validated for a Dutch population.

2.2. Data collection

Ground reaction forces and muscle activity of 10 lower extremity muscles were measured simultaneously and synchronously during the transition from DLS to SLS. Ground reaction forces were measured by a force plate (Bertec Corporation®) at 500 Hz using a Micro 1401 data-acquisition system and Spike2 software (Cambridge Electronic Design, UK) and low pass filtered with a cut-off frequency of 5 Hz. Surface electromyography (EMG) (Noraxon Myosystem 1400®) signals were measured at 2000 Hz using MyoResearch 2.0 (Noraxon USA, Inc., Scottsdale, AZ) and Spike2 software. The gluteus maximus, gluteus medius, tensor fascia latae, vastus lateralis, vastus medialis obliquus, hamstrings medial, hamstrings lateral, tibialis anterior, peroneus longus and gastrocnemius were measured unilaterally on the upcoming SLS leg. Placement of the electrodes was based on the instructions of Basmajian and De Luca (1985). One reference electrode was put on the anteromedial side of the tibia. The silver–silver chloride, pre-gelled bipolar surface EMG electrodes (Medicotest Inc., Rolling Meadows, IL) were placed over the muscle belly and aligned with the longitudinal axis of the muscle, with a center-to-center distance of 10 mm. The minimum distance between electrode pairs was set at 3 cm to reduce the possibility of cross-talk between neighboring muscles. The skin area where electrodes were applied was shaved and gently cleaned with 70% isopropyl alcohol to reduce the impedance. The EMG signals were stored on a PC (Dell, Round Rock, Texas, USA) for further analysis. The position of the electrodes was confirmed by isolated manual muscle tests.

2.3. Procedure

The procedure used in this study is based on previous studies (Dingenen et al., 2013, 2015, 2015a, 2015b, 2015c). Subjects were asked to stand barefoot on a force plate with the feet separated by the width of the hips and the arms hanging loosely at the side. They performed a transition task from DLS (13s) to SLS (13s). Both legs of both groups were tested. The leg that was tested first was assigned randomly. The position of the feet during DLS was indicated on a paper lying on the force plate to ensure that subjects returned to the same starting position after each trial. Subjects were instructed to lift one leg on the command of the examiner towards approximately 60° of hip flexion within 1 s, using a metronome as a reference. The transition task from DLS to SLS was tested with eyes open and with eyes closed. Both conditions were performed 3 times in an alternating order. In the eyes open tests, subjects were instructed to keep their gaze straight ahead facing a white wall. No other visual or auditory cues were present during the tests. All subjects were allowed to familiarize with the test conditions and movement speed by performing 2 practice trials of each condition before the actual measurements. For all subjects, an equal number of fake trials (shifting the weight to the non-tested leg) were randomly included to avoid preparedness. Between conditions, subjects could rest to avoid fatigue. Afterwards, body height and weight were measured.

2.4. Data analysis

Force plate and EMG signals of each trial of each condition were exported from Spike2 into LabVIEW (National Instruments Corp., Austin, Tex). The raw EMG data were first rectified and then filtered by 6th order Butterworth low-pass filter with a cut-off frequency of 45 Hz to generate the envelope of the EMG. A fixed window of 100 ms before the stance transition (during the DLS phase) was compared with a moving window of the same length along the measurement (Dingenen et al., 2015b, 2015c). An increase of more than 2 standard deviations on top of the average baseline activity was identified as the onset of muscle activity in reaction to the transition (Hodges & Bui, 1996). The onset of muscle activity determined by the algorithm was checked against the muscle activity onset identified visually (Hodges & Bui, 1996).

When moving from DLS to SLS, the center of pressure first moves towards the opposite direction of the final standing leg with a certain maximum deviation (contralateral push-off movement) (Dingenen et al., 2013). This is followed by a movement towards the single standing leg to finally finish on one leg (SLS phase) (Dingenen et al., 2013). Onset of muscle activity was calculated with respect to the mediolateral moment of force (Mx) (Dingenen et al., 2015b, 2015c). First, the average Mx was calculated for the DLS phase. The time point at which the Mx crossed that average after the contralateral push-off movement during the weight transfer to the single standing leg was defined as the Mx onset. An increase of muscle activity before Mx onset is reflected by a negative value for the onset of muscle activity. A positive value indicates an increase of muscle activity after Mx onset (Dingenen et al., 2015b, 2015c).

2.5. Statistical analysis

2.5.1. Subject characteristics

All subject characteristics showed a normal distribution (Shapiro–Wilk), except for age. Comparisons between groups for height, weight and body mass index were done with independent *t*-tests, for age with the Mann–Whitney *U* test and for gender with the chi-squared test (Dingenen et al., 2015a).

2.5.2. Muscle activation onset times

The average of 3 trials of all muscle activation onset times was calculated for each condition of each subject.

First, the injured leg of the ACLI group (dominant/non-dominant) was matched with the same dominant/non-dominant leg of the control group (Dingenen et al., 2015a; Negahban, Ahmadi, Salehi, Mehravar, & Goharpey, 2013). Hereby, the 15 injured legs of the ACLI group were compared with 4 randomly chosen dominant legs and 11 randomly chosen non-dominant legs of the control group, because 4 subjects of the ACLI group had an ACL injury on the dominant leg, and 11 subjects on the non-dominant leg. After matching, all muscle activation onset times were normally distributed for both groups and both vision conditions (Shapiro–Wilk). Differences between groups were analyzed with independent *t*-tests for eyes open and eyes closed separately.

Second, differences between legs within both groups (dominant versus non-dominant leg for the control group, and injured versus non-injured leg for the ACLI group) were tested with dependent *t*-tests for eyes open and eyes closed separately. For these analyses within groups, no matching for leg dominance between groups was required. All muscle activation onset times were normally distributed for both legs, both vision conditions and both groups, except vastus lateralis in the eyes open condition in the control group (Shapiro–Wilk). Vastus lateralis muscle activation onset times were compared between legs in the eyes open condition in the control group with the Wilcoxon Signed Rank test.

Statistical significance was set at $P < .05$ for all analyses. Statistical analyses were performed using IBM SPSS Statistics for Windows, Version 19.0, Armonk, NY: IBM Corp., USA.

3. Results

3.1. Subject characteristics

No significant differences between groups were found for age, gender, height, weight and body mass index ($P > .05$) (Table 1) (Dingenen et al., 2015a).

Table 1
Subject characteristics.

	Control group	ACLI group	*P*-value
Subjects (*n*)	15	15	
Age (M ± SD)	24.4 ± 2.1	24.7 ± 5.7	.361
Gender			.456
Men (*n*)	5	7	
Women (*n*)	10	8	
Body height (cm) (M ± SD)	172.2 ± 9.9	173.4 ± 8.4	.728
Body weight (kg) (M ± SD)	67.0 ± 10.3	69.3 ± 6.2	.461
Body mass index (kg/m^2) (M ± SD)	22.5 ± 2.0	23.1 ± 1.7	.393

ACLI: anterior cruciate ligament injured; *n*: number of subjects; M: mean; SD: standard deviation.

The IKDC Subjective Knee Form score for the ACLI group was 45.3 ± 15.0. The KOOS scores were 65.8 ± 19.0 for pain, 59.3 ± 19.6 for symptoms, 74.9 ± 18.5 for activities of daily living, 27.0 ± 28.2 for sport and recreation function, and 37.5 ± 17.0 for quality of life (Dingenen et al., 2015a).

3.2. Muscle activation onset times

3.2.1. Differences between groups

In the eyes open condition, significantly delayed muscle activation onset times of gluteus maximus ($P < .001$), gluteus medius ($P = .015$), vastus lateralis ($P < .001$), vastus medialis obliquus ($P = .001$), hamstrings medial ($P = .001$), hamstrings lateral ($P < .001$), tibialis anterior ($P < .001$), peroneus longus ($P < .001$) and gastrocnemius ($P = .003$) were found in the ACLI group compared to the control group (Fig. 1A).

In the eyes closed condition, significantly delayed muscle activation onset times of tensor fascia latae ($P = .027$), vastus lateralis ($P = .001$), vastus medialis obliquus ($P = .004$), hamstrings medial ($P = .005$), hamstrings lateral ($P = .004$), tibialis anterior ($P < .001$), peroneus longus ($P < .001$) and gastrocnemius ($P = .026$) were found in the ACLI group compared to the control group (Fig. 1B).

3.2.2. Differences between legs within each group

In the control group, a significantly delayed muscle activation onset time of gluteus maximus was found in the non-dominant leg compared to the dominant leg in the eyes closed condition ($P = .001$). All other muscles were not significantly different between legs for both the eyes open (Fig. 2A) and eyes closed condition (Fig. 2B) ($P > .05$).

In the ACLI group, significantly delayed muscle activation onset times of tibialis anterior in the eyes open condition ($P = .004$) and gastrocnemius in the eyes closed condition ($P = .007$) were found in the injured leg compared to the non-injured leg. All other muscles were not significantly different between legs for both the eyes open (Fig. 3A) and eyes closed condition (Fig. 3B) ($P > .05$).

4. Discussion

The majority of previous studies assessing muscle activation patterns after ACL injury mainly focused on muscles surrounding the injured knee joint (Aalbersberg et al., 2009; Beard et al., 1993; Boerboom et al., 2001; Branch et al., 1989; Bryant et al., 2009; Ciccotti et al., 1994; Gauffin & Tropp, 1992; Houck et al., 2007; Kalund et al., 1990; Klyne et al., 2012; Lindstrom et al., 2009; Sinkjaer & Arendt-Nielsen, 1991; Swanik et al., 1999, 2004; Wojtys & Huston, 1994). The current study is the first to evaluate muscle activation onset times of the knee, hip and ankle after ACL injury during a standardized functional task, the transition from DLS to SLS.

The results of our study show that control subjects initiated muscle activity before the time point where the upcoming supporting leg is suggested to be loaded (Mx onset) (Dingenen et al., 2015b, 2015c) for all muscles except gluteus maximus in the eyes closed condition. This anticipatory muscle activity prepares the lower extremity for the upcoming postural perturbation created by the voluntary movement (shifting the weight from DLS to SLS) and is believed to be beneficial to develop efficient joint stabilization strategies (Aruin & Latash, 1995; Bouisset & Do, 2008; Hodges & Richardson, 1997; Wikstrom et al., 2006; Williams et al., 2001). In contrast, the ACLI group initiated muscle activity typically after the Mx onset, except for tensor fascia latae in both vision conditions and gluteus medius in the eyes open condition. Significant delayed muscle activation onset times between groups were not only found around the knee joint, but also around the hip and ankle. These results are in line with our first hypothesis and with the observations that neuromuscular deficits are not only present around the injured joint in other lower extremity pathologies (Barton, Lack, Malliaras, & Morrissey, 2013; Van Deun et al., 2007). This may decrease the ability to protect the lower extremity joints from the postural perturbation created by voluntary movements (Bouisset & Do, 2008; Crow, Pizzari, & Buttifant, 2011; Hodges & Richardson, 1997). Comparing our results with previous literature where muscle activation onset times were measured in ACLI subjects is difficult because of the variety of experimental tasks, methodologies and time since injury (Beard et al., 1993; Kalund et al., 1990; Sinkjaer & Arendt-Nielsen, 1991; Wojtys & Huston, 1994). Nevertheless, the delayed muscle activation onset times of the knee muscles in the acute phase after ACL injury are in line with the findings of Wojtys and Huston (1994). Other studies measuring muscle activation onset times after ACL injury mainly included chronic ACLI subjects (>6 months after injury) and reported mixed results (Beard et al., 1993; Bryant et al., 2009; Kalund et al., 1990; Lindstrom et al., 2009; Sinkjaer & Arendt-Nielsen, 1991). A previous study using the same experimental task in subjects with chronic ankle instability also reported delayed muscle activation onset times not only around the injured joint, but the absolute values of the delays cannot be compared, as the speed of movement was not standardized and the Mx onset was defined differently in their study (Van Deun et al., 2007).

Apart from the comparison between groups, we also compared both legs within each group. Interestingly, no significant different muscle activation onset times were found between the injured and non-injured leg of the ACLI group, except for tibialis anterior in eyes open and gastrocnemius in eyes closed condition. These differences for tibialis anterior and gastrocnemius between legs were considered less consistent and less clinically relevant compared to the between group differences, as significant differences were only found for one vision condition, and the magnitude of the absolute differences in muscle activation onset times were rather small. The significant slower muscle activation onset time of gluteus maximus in the

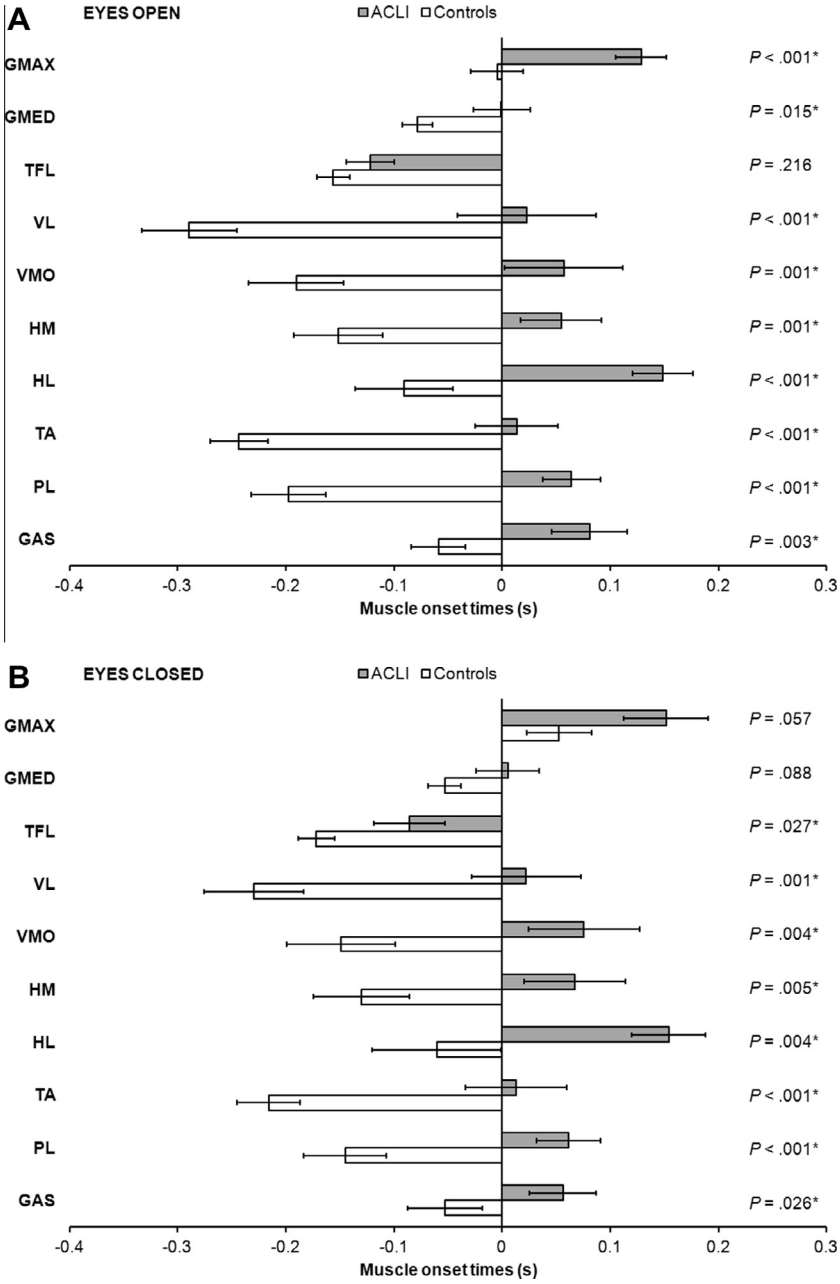

Fig. 1. Means and standard errors of mean of the onsets of muscle activity of gluteus maximus (GMAX), gluteus medius (GMED), tensor fascia latae (TFL), vastus lateralis (VL), vastus medialis obliquus (VMO), hamstrings medial (HM), hamstrings lateral (HL), tibialis anterior (TA), peroneus longus (PL) and gastrocnemius (GAS) of the injured leg of the anterior cruciate ligament injured (ACLI) group and the matched leg of the control group with eyes open (A) and eyes closed (B). *Significant at $P < .05$. Negative and positive values on the *x*-axis represent an onset of muscle activity respectively before and after the time point at which the mediolateral moment of force crossed the average mediolateral moment of force of the double-leg stance phase after the contralateral push-off movement.

non-dominant leg compared to the dominant leg in the eyes closed condition in the control group may have contributed to the non-significant difference (marginally significance) of the between group comparison for gluteus maximus during the eyes closed condition, as 11 of the 15 matched legs of the control group were non-dominant legs. A proper function of the gluteus maximus is essential to control lower extremity and pelvic postures and movements when transitioning towards single-leg weight-bearing activities (Ford et al., 2015; Willson et al., 2006; Zazulak et al., 2005). In conclusion, our results confirm that caution is warranted when using the non-injured leg as a normal reference when evaluating muscle activation patterns after a peripheral joint injury (Hewett, Paterno, & Myer, 2002; Urbach et al., 1999).

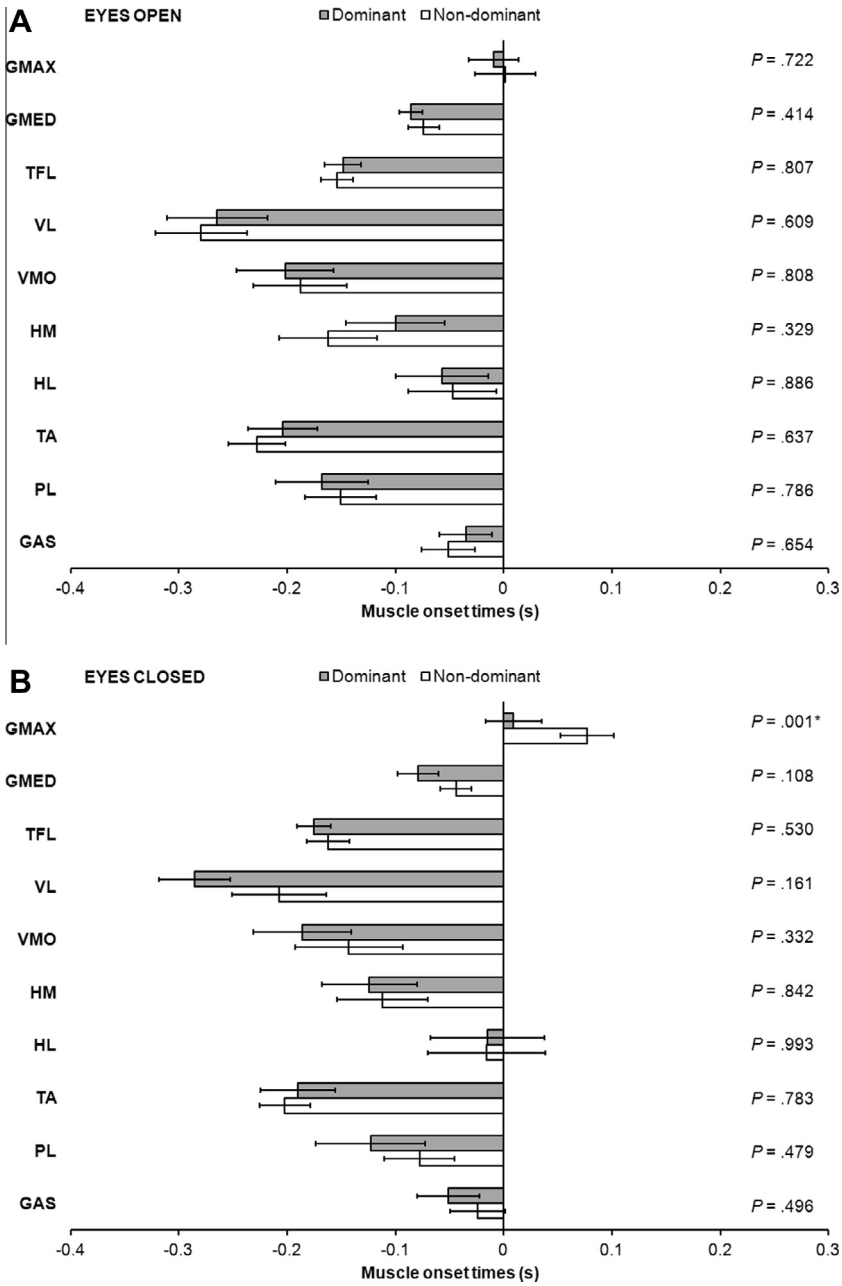

Fig. 2. Means and standard errors of mean of the onsets of muscle activity of gluteus maximus (GMAX), gluteus medius (GMED), tensor fascia latae (TFL), vastus lateralis (VL), vastus medialis obliquus (VMO), hamstrings medial (HM), hamstrings lateral (HL), tibialis anterior (TA), peroneus longus (PL) and gastrocnemius (GAS) of the dominant and non-dominant leg of the control group with eyes open (A) and eyes closed (B). *Significant at $P < .05$. Negative and positive values on the *x*-axis represent an onset of muscle activity respectively before and after the time point at which the mediolateral moment of force crossed the average mediolateral moment of force of the double-leg stance phase after the contralateral push-off movement.

The mechanisms behind the less optimal, reactive neuromuscular control strategies adopted by the ACLI subjects are only poorly understood. The speed of movement may be crucial. Subjects with pathology have the tendency to perform slower during voluntary motor control tasks compared to non-injured control subjects (Dingenen et al., 2013). This may be important when analyzing muscle activation onset times, as earlier muscle activation onset times have been found when transitioning faster (Rogers, 1992; Rogers & Pai, 1990). However, in a previous study of our group where the center of pressure characteristics were analyzed during the same transition from DLS to SLS in the same ACLI and control subjects, we showed that the duration of the contralateral push-off movement and the peak center of pressure velocity when transitioning to the upcoming SLS leg were not significantly different between the ACLI and control group, probably as a

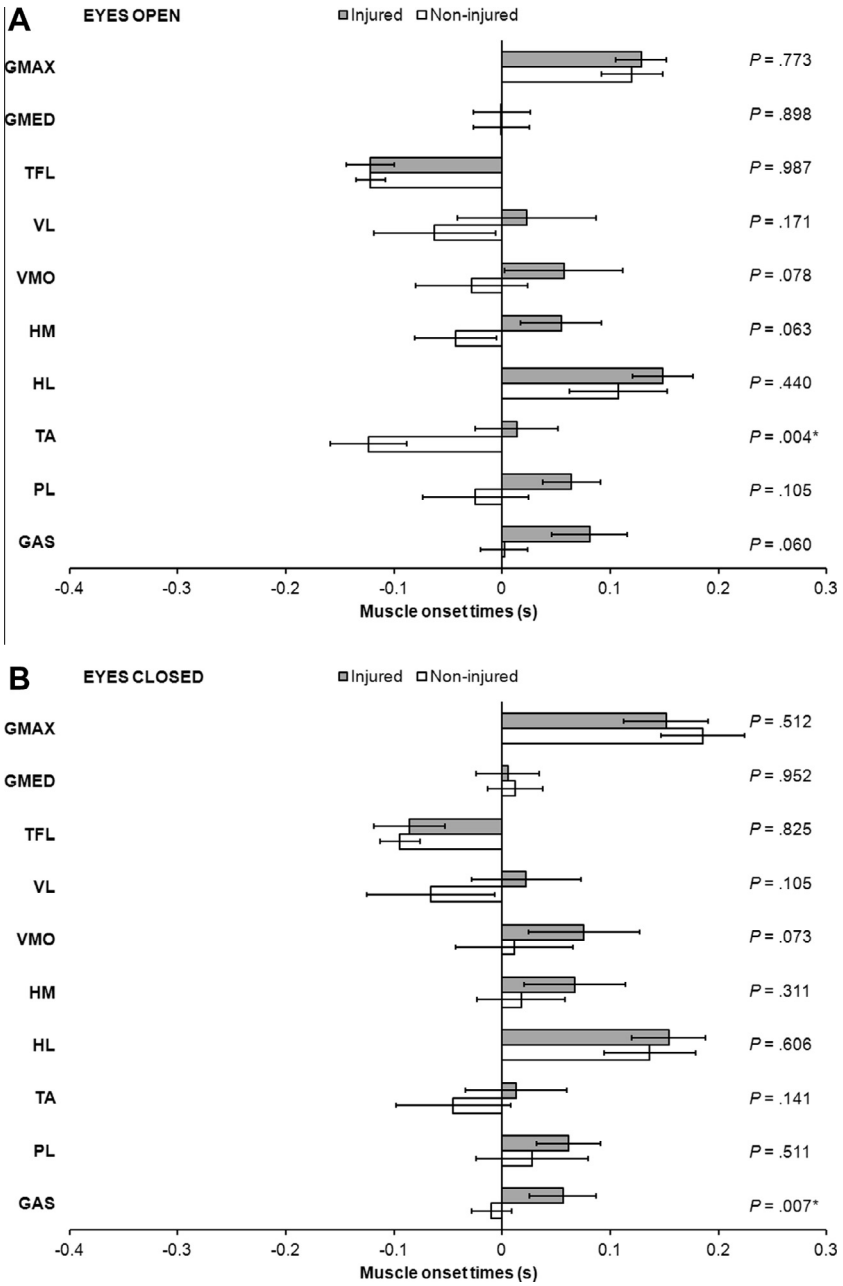

Fig. 3. Means and standard errors of mean of the onsets of muscle activity of gluteus maximus (GMAX), gluteus medius (GMED), tensor fascia latae (TFL), vastus lateralis (VL), vastus medialis obliquus (VMO), hamstrings medial (HM), hamstrings lateral (HL), tibialis anterior (TA), peroneus longus (PL) and gastrocnemius (GAS) of the injured and non-injured leg of the anterior cruciate ligament injured (ACLI) group with eyes open (A) and eyes closed (B). *Significant at $P < .05$. Negative and positive values on the *x*-axis represent an onset of muscle activity respectively before and after the time point at which the mediolateral moment of force crossed the average mediolateral moment of force of the double-leg stance phase after the contralateral push-off movement.

result of the standardization of movement speed (Dingenen et al., 2015a). Therefore, differences in movement speed between groups are probably not the reason for the delayed muscle activation onset times in the current study.

More valuable explanations for the slower muscle activation patterns may originate from the neurophysiological alterations after ACL injury. Mechanoreceptors on the surface of the ACL are involved in providing the CNS with proprioceptive information by converting mechanical signals into specific neural signals (Kennedy, Alexander, & Hayes, 1982; Schultz, Miller, Kerr, & Micheli, 1984; Schutte, Dabezies, Zimny, & Happel, 1987). This afferent neural input may be altered after ACL injury through a variety of mechanisms, including the presence of joint effusion, excessive movement, pain, as well

as the loss of mechanoreceptors after disruption of the ACL itself (Palmieri-Smith & Thomas, 2009). A diminished joint position sense and increased threshold to detection of passive motion have been demonstrated in ACLI subjects compared to non-injured subjects (Gokeler et al., 2012). As the acquisition of proprioceptive information is considered to be essential for neuromuscular control mechanisms (Riemann & Lephart, 2002a), the delayed muscle activation onset times in this study may therefore be attributed to the altered proprioceptive information after ACL injury. However, the validity of this theory can be questioned, as it cannot fully explain the altered muscle activation patterns in the other lower extremity joints, and the lack of consistent differences between the injured and non-injured leg of the ACLI group. Furthermore, proprioceptive deficits have only a low-to-moderate correlation with function after ACL injury (Gokeler et al., 2012). In this perspective, it is important to know that the acquisition of peripheral proprioceptive information reflects only one aspect of a long and complex sensorimotor pathway. During recent years, evidence is emerging that a decreased ability of the CNS to reorganize the integration and interpretation of the altered proprioceptive afferent information and plan the motor commands may be the underlying mechanism of altered neuromuscular control patterns after ACL injury (Baumeister et al., 2008, 2011; Grooms et al., 2015; Kapreli et al., 2009; Needle et al., 2014; Ward et al., 2015). Using functional magnetic resonance imaging of the brain while performing flexion and extension of the knee while lying supine, an increased activation of specific brain areas including the pre-supplementary motor area and the posterior inferior temporal gyrus was found in ACLI subjects compared to matched controls (Kapreli et al., 2009). These findings might imply that ACLI subjects have an increased need for more conscious movement planning, visualization and feedback, even when performing a simple movement. Using electroencephalography, an increased frontal Theta-power and decreased Alpha-2 parietal power during a joint position sense task (Baumeister et al., 2008), and increased frontal Theta-power during a force matching task (Baumeister et al., 2011) were demonstrated in ACL reconstructed subjects compared to controls. These alterations may reflect a higher attentional demand and differences in sensory information processing in the somatosensory cortex during cognitive and sensorimotor tasks, hereby highlighting the major role of the working memory (Baumeister et al., 2011). In summary, these studies support the concept that neuroplastic changes occur within the CNS after ACL injury and reflect a higher neurocognitive involvement and increased neural load to perform movements after ACL injury (Baumeister et al., 2011; Grooms et al., 2015).

In the current study, subjects had to lift one leg on the command of the examiner at a certain point in time. They were unaware when the examiner would give the command to lift a leg, or which leg the examiner would command to lift, creating a certain level of task uncertainty. Furthermore, a standardized speed of movement was used. These task constraints may have led to a neurocognitive overload during the transition from DLS to SLS for the ACLI subjects, hereby exceeding the capability of their compensatory attentional and motor planning resources, resulting in delayed muscle activation onset times (Grooms et al., 2015; Needle et al., 2014; Ward et al., 2015). The fact that differences between groups were also apparent during the eyes open condition, and not only during the eyes closed condition, does not support the compensatory increased need for visualization (Kapreli et al., 2009) which was previously demonstrated in ACLI subjects when evaluating center of pressure outcomes during the SLS phase of the transition task from DLS to SLS (Dingenen et al., 2015a). We hypothesize that the previously mentioned task constraints were already difficult enough to show differences in muscle activation onset times between groups during this particular transition phase of the current weight-shifting task, and surpassed the influence of the visual occlusion.

Alternatively, the reported slower muscle activation onset times may represent ongoing deficits that were already present before the ACL injury occurred, rather than changes that occurred after the ACL injury (Saunders, McLean, Fox, & Otago, 2014). A decreased performance on a set of neurocognitive tests was prospectively related with increased non-contact ACL injury risk (Swanik, Covassin, Stearne, & Schatz, 2007). These neurocognitive deficits may have contributed to the delayed muscle activation onset times, hereby limiting the capacity of the active restraints to effectively protect the ligamentous structures and increasing ACL injury risk (Needle et al., 2014; Swanik et al., 2007). Based on the retrospective design of this study, it remains unclear whether the delayed muscle activation onset times are the cause or the result of the ACL injury, or a combination of both. Prospective studies should clarify this in the future.

ACL reconstruction continues to be the most cost-effective standard procedure for the majority of ACLI subjects (Marx, Jones, Angel, Wickiewicz, & Warren, 2003; Mather et al., 2013). Rehabilitation after ACL injuries preferably starts shortly after the injury as patients who are undergoing pre-operative rehabilitation programs may progress more easily through the important early postoperative phases (Wilk, Macrina, Cain, Dugas, & Andrews, 2012). Clinicians involved in this rehabilitation should be aware of the neuromuscular deficits and their underlying mechanisms to efficiently target interventions. The results of the current study indirectly support the link between CNS reorganization and altered muscle activation patterns after ACL injury. Therefore, it can be suggested that rehabilitation should focus on the cause of the altered muscle activation patterns, e.g., CNS reorganization with targeted CNS re-education training, rather than exclusively focusing on the final clinical impairments of the sensorimotor system, e.g., peripheral muscle function (Baumeister et al., 2011; Kapreli et al., 2009; Pietrosimone et al., 2015). Indeed, traditional resistance training alone is not sufficient to successfully refine more anticipatory neuromuscular strategies (Crow et al., 2011; Pietrosimone et al., 2012).

In the acute phase after injury, disinhibitory interventions such as transcutaneous electrical nerve stimulation or cryotherapy can be used with the purpose to address the altered afferent information coming from the injured knee joint that contributes to neuromuscular inhibition following injury (Harkey, Gribble, & Pietrosimone, 2014; Kapreli et al., 2009; Pietrosimone et al., 2015). Restoring upstream neural function may have significant effects on downstream neuromuscular control (Pietrosimone et al., 2012). In addition, highly repetitive voluntary motor control training is one of the possible strategies to accelerate muscle activation onset times (Crow et al., 2011). However, this type of motor control exercises often

includes very conscious and pre-planned training of the muscles surrounding the injured joint with an internal focus of attention, which may hamper the transfer to automatic and less predictable functional real-life conditions due to an increased cortical attention and neurocognitive overloading, hereby reinforcing the previously mentioned maladaptive reorganization of the CNS (Baumeister et al., 2008; Benjaminse et al., 2015; Grooms et al., 2015). Therefore, it may be worthwhile to consider practicing tasks in conditions of uncertainty, whereby the time to plan movements is progressively reduced (Kapreli et al., 2009). Using an external focus of attention (Benjaminse et al., 2015; Grooms et al., 2015; Pietrosimone et al., 2015; Snodgrass et al., 2014), and involving the entire lower extremity of both legs within functional, varying and progressively challenging contexts as soon as possible after ACL injury require the patient to actively solve motion solutions which may help to stimulate adaptive CNS motor learning processes and develop automatic anticipatory multi-joint neuromuscular engrams (Ageberg, 2002; Chmielewski, Hurd, Rudolph, Axe, & Snyder-Mackler, 2005; Chmielewski et al., 2002; Fitzgerald, Axe, & Snyder-Mackler, 2000; Hewett et al., 2002; Kapreli et al., 2009; Nyland, Brosky, Currier, Nitz, & Caborn, 1994; Snodgrass et al., 2014). The differences between groups for both vision conditions may implicate that both eyes open and eyes closed conditions can be used to refine muscle activation patterns in clinical care. Based on a thorough understanding of the underlying mechanisms of the alterations in neuromuscular control after ACL injury, future studies should further specify these therapeutic concepts to describe the most optimal rehabilitation strategies.

We acknowledge that this study has some limitations. Despite the significant differences between groups, it is important to know that inter-individual differences can exist. A variety of muscle activation patterns can be used during the execution of voluntary motor control tasks. Care should be taken when generalizing the results of the current study to a specific individual. Based on the rather small sample size, we did not make a distinction between potential copers and non-copers. Future studies may relate individual muscle activation patterns to functional status after injury. No structured rehabilitation was performed by the ACLI subjects. Future studies should also evaluate the influence of rehabilitation on these muscle activation onset times. The findings of this study support the discriminative value of a weight-shifting task to assess lower extremity neuromuscular control. However, it remains unknown whether muscle activation onset times during one specific task, the transition from DLS to SLS, can be generalized to other more dynamic tasks. Current literature suggests that this extrapolation may be too simplified (Duysens & Levin, 2010). In addition, we did not include any measurements for possible static or dynamic malalignments of the lower extremity, pelvis, or trunk. As we measured each subject only once, it remains unclear if these muscle activation patterns change over time. The eyes open and eyes closed conditions were not compared, because the transition is generally performed slower during eyes closed conditions despite the efforts to standardize movement speed (Dingenen et al., 2015a). Furthermore, this comparison was not the main objective of this study. We are aware that the proposed CNS mechanisms are indirectly deduced from the current results. Future studies should evaluate the relation between neuromuscular control and CNS reorganization more directly (Ward et al., 2015).

5. Conclusions

The results of this study showed significantly delayed muscle activation onset times not only around the knee, but also at the hip and ankle muscles in acute ACLI subjects compared to a non-injured control group during the transition from DLS to SLS during eyes open and eyes closed conditions. No significant differences between the injured and non-injured leg of the ACLI group were found, except for tibialis anterior and gastrocnemius. These findings indirectly support the link between CNS reorganization and altered muscle activation patterns after ACL injury. Clinicians should focus on the underlying mechanisms of the altered muscle activation patterns during rehabilitation with targeted CNS re-education training, rather than exclusively focusing on the improvement of peripheral muscle function.

Funding

None.

Acknowledgment

None.

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