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TITLE

Early compensatory and anticipatory postural adjustments following anterior cruciate ligament reconstruction

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ABSTRACT

Purpose. Early identification of postoperative neuromuscular deficits has been advocated to prevent muscle weakness and maximise functional outcomes following anterior cruciate ligament reconstruction (ACLR). The purpose of this study was to investigate neuro-mechanical changes in compensatory and anticipatory postural adjustments, which play a major role in minimizing unpredictable and predictable disturbances, respectively, as early as 2 months after ACLR.

Methods. Nine young male individuals who underwent ACLR with patellar tendon and 9 age-matched healthy controls were exposed to 2 blocks of 10 either unexpected or expected loading perturbations of the knee joint while semi-reclined on a raised plinth. Amplitude and latency of postural responses in the vastus lateralis (VL), rectus femoris (RF) and biceps femoris (BF) muscles were determined by surface electromyography.

Results. Latency of compensatory responses was higher in patients with ACLR than in healthy participants for VL (82±15 ms *vs* 68±10 ms, $P<0.05$) and RF (81±21 ms *vs* 63±10 ms, $P<0.05$). Amplitude of compensatory responses was 54% lower in patients with ACLR than in healthy participants for VL ($P<0.05$). Onset of anticipatory responses occurred earlier in patients with ACLR than in healthy participants for VL (-83±45 ms *vs* -26±21 ms, $P<0.05$), RF (-59±48 ms *vs* -10±13 ms, $P<0.05$) and BF (-72±42 ms *vs* -12±14 ms, $P<0.01$).

Conclusion. Patients with ACLR showed early abnormalities in compensatory and anticipatory postural adjustments, which may reflect the inability to quickly detect sudden changes in muscle length or to completely activate muscles surrounding the knee, and may be addressed by specific training interventions.

KEYWORDS

Knee surgery, postural control, feed-forward strategies, stretch reflex

ABBREVIATIONS

ACL	Anterior cruciate ligament
ACLR	Anterior cruciate ligament reconstruction
ANOVA	Analysis of variance
APAs	Anticipatory postural adjustments
BF	Biceps femoris
CNS	Central nervous system
CPAs	Compensatory postural adjustments
RF	Rectus femoris
RMS	Root mean square
EMG	Electromyography
VL	Vastus lateralis

INTRODUCTION

Any motor act combines the displacement of one or more segments toward a given goal and the postural stabilization of other segments (Dufossè et al. 1985; Massion 1992; Bennis et al. 1996; Baldissera et al. 2008; Bolzoni et al. 2012). Postural stability is achieved during voluntary movement by compensatory and anticipatory strategies aimed at minimizing unpredictable and predictable disturbances, respectively (Bennis et al. 1996; Kanekar and Aruin 2014). Since the work by Hugon et al. (1982), a number of authors have investigated compensatory and anticipatory strategies using bimanual loading/unloading tasks of the upper limb (Dufossè et al. 1985; Bennis et al. 1996; Massion et al. 1999). When a loading/unloading perturbation to the forearm is imposed

unexpectedly by the experimenter, compensatory postural adjustments (CPAs) are observed at the level of the forearm muscles following the perturbation. CPAs reflect a feedback control wherein changes in muscle activity are triggered by sensory signals in response to unpredictable postural perturbations (Park et al. 2004; Alexandrov et al. 2005). In contrast, when the loading/unloading perturbation to the forearm is voluntarily performed by the subject's contralateral hand, anticipatory postural adjustments (APAs) of the forearm muscles precede the onset of postural perturbation. APAs reflect a feed-forward control wherein changes are seen in the background activity of muscles prior to an expected external perturbation or a forthcoming self-initiated movement (Bouisset and Zattara 1987; Massion 1992; Aruin and Latash 1995; Aruin et al. 1997).

The ability to minimize postural disturbances plays a major role in preventing injuries of the lower limb during sports activities. Among the most common sport-related injuries is the tear of the anterior cruciate ligament (ACL) of the knee joint (Griffin et al. 2000), which occurs when a sudden postural perturbation appears during voluntary movement of the lower limb (Hewett et al. 2006). Appropriate surgical reconstruction of the ligament has been shown to restore mechanical stability of the knee joint (Cascio et al. 2004; Van Grinsven et al. 2010), although neuromuscular control of the lower limb may be undermined for years after surgery (Konishi et al. 2002). Persistent weakness of the muscles acting on the knee joint has been shown to arise from a reduced rate of voluntary activation (Elmqvist et al. 1989; Lorentzon et al. 1989; Snyder-Mackler et al. 1995; Urbach et al. 2001), perhaps via impaired gamma drive (Konishi et al. 2002). In turn, this might lead to functional instability during daily living tasks (Bulgheroni et al. 1997; Ferber et al. 2003; Lustosa et al. 2011) and, hence, increased risk of re-injury (Chappell et al. 2007; Hewett et al. 2013).

Early identification and quantification of postoperative neuromuscular deficits have been advocated to prevent the loss of muscle strength and maximize functional outcomes in patients with ACL reconstruction (ACLR; Myer et al. 2006; Adams et al. 2012; Laudani et al. 2014). To the best of the authors' knowledge, however, abnormalities in CPAs and APAs have been investigated by previous authors during complex voluntary movements, which are not safe when carried out under

challenging postural conditions earlier than 3 months from surgery (Ferber et al. 2003; Lustosa et al. 2011; Bryant et al. 2009). Noteworthy, the results of these studies have pointed out that late postoperative abnormalities in postural responses are associated to the patient's functional level (Lustosa et al. 2011) and the time from ACLR (Gokeler et al. 2010; Bryant et al. 2009), thus suggesting that abnormalities in both CPAs and APAs might be reversed by appropriate early identification and intervention.

In light of the crucial role played by active postural stabilization of the lower limb segments during voluntary movement in patients with ACLR, early identification and quantification of abnormal CPAs and APAs are paramount to design appropriate rehabilitation protocols and maximize the patient's outcome following surgery. In the present study, we dealt with this issue by designing a joint perturbation task involving unpredictable and predictable postural disturbances to the lower limb for the evaluation of CPAs and APAs, respectively, as early as 2 months from ACLR. The task was derived from the loading/unloading bimanual paradigm (Massion et al. 1999), which allowed to focus on postural adjustments of the knee extensor and flexor muscles exclusively, thus ruling out confounding factors arising from postural control of muscles acting on joints other than the knee. Based on the findings of previous studies, which suggest a strong association between abnormalities in postural adjustments during voluntary movements of daily life and the time from ACLR (Ferber et al. 2003; Lustosa et al. 2011; Vairo et al. 2008; Bryant et al. 2009; Gokeler et al. 2010), it was hypothesized that patients with ACLR would have shown delayed compensatory responses and earlier anticipatory responses compared to healthy participants.

MATERIALS AND METHODS

Participants

Nine male patients (age: 24 ± 6 years; body mass: 76.3 ± 5.2 kg; stature: 1.78 ± 0.04 m) with unilateral isolated rupture of the ACL in the dominant limb were recruited to participate in the study. The dominant limb was determined by asking the participants which leg they use to kick a

ball (Elias and Bryden 1998; Macaluso et al. 2003). Physical activity levels before injury were evaluated in all patients by means of the Tegner Activity Score (Tegner and Lysholm 1985) and only patients with activity level between 3 and 5 were included in the study, while sedentary subjects and competitive sport athletes were excluded. Arthroscopic ACLR with ipsilateral autologous bone–patellar tendon–bone graft was performed by only one surgeon from 10 to 30 days after ACL injury. Inclusion criteria were full range of motion at the knee joint and lack of pain, locking and swelling of the knee joint as evaluated by the Lysholm Knee Scoring Scale (Tegner and Lysholm 1985). Exclusion criteria were concomitant injury to any other knee ligament or lower limb muscle, associated meniscus tear, and previous surgery on either knee. All patients were asked to undertake a standardized rehabilitation protocol under supervision of physical therapists 5 days per week, as described by Laudani et al. (2014). Briefly, they were asked to wear a brace immediately after surgery and to bear weight on the second day. During the first 2 weeks, the rehabilitation program consisted of continuous passive mobilizations, together with neuromuscular electrical stimulations of the knee extensor and flexor muscles, and isometric straight leg rises, which were carried out until the end of the first month. Squatting exercises were incorporated within the first 3–4 weeks. During the second month, strengthening exercises and hydrokinesis were implemented. Exercises in water involved cycling, walking, and stepping movements. At the end of the second month and prior to experimental testing, all patients were assessed for maximal isometric strength of the knee extensor muscles in each limb on a leg extension machine (Technogym, Forli-Cesena, Italy) with a load cell connected to a computerized system unit (MuscleLab, Bosco-System Technologies, Rieti, Italy). Only patients with a limb symmetry index, which was quantified as the percentage ratio between peak force of the involved and uninvolved limb, between 75 and 100 % were allowed to participate in the study.

Nine male volunteers (age: 25 ± 3 years; body mass: 70 ± 6 kg; stature: 1.74 ± 0.04 m), with no disorder or history of knee injury, served as the control group. Only physically active individuals who were not engaged in regular training or sport practice more than 3 times a week, for more than

40–60 min each time, were included in the study. With Ethics Committee approval of the University of Rome "La Sapienza", the study was carried out in accordance with the Declaration of Helsinki, and informed consent was obtained from all participants.

Experimental procedure

The experiment was completed in an isolated room and subjects were blindfolded to eliminate visual input during testing. Participants wore loose-fitting shorts and sat comfortably above a raised plinth on a semi-reclined position, with their trunk inclined approximately of 40° from the horizontal. Prior to each perturbation trial, the involved limb, i.e., the operated knee of patients with ACLR and the dominant knee of healthy volunteers, was placed in a reference position with the knee joint in full extension and the ankle joint in neutral position (Fig. 1). Participants were instructed to fully relax the muscles of the involved limb before the perturbation. The limb was then dropped either by the experimenter, i.e., unexpected perturbation (unpredictable condition) or by participant contralateral limb, i.e., expected perturbation (predictable condition). During the unpredictable condition, the experimenter entirely supported the subject's involved limb with his palm open under the heel and, after 8–12 s, unexpectedly removed his palm. The participants were instructed to resist the perturbation and restore the full extension reference position as quickly as possible. During the predictable condition, participants placed their contralateral big toe under the heel of the involved limb, which was then entirely supported and kept in the reference position. After a verbal signal of the experimenter, participants were instructed to wait from 8 to 12 s and then quickly move away the supporting limb, while maintaining the involved limb in full extension. Each participant completed one series of 10 consecutive predictable perturbation trials and one series of 10 consecutive unpredictable perturbation trials, in a random order. Successive trials were separated by a minimum of 60 s. The participants were allowed to rest 5 min between series. For both the unpredictable and the predictable conditions, each subject was allowed a maximum of 5 practice trials for familiarization with the experimental procedures (Bennis et al. 1996). Since

neuromuscular adaptations likely include central components, which are known to affect both limbs of patients with ACLR (Arockiaraj et al. 2013), we compared their operated limb to the dominant limb of age-matched controls rather than to the contralateral limb.

Recording systems and data analysis

Angular displacement of the knee joint was recorded by an electrogoniometer (Biometrics Ltd., Gwent, UK) placed on the lateral side of the involved limb with the two arms aligning with the thigh and leg axes. Electromyography (EMG) data were collected using surface electrodes placed over the vastus lateralis (VL), rectus femoris (RF) and biceps femoris (BF) muscles of the involved limb, and the VL of the uninvolved limb. For each muscle, after appropriate skin cleaning, two electrodes were attached 0.02 m apart (center-to-center) on the skin half way between the center of the belly and the distal myotendinous junction, in accordance with SENIAM recommendations (Hermens et al. 2000). These muscles were considered to be representative of the knee extensor and flexor groups as in previous studies (Macaluso and De Vito 2003; Laudani et al. 2013; Mair et al. 2014). Electrogoniometer and EMG signals were sampled at 1 kHz by a portable device (FreeEMG, BTS, Milan, Italy). Kinematic data were low-pass filtered with a zero-lag second-order Butterworth filter with 10 Hz cutoff frequency. EMG signals were first high-pass filtered at 20 Hz to remove movement artifacts, and then full-wave rectified for further signal conditioning.

In both unpredictable and predictable conditions (Fig. 2a, b respectively), the knee joint of the involved limb flexed briskly due to the perturbation, then extended and stabilized after a few damped oscillations. Kinematic measurements were processed to obtain the following angular parameters: onset angle, which corresponded to the knee angle at reference position before the perturbation; peak angle, which corresponded to the first maximum knee flexion angle reached after the perturbation; repositioning angle, which corresponded to the angle at leg extension reached after the stabilizing damped oscillations. Each kinematic parameter was normalized for the plateau angle, which corresponded to the knee angle at the gravitational resting position, while semi-reclined on

the raised plinth. In the unpredictable condition, the time of perturbation onset was identified by the onset of knee angular displacement, which was agreed on by two of the experimenters after visual inspection of the kinematic trace. In the predictable condition, the time of perturbation onset was identified as the offset of the VL in the uninvolved limb. The peak time was then evaluated as the time interval from the onset of perturbation to the instant of peak angle.

For the unpredictable condition, the latency of compensatory muscle responses of the involved limb was evaluated with respect to the onset of leg movement. For the predictable condition, latency of anticipatory muscle responses of the involved limb was evaluated with respect to the onset of postural perturbation, which was identified as the offset of the VL in the uninvolved limb. Both onset and offset of either compensatory or anticipatory muscle responses were agreed by two of the experiments after visual inspection of the high-pass filtered (20 Hz), full-wave rectified EMG trace. Amplitude of muscle responses was computed as the integral of the filtered EMG signal (low-pass filter: second-order Butterworth filter with a cutoff frequency of 50 Hz) over the time interval defined by the latency of muscle responses and further divided by such latency. Amplitude of the VL and RF muscle responses was normalized to the integral of a central 5 s window of the EMG signal while maintaining for 10 s a full extension knee position. To account for inter-individual differences in anthropometric limb properties (i.e., mass and the length of lower leg), amplitude of each muscle response was further normalized to the gravitational torque (mLg), which was calculated as:

Gravitational torque = $m \times L \times g$

where m represents the mass of the oscillating leg–foot complex, L represents the distance between the rotation axis of knee joint to the leg–foot complex's center of mass, and g represents the gravitational acceleration. Measurements of body mass and stature were collected and used to predict m and L according to the anthropometric tables provided by Winter (2009). Such anthropometric properties of the lower leg, which rotates around the knee joint during our perturbation task, are known to affect the EMG amplitude response (Babault et al. 2003).

Normalization of the EMG signals to the gravitational torque rules out such 'influencing' factors on the between-subjects differences in the EMG amplitude by taking into account the most relevant active and passive components of the moving lower leg (mass and length).

Statistical analysis

The effect of trials sequence on both EMG and kinematic variables was evaluated by a linear mixed model during the predictable and the unpredictable experimental conditions in both healthy participants and ACLR patients (Maxwell and Delaney 2004). The results showed no significant effect of the trials sequence on any EMG or kinematic variable. As a result, mean and standard deviation of each parameter were calculated for both the unpredictable and predictable conditions of each participant, which were used for further analysis. Statistical differences in EMG- and kinematic-dependent variables were then evaluated by a two-way analysis of variance (ANOVA) with group (healthy participants and patients with ACLR) as between-subjects factor and predictability condition (unpredictable and predictable) as a within-subjects factor. When the ANOVA provided significant results, follow-up comparisons were corrected by applying the method of Benjamini and Hochberg to control the false discovery rate (FDR) (Hochberg and Benjamini 1990). The level of significance was set to $P < 0.05$ (FDR-corrected). Data analysis was performed using Matlab version R2009b (Mathworks Inc, Natick, MA) and SPSS version 20.0 (SPSS, Inc., Chicago, IL—IBM, Somers, NY, USA).

RESULTS

EMG parameters

The ANOVA showed a main effect of predictability on both the latency and amplitude of the VL ($F = 142.50$; $P < 0.001$ and $F = 21.67$; $P < 0.01$, respectively), RF ($F = 103.49$; $P < 0.001$ and $F = 8.58$; $P < 0.05$, respectively) and BF muscles ($F = 141.50$; $P < 0.001$ and $F = 19.15$; $P < 0.01$, respectively). There was a significant interaction between predictability and group for the

latency of the VL ($F = 11.33$; $P < 0.01$), RF ($F = 12.21$; $P < 0.01$) and BF muscles ($F = 12.26$; $P < 0.01$).

During the unpredictable condition, the ANOVA showed a significant effect of the group factor on the latency of compensatory responses for the VL ($F = 5.26$; $P < 0.05$) and RF muscles ($F = 5.40$; $P < 0.05$). In particular, as shown in Fig. 3, patients with ACLR showed higher latency of compensatory responses than healthy participants for the VL and the RF muscles (82 ± 15 vs 68 ± 10 ms and 81 ± 21 vs 63 ± 10 ms, respectively). A detailed distribution of the compensatory response latencies measured during each trial for each muscle in healthy participants and patients with ACLR is given in Fig. 4a, b, respectively. Visual inspection shows that the majority of response latencies recorded in healthy participants fell below either 50 or 100 ms (VL: 94,2 %; RF: 95,5 %; BF: 77,3 %), while almost all the latencies recorded in patients with ACLR fell above either 50 or 100 ms (VL: 90,9 %; RF: 90,9 %; BF: 93,9 %). As shown in Fig. 5, the amplitude of compensatory responses was 54 % lower in patients with ACLR than in healthy participants for the VL muscle only ($F = 5.03$; $P < 0.05$).

During the predictable condition, the ANOVA showed a significant effect of the group factor on the latency of anticipatory responses for the VL ($F = 8.32$; $P < 0.05$), RF ($F = 5.62$; $P < 0.05$) and BF muscles ($F = 10.56$; $P < 0.01$). In particular, as shown in Fig. 6a, patients with ACLR showed earlier onset latency of anticipatory responses than healthy participants for the VL (−83 ± 45 vs −26 ± 21 ms; $P < 0.05$), RF (−59 ± 48 vs −10 ± 13 ms; $P < 0.05$) and BF muscles (−72 ± 42 vs −12 ± 14 ms; $P < 0.01$). There were no significant differences in the amplitude of anticipatory responses between patients with ACLR and healthy participants (Fig. 6b).

Kinematic parameters

The repeated-measures ANOVA showed a main effect of the experimental condition on the peak angle normalized for the plateau angle ($F = 92.88$; $P < 0.001$) and no interaction between condition and group, thus indicating that the normalized peak angle was significantly lower during

the predictable condition with respect to the unpredictable condition in both patients with ACLR and healthy participants (Fig. 7). There was a tendency for the absolute peak angle of compensatory responses to be higher in patients with ACLR than in healthy participants (24.5 ± 6.7° and 19.2 ± 4.5°, respectively; $P = 0.075$); however, there were no significant differences between the two groups in the peak angle normalized for the plateau angle.

The peak time was significantly higher in patients with ACLR than in healthy participants during the unpredictable condition (0.24 ± 0.02 and 0.20 ± 0.03 s, respectively; $F = 8.27$; $P < 0.05$).

DISCUSSION

For the first time, to the best of the authors' knowledge, a perturbation task involving either unpredictable or predictable perturbations to the knee joint has been applied to individuals who underwent ACLR for the early investigation of both compensatory and anticipatory mechanisms underlying maintenance of the lower limb posture. The results show that such a task is effective to distinguish between patients with ACLR and healthy individuals in the magnitude and timing of the lower limb postural responses to forthcoming perturbations under either unpredictable or predictable conditions. The main findings of the present study are that patients with ACLR showed delayed onset of CPAs to unpredictable perturbations and earlier onset of APAs to predictable perturbations with respect to healthy control participants.

Compensatory postural responses to unpredictable perturbations

During the unpredictable condition of the present study, almost all the latencies of the compensatory responses recorded in the knee extensor and flexor muscles of healthy participants fell below either 50 or 100 ms (VL: 94,2 %; RF: 95,5 %; BF: 77,3 %) and may then be considered short- and long-latency reflexes (Matthews 1991; Casabona et al. 2012). Conversely, almost all the latencies of the knee extensor and flexor muscles recorded in patients with ACLR fell above either 50 or 100 ms (VL: 90,9 %; RF: 90,9 %; BF: 93,9 %) and may then be considered long-latency

reflexes or voluntary responses (Pruszynski and Scott 2012; Valle et al. 2013). Such a difference between patients with ACLR and healthy participants might be due to surgery-related neural adaptations of either peripheral or central origin, or to a combination of both. An efficient reaction to unexpected postural perturbations would depend on a feedback control system where an error signal, which is carried by sensory afferences, is processed to restore a given set point (Marsden et al. 1983; Matthews 1991; Pruszynski and Scott 2012; Petersen et al. 1998). Muscle spindles and joint receptors should be the main sources of sensory signals informing the brain on the onset of leg fall. The spinal cord would then use this information to elaborate very fast motor responses, such as the monosynaptic stretch reflex, exhibiting latency of muscle activation below 50 ms. Later, the information signal is thought to reach cortical or subcortical structures, which produce long-latency reflexes that are non-voluntary feedback reactions with latencies between 50 and 100 ms. Above 100 ms, long-latency reflexes may be superimposed by voluntary muscle activity. Peripheral adaptations in patients with ACLR might arise from surgery-related changes in sensory signals informing the brain on the onset of leg fall. Beside its mechanical role, the ACL is recognized as a major sensory structure, which contains receptors that have low thresholds to mechanical stimulation, and others that are activated only when the tension of the ligament is very high (Sjölander et al. 2002; Solomonow 2006). Afferents emanating from joint mechanoreceptors have been shown to project to spinal motoneurons and interneurons, as well to a number of supraspinal structures (Sjölander et al. 2002). Overall, these findings would strongly suggest that mechanosensitive nerve endings of the ACL are involved in providing the CNS with information about the knee joint position and movement. This is indirectly supported by an impaired ability to recognize the knee position in space, referred to as joint position sense, in individuals with ACL-deficient or reconstructed knees (Skinner and Barrack 1991; Carter et al. 1997). In previous histological studies, it has been shown that sensory innervation of the patellar tendon autograft needs a minimum of 5 months to be fully recovered after surgery (Aune et al. 1996). Therefore, since our task involved postural disturbances exclusively at the knee level, it is plausible that the

loss of joint afferents from the ligament and the resulting lack of information on the onset of leg fall might have contributed to the delayed launch of CPAs in patients with ACLR with respect to healthy participants.

Patients with ACLR exhibited lower mean amplitude of compensatory responses for all of the three muscles than healthy participants, although the between-group differences were significant only for the VL muscle. This might be due to altered regulation of joint and muscle stiffness as a result of abnormalities in the gamma-muscle spindle system of patients with ACLR compared to healthy individuals. Abnormal gamma loop sensitivity, in fact, has been reported after ACLR (Konishi et al. 2002) and might alter signals from muscle spindles, which are known to play a major role in signaling information about the limb position in terms of muscle length (Proske and Gandevia 2012). The ability of a joint to maintain stability against a perturbation seems to depend on its stiffness which, among other factors, depends on the stiffness of the muscles crossing it (Obusek et al. 1995). Joint and muscle stiffness can be dynamically regulated by means of modification in the intensity of muscle activation (Silva et al. 2009). Hence, an increased activation of the muscles that cross a joint would increase joint stiffness and result in a greater capability of the joint to resist external loads (i.e., greater dynamic stability); on the contrary, diminished levels of muscle contraction might decrease a person's ability to resist perturbation (Fonseca et al. 2004), which might be the case as in the present study. Our results, however, are in contrast with those of Madhavan and Shields (2011), who reported higher amplitude of long-latency muscle responses to unexpected perturbations in patients with ACLR compared to healthy individuals during a dynamic single-leg weight-bearing task. The differences between Madhavan and Shields (2011) and the results of the present study might be attributed to the task being performed, since long-latency responses may be modulated in a manner appropriate to meet the motor demands and give protection to forthcoming perturbations (Dietz et al. 1994; Shemmell et al. 2010; Pruszynski et al. 2011). The weight-bearing task being performed in Madhavan and Shields (2011) is a closed kinetic chain movement, in which activation of the knee extensor muscles causes lower anterior shear

forces on the tibia and lower strain on the ACL than during open kinetic chain movements (Lutz et al. 1993) that are similar to the semi-reclined task in this study. Furthermore, it is noteworthy that such a reduction in the magnitude of activation was found only for the VL muscle, which acts exclusively on the knee joint, and not for the RF muscle, which acts on both the knee and the hip joints (Jacobs et al. 1993). The significant reduction in activation magnitude of the VL muscle in ACL patients with respect to healthy participants, hence, may represent a protective mechanism to minimize anterior shear forces caused by the forthcoming perturbation to the knee joint (Venema and Karst 2012) and, in particular, to the reconstructed ligament. From a motor control point view, when the time of perturbation is unpredictable, but its amount (corresponding to the knee gravitation torque in our task) is known, the central nervous system may pre-program an appropriate level of muscle activation and wait for the sensory signal to drive the motor command. Since long-loop circuits are involved, this process can take place, thus allowing the sensory signal to be specifically associated to the task and the motor response to be adapted instantaneously to the actual circumstances (Lewis et al. 2006; Kurtzer et al. 2008; Pruszynski and Scott 2012; Pruszynski et al. 2011; Casabona et al. 2012; Valle et al. 2013).

Anticipatory postural responses to predictable perturbations

During the predictable condition of the present study, in healthy participants, almost all onset times of the postural responses of the involved limb muscles occurred earlier or at the same time than the beginning of the expected perturbation caused by the supporting limb movement. This is in agreement with previous studies on bimanual tasks, wherein an anticipatory adjustment of the forearm muscles in one upper limb prevents the postural disturbances related to a forthcoming self-initiated movement of the contralateral upper limb (Dufossè et al. 1985; Bennis et al. 1996; Massion et al. 1999). In the perturbation task adopted in the present study, APAs of the involved limb muscles must be implemented by the central nervous system (CNS) to predict and prevent the perturbing consequences of the supporting limb action, hence bypassing long delays associated with

feedback (Santello 2005; Aimola et al. 2011). Noteworthy, the onset of anticipatory postural responses in patients with ACLR occurred earlier than in healthy participants for all of the lower limb muscles. This might represent a safety strategy adopted by patients with ACLR to ensure minimization of postural disturbances and maintain functional joint stability. Similar findings have been reported in ACL-deficient individuals during complex motor tasks, such as landing from jumping (Swanik et al. 2004) and an abrupt deceleration while running (Steele and Brown 1999). Accordingly, Gokeler et al. (2010) reported that muscle onset times prior to landing occurred significantly earlier in the operated limb than in the opposite limb of patients with ACLR 6 months after surgery. In contrast, however, Bryant et al. (2009) reported no significant differences in muscle activation patterns prior to landing between patients with ACLR 1 year after surgery and healthy individuals. Yet, Venema and Karst (2012) found no delayed anticipatory muscle onsets of the lower limb muscles during a standing reaching task in individuals with knee arthroplasty following 3 months from surgery compared with healthy control subjects. Such a discrepancy with the results of the present study, however, might be reasonably due to differences in the time from surgery of the participants. As the task adopted in the present study does not excessively overload the knee joint, it can be safely carried out as early as 2 months after ACLR. To the knowledge of the authors, therefore, this is the first study reporting abnormalities in APAs during the early phase of rehabilitation after ACLR. Furthermore, such a task involves rotation of the knee joint only, thus ruling out compensatory responses from muscles acting on other joints, but highlighting abnormalities in APAs of the knee extensor and flexor muscles.

Alternatively, the earlier anticipatory timing in patients with ACLR than in healthy participants might be related to an altered knowledge of the limb properties, as a result of the disrupted sensorial information arising from the involved knee of patients with ACLR, which has been discussed in the section on compensatory postural responses to unpredictable perturbations. The ability to predict the effects of the forthcoming disturbances on postural control, in facts, is

thought to rely on knowing the interactions between the environment and the mechanical properties of the involved limb, i.e., an internal model (Kurtzer et al. 2008).

It is noteworthy that the earlier onset of anticipatory activation in patients with ACLR than in healthy participants occurred both in the knee extensor and flexor muscles. This is in agreement with previous studies suggesting that knee agonist–antagonist muscle co-activation would represent a protective mechanism aimed at preventing anterior translation of the tibia and ensuring knee stability in ACL reconstructed patients (Lustosa et al. 2011; Madhavan and Shields 2011). The early pre-tension of the limb muscles before the perturbation, in fact, is supposed to increase the sensitivity of muscle spindles, thus allowing joint perturbation to be detected more quickly (Dyhre-Poulsen et al. 1991).

Conclusions

In conclusion, the present study showed that abnormalities in both CPAs and APAs of the knee extensor and flexor muscles may be identified and quantified by means of a joint perturbation task as early as 2 months from ACLR. Such abnormalities might reflect the inability of the central nervous system to quickly detect sudden changes in muscle length or to completely activate muscles surrounding the knee. The earliest goals after ACL reconstruction, therefore, should be to target both CPAs and APAs as soon as possible to progress to further stages of the rehabilitation program. Future studies with a prospective and longitudinal design should focus on whether or not these abnormalities change over time and can improve by rehabilitation.

ETHICAL STANDARDS

The above outlined experiment complied with the ethical standards and laws of the country in which it was carried out.

CONFLICT OF INTEREST

The authors declare that they have no conflict of interest.

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FIGURE CAPTIONS

Fig 1 Experimental setup and starting reference position of participants prior to each perturbation trial.

Fig 2 Representative kinematic and EMG traces recorded in one of the healthy participants during an unpredictable (a) and a predictable (b) perturbation trial. Angular displacement of the knee joint is shown on the upper trace of each example and is followed downward by the full-wave rectified and filtered EMG traces of the contralateral and ipsilateral vastus lateralis (VL) muscles.

Fig 3 Latency of compensatory postural responses for the vastus lateralis (VL), rectus femoris (RF) and biceps femoris (BF) muscles with respect to the onset of leg movement, which was identified relative to the onset of the knee angular displacement, in patients with ACLR and healthy participants. * = significantly different from healthy participants.

Fig 4 Distribution of the latencies of compensatory postural responses for the vastus lateralis (VL), rectus femoris (RF) and biceps femoris (BF) muscles with respect to the onset of leg movement in patients with ACLR (a) and healthy participants (b).

Fig 5 Amplitude of compensatory postural responses for the vastus lateralis (VL), rectus femoris (RF) and biceps femoris (BF) muscles in patients with ACLR and healthy participants. * = significantly different from healthy participants.

Fig 6 Latency (a) and amplitude (b) of anticipatory postural responses for the vastus lateralis (VL), rectus femoris (RF) and biceps femoris (BF) muscles in patients with ACLR and healthy participants. Latencies were calculated with respect to the onset of movement, which was identified

as the offset of the VL muscle in the supporting limb. * = significantly different from healthy participants.

Fig 7 Peak angle normalized for the plateau angle in patients with ACLR and healthy participants during the unpredictable and the predictable condition.

Figure 1

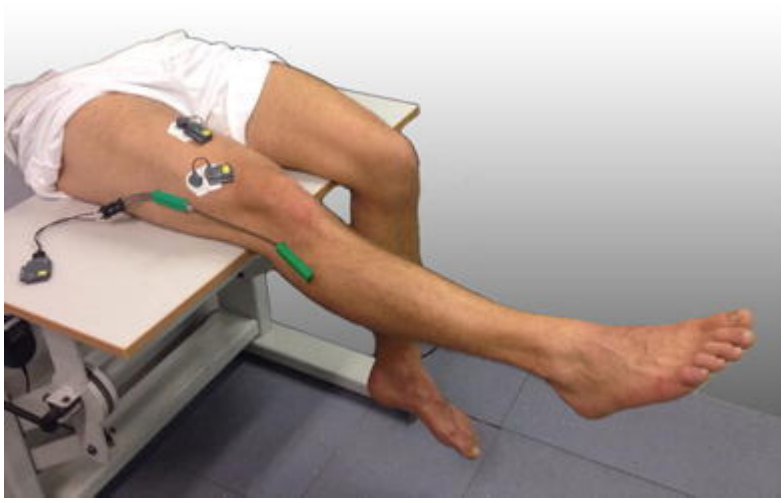

Figure 2

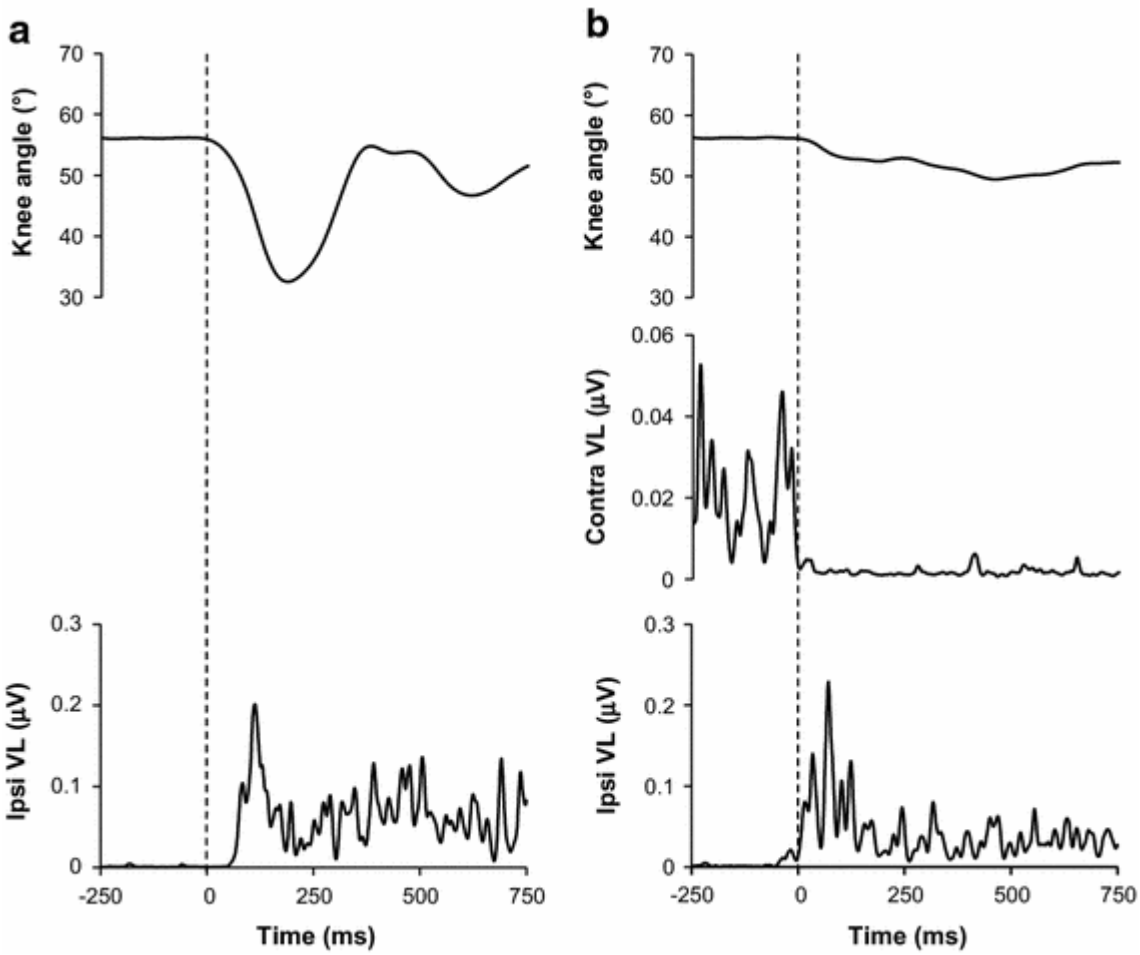
a
b
Knee angle (°)
70
60
50
40
30
Contra VL (μV)
0.06
0.04
0.02
0
Ipsi VL (μV)
0.3
0.2
0.1
0
-250
0
250
500
750
Time (ms)

Figure 3

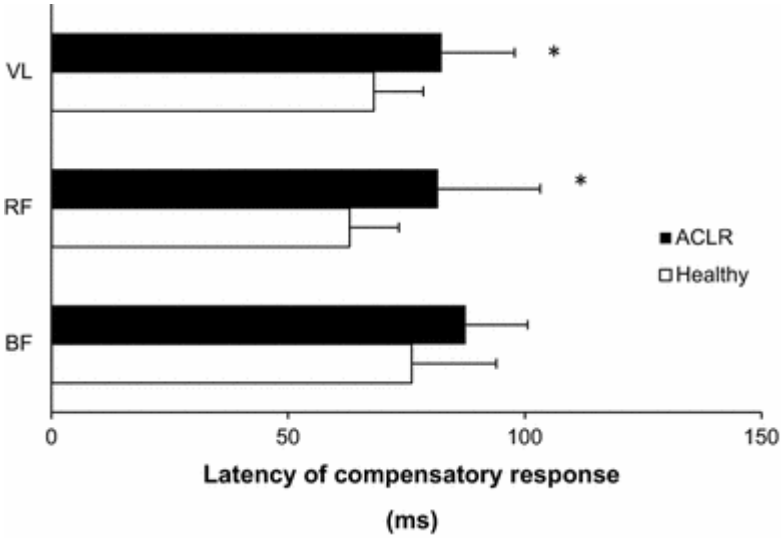
VL
RF
BF
*
*
ACLR
Healthy
0
50
100
150
Latency of compensatory response
(ms)

Figure 4

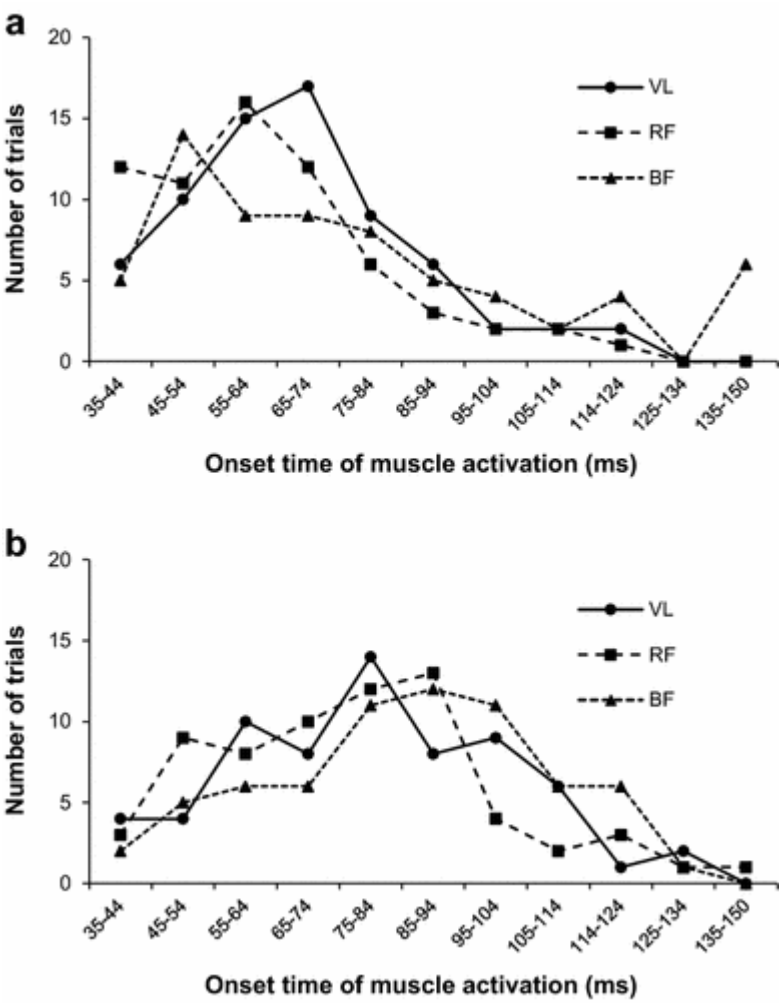
a
Number of trials
20
15
10
5
0
VL
RF
BF
35-44
45-54
55-64
65-74
75-84
85-94
95-104
105-114
114-124
125-134
135-150
Onset time of muscle activation (ms)
b
Number of trials
20
15
10
5
0
VL
RF
BF
35-44
45-54
55-64
65-74
75-84
85-94
95-104
105-114
114-124
125-134
135-150
Onset time of muscle activation (ms)

Figure 5

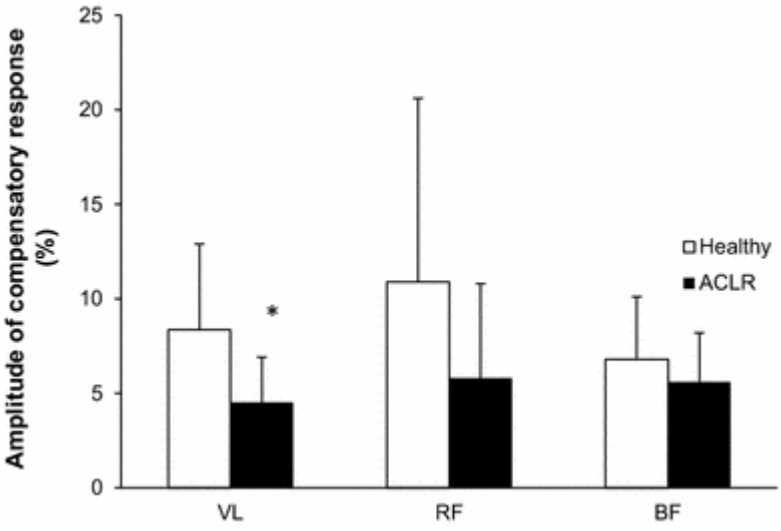
Amplitude of compensatory response (%)
25
20
15
10
5
0
*
Healthy
ACLR
VL
RF
BF

Figure 6

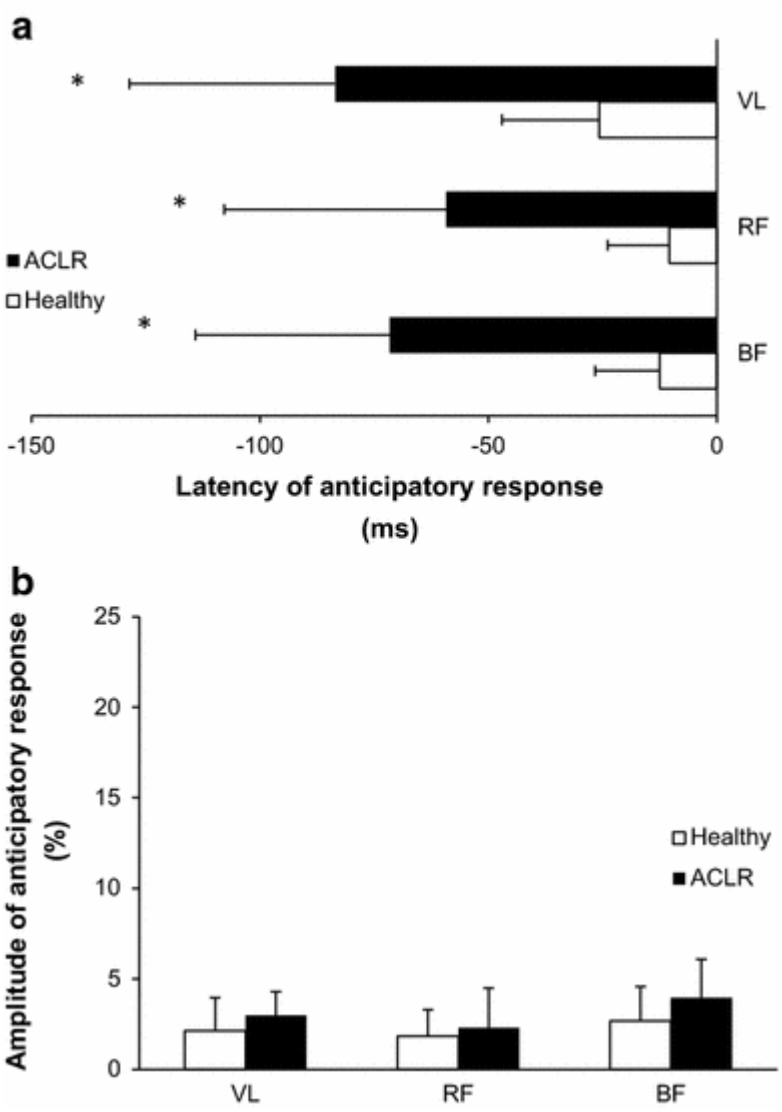
a
*
*
*
VL
RF
BF
ACLR
Healthy
-150
-100
-50
0
Latency of anticipatory response
(ms)
b
Amplitude of anticipatory response
(%)
25
20
15
10
5
0
Healthy
ACLR
VL
RF
BF

Figure 7