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ORIGINAL ARTICLE

Electromechanical delay of the hamstrings following semitendinosus tendon autografts in return to competition athletes

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Abstract

Purpose Knee flexor electromechanical delay (EMD) has been proposed as a contributing factor to non-contact anterior cruciate ligament (ACL) injury risk and the semitendinosus (ST) autograft technique has been shown to impair knee flexor torque at large angles of knee flexion. The purpose of this study was to analyse the effects of ACL reconstruction (ACLR) using the ST tendon autograft technique on knee flexor EMD across the knee flexion range of motion, in athletes who had returned to competition.

Methods Athletes with ACLR ($n = 8$ females, $n = 3$ males, 1.7 ± 0.5 years post-surgery) and non-injured control athletes ($n = 6$ females, $n = 4$ males) performed rapid maximal voluntary contractions of isometric knee flexion and extension at 30°, 50°, 70°, 90°,and 105° of knee flexion. Electrical activity of the ST, biceps femoris (BF), vastus lateralis, and vastus medialis was recorded using surface electromyography.

Results No change in EMD for the knee flexors or extensors was observed across joint angles. Greater EMD was found only for the BF in the ACLR limb of injured athletes compared to the contralateral limb ($P < 0.05$). In post-hoc analysis, evidence of ST tendon regrowth was noted for only 2/11 athletes.

Conclusion While the EMD-joint angle relationship appeared to be unaffected by ST tendon harvest for ACLR, the absence of ST tendon regrowth should be considered. Despite return to competition, greater BF EMD was found, which may impair knee joint stabilization capacity by delaying the transfer time of muscle tension to the tibia after ST autograft.

Keywords ACL reconstruction · Hamstring autograft · Semitendinosus · Electromechanical delay · EMD

Abbreviations

ACL	Anterior cruciate ligament
ACLR	Anterior cruciate ligament reconstruction/reconstructed
EMD	Electromechanical delay
EMG	Electromyography
MVC	Maximal voluntary contraction
BF	Biceps femoris
ST	Semitendinosus
SM	Semimembranosus
RFD	Rate of force development
VL	Vastus lateralis
VM	Vastus medialis

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Introduction

The hamstring muscles work synergistically with the anterior cruciate ligament (ACL), providing active restraint against anterior tibial translation to maintain knee joint stability (Baratta et al., 1988; Solomonow et al., 1987). For example, hamstring co-contraction reduces stress on the ACL that can be imposed by a high-force quadriceps contraction (Shimokochi and Shultz, 2008).

Improving maximal force capacity of the hamstrings in relation to the quadriceps has been suggested to increase knee joint stability and this may be protective against ACL injury (Aagaard et al. 1998; Holcomb et al. 2007).

Non-contact ACL injury during athletic movements, such as cutting or jump landing, is estimated to occur within 50 ms of initial foot contact with the ground (Krosshaug et al., 2007). In contrast, the time needed to reach maximal force in isometric contractions takes more than 300 ms. Therefore, the capacity to stabilize the knee joint and protect against ACL injury may depend less on hamstring maximal strength and more on hamstring rapid force producing ability (rate of force development—RFD) (Hannah et al. 2014; Jordan et al. 2014). Alongside RFD, knee joint stabilization may be affected by the delay in time from the onset of hamstring muscle electrical activity to the development of torque at the joint (Hannah et al. 2015; Hannah et al. 2014). This time difference is known as electromechanical delay (EMD) and is related to the size and length of the muscle, fibre type composition, stiffness of in-series elastic components of muscle, and presence of fatigue (Cavanagh and Komi, 1979; Muraoka et al. 2004; Zhou 1996). A longer EMD of the hamstring muscles decreases the fraction of hamstrings relative to quadriceps force in the first 50 ms of a fast muscle action, which may decrease knee joint stability and increase anterior tibial translation (Hannah et al. 2014).

Following ACL injury, reconstructive surgery (ACLR) is often performed to restore knee joint stability and improve the likelihood of successful return to sport (Ardern et al. 2011). A common ACLR technique involves harvesting the semitendinosus (ST) tendon from the athlete's injured limb to serve as graft material. The advantages of ST tendon autografts for ACLR include increased recovery of quadriceps strength, reduced incidence of anterior knee pain, and ease of harvest during surgery (Corry et al. 1999; Feller and Webster, 2003; Shaieb et al. 2002).

However, the ST tendon autograft procedure leads to significant neuromuscular deficits, such as reduced ST muscle cross-sectional area, volume and length (Konrath et al. 2017; Nishino et al. 2006), decreased knee flexor strength at large knee joint angles (Makihara et al. 2005; Nomura et al. 2014), and altered hamstring muscle activation (Messer et al. 2019) which can persist for years, even after return to sport. Further, despite possible regeneration of a tendon-like tissue, the mechanical properties of the neo-tendon remain altered beyond 12 months (Suydam et al. 2017). The changes in ST muscle and tendon mechanical and morphological properties after ST autograft lead to decreased rate of torque development consequent to neuromechanical factors like the EMD. Greater EMD has been reported in the ST of the injured limb following ACLR with ST tendon autograft during isometric contractions at 30° of knee flexion (Freddolini et al. 2015; Ristanis et al. 2009). However, while it is well established that ACLR using the ST autograft technique impairs the normal knee flexion torque–joint angle relationship especially at large angles of knee flexion (Makihara et al. 2005; Nomura et al. 2014), there is limited information on the impact of the ST tendon autograft procedure on the EMD–joint angle relationship. As knee flexion angle increases, the moment arm and activation level of the ST muscle increases, suggesting differing levels of contribution throughout the knee flexion range of motion (Herzog and Read 1993; Onishi et al. 2002). Consequently, impairment of EMD of the ST following ACLR may also contribute to knee flexor strength loss at large angles of knee flexion. Moreover, the available research on the effect of ACLR on the EMD of the lateral hamstring muscles, the biceps femoris (BF), is conflicting (Freddolini et al. 2015; Ristanis et al. 2009). Thus, a greater understanding of the effects of ACLR on EMD is warranted, particularly after athletes have returned to sport, where the incidence of ACL reinjury is the highest (Paterno et al. 2014).

The purpose of this study was to investigate the effect of the ST autograft procedure on the EMD of the hamstring muscles across the knee flexion range of motion in athletes with ACLR who had returned to competition. The EMD of the quadriceps muscles was also evaluated. We hypothesized that EMD would be greater as knee flexion angle increased, and based on previous findings (Ristanis et al. 2009), we hypothesized a longer EMD of both the ST and BF muscles in the ACLR limb compared to contralateral and control limbs. We also hypothesized that there would be no difference in quadriceps EMD between the ACLR and control limbs.

Methods

Participant characteristics

Eleven athletes competing at the national, provincial or university collegiate sport levels with a history of ACLR (females, $n=8$: age = 21.2 ± 4.0 years, mass = 72.0 ± 9.5 kg, 1.7 ± 0.4 years post-operative; males, $n=3$: age = 24.5 ± 3.7 years, mass = 85.4 ± 4.1 kg; 1.7 ± 0.8 years post-operative) and ten non-injured control athletes without ACLR (females, n = 6: age = 19.8 ± 1.7 years, mass = 70.0 ± 11.6 kg; males, n = 4: age = 21.0 ± 2.2 years, mass = 81.1 ± 7.0 kg) were recruited to participate in this study (Table 1). ACLR athletes were included if they sustained an isolated unilateral ACL tear without associated knee ligament injury, had ACLR using quadrupled ST tendon autograft, were between 12 and 36 months post-ACLR surgery, returned to competition without restriction and were pain free. Nine of the eleven ACLR athletes had injured their right limb. Control athletes currently being treated for a lower limb injury, currently experiencing lower limb pain or with history of lower limb surgery other than ACLR were excluded. The study protocol was approved by the Conjoint

Table 1 Participant characteristics (Mean ± SD)

	ACLR		Control	
	Male	Female	Male	Female
N	3	8	4	6
Age (years)	24.5 ± 3.7	21.2 ± 4.0	21.0 ± 2.2	19.8 ± 1.7
Mass (kg)	85.4 ± 4.1	72.0 ± 9.5	81.1 ± 7.0	70.0 ± 11.6
Post-Op (years)	1.7 ± 0.8	1.7 ± 0.4	N/A	N/A
Sport				
Winter Slope	3	3	3	3
Basketball	0	2	1	0
Volleyball	0	2	0	2
Rugby	0	1	0	1
Level				
National	3	1	3	1
Provincial	0	2	0	2
Collegiate	0	5	1	3

Research Ethics Board at the University of Calgary and all participants gave written informed consent to participate.

Protocol

Testing was completed following a rest day (no training or competition) to minimize the effects of fatigue on the outcome measures. After a standardized warm up, isometric maximum voluntary contractions (MVCs) of knee flexion and knee extension were performed on a customized dynamometer while hamstring and quadricep muscle electrical activity was recorded via surface electromyography (EMG) (Fig. 1). All testing was supervised by a certified exercise professional. The presence of ST tendon regenerated tissue in the ACLR limb was evaluated by a qualified ultrasound technician (> 7 years of experience) within 7 days of the isometric torque measurements.

Torque measurements

Torque measurements were collected on a Cybex dynamometer instrumented with a third-party load cell (Omega, LC703-500, Stamford, Connecticut, USA) sampled at 1500 Hz (MyoResearch Version 3.8, Noraxon, Scottsdale, Arizona, USA) (Jordan et al. 2020). Isometric knee flexion was performed with subjects in a prone position with straps firmly fastened over the hips to minimize any change in body position during the tests. Isometric knee extension was performed with subjects seated upright with a hip angle of 100° and strapped to the dynamometer with two belts crossing the chest and one belt crossing the hips. The load cell, encased in a firm and padded bracket, was strapped 2.5 cm above the lateral malleolus, and the lateral epicondyle of the femur was aligned with the axis of rotation of the dynamometer. Participants completed 3–5 submaximal warm-up contractions of increasing intensity, followed by 3 × 2 s isometric MVCs at 30°, 50°, 70°, 90°, and 105° of knee flexion (0° knee flexion = full knee extension). Participants began from a fully relaxed state and prior to each contraction were instructed to contract as "fast and hard" as possible (Maffiuletti et al. 2016). The tester provided strong verbal encouragement during contractions. A 20-s rest period was provided between contractions and a 2-min rest period was provided between angles. The order of angle, limb and contraction type were randomized for each subject.

The effect of gravity on the dynamometer force reading was digitally corrected at the start of each recording. Knee joint torque was calculated by measuring the distance from the axis of rotation to the point of force application. Torque-time curves were smoothed using Matlab 'smooth' function with a 25-ms centred moving average window. The onset of the torque signal was defined as a 3.6 Nm deviation above the baseline level (Freddolini et al. 2015; Ristanis et al. 2009; Zhou et al. 1995).

Surface electromyography recordings

The vastus medialis (VM), vastus lateralis (VL), BF long head, and the ST were identified by the same researcher. The skin was shaved and cleaned using isopropyl alcohol solution. Bipolar surface electrodes (Norotrode, Ag, AgCl electrodes) with a 2-cm interelectrode distance were applied parallel to the muscle fibers on the middle of the muscle belly, according to SENIAM recommendations (Hermens et al. 2000). The EMG preamplifiers were taped to the skin and large pieces of medical bandage were used to cover the electrodes and amplifiers to reduce movement artifacts. Signals from the eight EMG electrodes were recorded using a telemetric EMG receiver (Telemyo DDTS Desktop Receiver; Noraxon, Scottsdale, AZ). EMG signals were preamplified with an overall gain of 500 and filtered with a first-order high-pass filter set at 10 Hz. The EMG signals were recorded simultaneously at 2000 Hz with the torque signals (MyoResearch Version 3.10, Noraxon). EMG data were band-pass filtered offline (third-order Butterworth filter; 20–500 Hz) using the 'filtfilt' zero-phase digital filtering function from Matlab, and were smoothed using a point by point 50 ms symmetric root mean square filter (filtfilt function, Matlab).

The onset of the EMG signal for knee flexion was defined as a 15 μV deviation from the baseline (Freddolini et al. 2015; Ristanis et al. 2009; Zhou et al. 1995) and EMD was calculated as the time interval between the onset of the EMG signal and the onset of torque (Fig. 2). For knee extension, the onset was reduced to a 5 μV deviation from the baseline to account for the reduced time delay between the EMG and torque signal. Quadriceps EMD for one ACLR athlete and one control athlete could not be analysed due to signal complications. The mean EMD values of the three contractions at each angle were used for statistical analysis.

Statistical analysis

To detect differences in maximal knee flexion/extension EMD, linear mixed effects models (lme4 package, R, Version 1.1-26) were fit with fixed effects for angle (factor levels: 30°, 50°, 70°, 90°, and 105°), limb status (factor levels: Control-Left, Control-Right, Injured-Contralateral, Injured-ACLR), and muscle (factor levels: BF and ST, or VM and VL) as well as the interaction between angle, limb status, and muscle. Athletes were included in the model as random intercepts, with limb (left and right) nested in athlete to account for the repeated measurements at each angle. Likelihood ratio tests were used to assess fixed effects by comparing the full model with the effect in question against the model without the effect in question ($\alpha = 0.05$). When an interaction effect was revealed by the model, contrast analyses were performed to compare model estimates for each angle and muscle across limb status. Model residuals were assessed for deviations from homoscedasticity and normality, and all assumptions were met. Post-hoc comparisons were made using the Tukey adjustment (emmeans package, R, Version 1.4.1).

Results

A limb status by muscle interaction effect was found for knee flexor EMD ($\chi^2 = 12.9$, df = 2, $P < 0.01$) (Fig. 3). EMD of the BF was longer in the ACLR limb and Control limb at all angles ($P < 0.05$) compared to the contralateral limb. Post hoc analyses revealed no statistical difference in EMD between limb status for the ST. Pairwise comparisons for the ACLR limb versus non-injured contralateral limb are presented in Fig. 4.

A limb status by muscle interaction effect was found for knee extensor EMD ($\chi^2 = 13.1$, df = 2, $P < 0.01$) (Fig. 5). EMD of the VM was longer in the Control limb compared to the ACLR limb at all angles ($P < 0.05$). No differences in EMD were found between limb status for the VL. Pairwise comparisons for the ACLR limb versus non-injured contralateral limb are presented in Fig. 6.

Discussion

In the present study, we evaluated the EMD of the ST and BF across the knee flexion range of motion in athletes who had recently returned to competition following ACLR with the ST tendon autograft procedure. EMD is a determinant of explosive strength capacity measured as the rate of force development (RFD), which is important for sport performance and sport injury prevention (Hannah et al. 2015; Tillin et al. 2010). To our best knowledge, this study contributes new information on the effects of ST autograft on EMD at both small and large angles of knee flexion. Since non-contact ACL injury occurs in a short time frame (< 50 ms after initial ground contact), the ability to rapidly produce knee flexion torque is considered critical for knee joint stabilization capacity and safeguarding against anterior tibial translation causing ACL injury (Hannah et al. 2014; Jordan et al. 2014). Further, the prevalence of ACL reinjury is high, and while the ST autograft procedure has clinical advantages such as a reduction in anterior knee pain, the risk of ACL revision may be higher in patients with an ST autograft procedure compared to other surgical methods (Mohtadi et al. 2016; Spindler et al. 2020). Therefore, studies aimed at understanding the comorbidities associated with an autograft procedure are of importance to surgeons and clinicians working with athletic populations. Contrary to our study hypotheses, only the BF of the ACLR limb displayed a significantly greater EMD compared to the contralateral limb. Additionally, a similar EMD was observed in the ACLR limb and Control limb conditions for both BF and ST. Further, there was no change in the EMD of the quadriceps or hamstrings throughout the knee flexion range of motion. Though not described here, deficits in knee flexion torque, rate of torque development, and hamstring muscle cross-sectional area of the ACLR limb were also found (Morris et al. 2020).

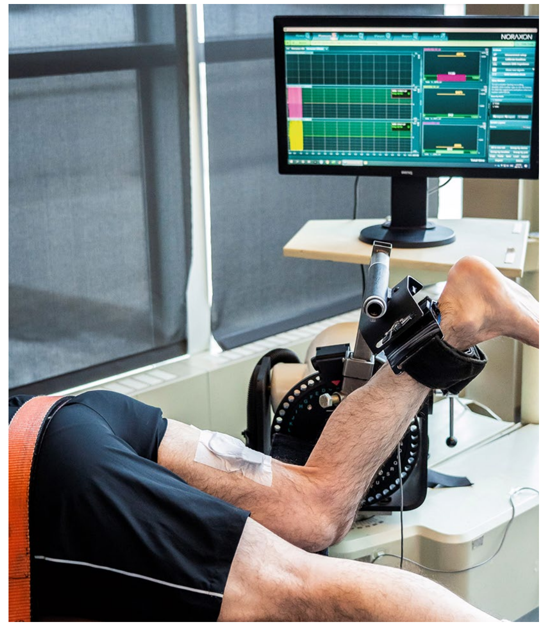

Fig. 1 The customized dynamometer used for testing

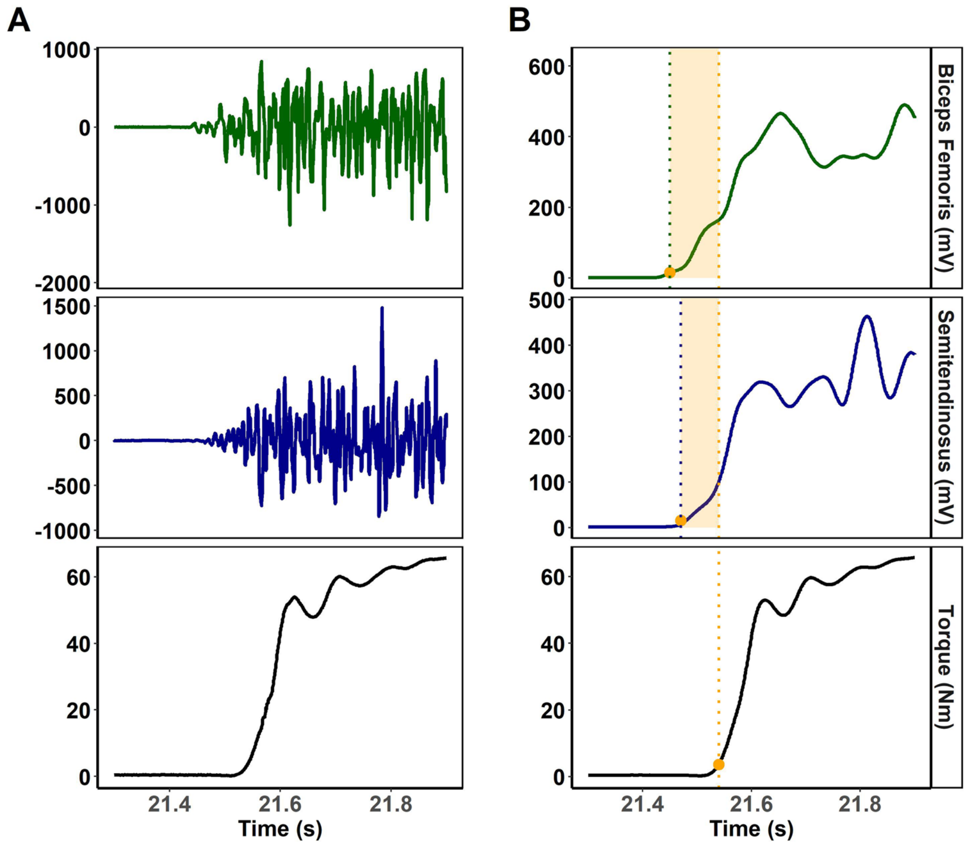

Fig. 2 Panel A displays the synchronized unprocessed electromyographic (EMG) signal for the biceps femoris (BF) and the semitendinosus (ST) muscles and knee flexion torque for a knee flexion contraction. Panel B displays the processed EMG signal (50 ms symmetric root mean square filter) for the ST and BF muscles and the smoothed knee flexion torque signal (25 ms centred moving average window). The large orange dots in Panel B mark the time of onset for the EMG and torque signals as determined by the automatic thresholds of 15 mV and 3.6 Nm, and the orange shaded areas illustrate the electromechanical delay

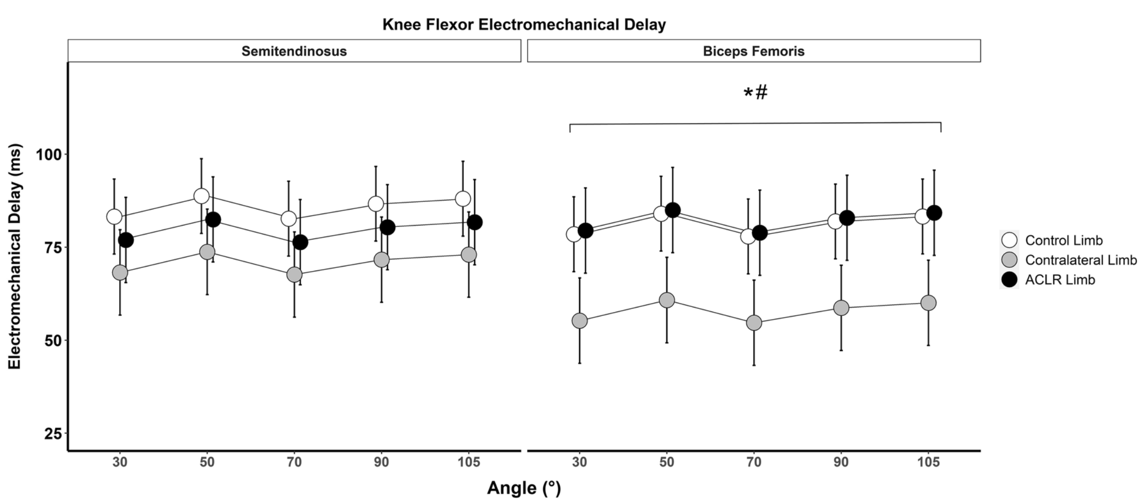

Fig. 3 Comparison of knee flexor electromechanical delay (EMD) between limb conditions and across joint angles. EMD of the biceps femoris (BF) was longer for the anterior cruciate ligament reconstructed (ACLR) limb and Control limb compared to the non-injured contralateral limb [* ACLR limb significantly different from contralateral limb ($P<0.05$); # Control limb significantly different from contralateral right limb ($P<0.05$)]. Data are presented as the model effects and standard error of the effects

A primary aim of this study was to assess EMD across the knee flexion range of motion to determine whether deficits in EMD resulting from the ST autograft procedure were specific to knee joint angle. As knee flexor strength deficits have been shown to be more pronounced at larger angles of knee flexion in patients with ST autografts, an understanding of the neuromechanical changes across the knee flexion range of motion may be important for informing rehabilitation training strategies (i.e., when the hamstrings are in a shortened position) (Makihara et al. 2005; Nomura

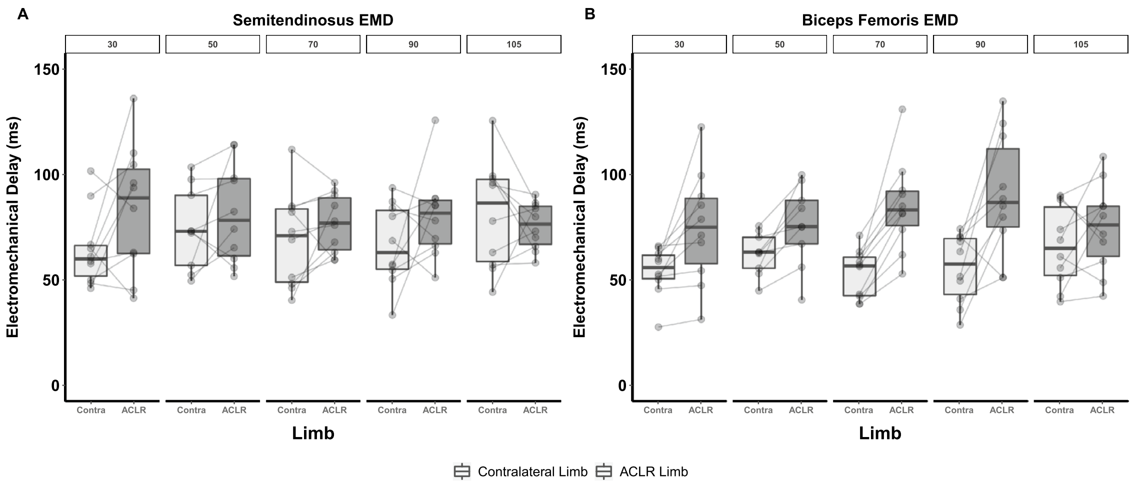

Fig. 4 Pairwise comparison of the anterior cruciate ligament reconstructed (ACLR) limb versus non-injured contralateral (Contra) limb across the tested joint angles for electromechanical delay (EMD) of the semitendinosus muscle (ST) (Panel A) and the biceps femoris muscle (BF) (Panel A). A systematically greater EMD in the ACLR limb was found across all angles of knee flexion for the BF (Panel B)

et al. 2014). For example, the ACL is tensioned in extension and large angles of knee flexion, and a common ACL injury mechanism in winter slope sports involves landing back weighted in a highly flexed knee position (Jordan et al. 2017). However, no relationship between knee angle and EMD was found. Previous research investigating the effect of joint angle on EMD of the upper extremities in healthy subjects suggests that EMD is independent of joint angles when tendon slack is fully taken up by an elongated muscle–tendon unit (Lacourpaille et al. 2017; Muraoka et al. 2004; Sasaki et al. 2011). More specifically, EMD is only affected by joint angle when the muscle–tendon unit length is shorter than the length that results in tendon slack (i.e., the length below which muscle begins to develop passive elastic force). For example, during isometric contractions of elbow flexion, the biceps brachii EMD is unchanged with increasing elbow joint flexion until a joint angle of 90°at which point the tendon becomes slack and EMD increases (Lacourpaille et al. 2017; Sasaki et al. 2011). It is possible that the maximum tested knee flexion angle of 105° was not large enough to sufficiently slacken the muscle–tendon unit of the hamstrings to observe an increase in EMD. Since the knee flexion active range of motion in a healthy knee joint is approximately 130–140°, future studies should look to assess EMD across a larger range of motion (Knapik et al. 1991; Roach and Miles, 1991).

No difference was found between the EMD of the ST between the ACLR and Contralateral limb, which is inconsistent with the scientific literature. Using similar methodologies, Ristanis et al. and Freddolini et al. found significantly greater EMD values of 22% and 12% for the ST in the ACLR limb 2 years after surgery (Freddolini et al. 2015; Ristanis et al. 2009). These observations were attributed to the structural, anatomical and mechanical changes of the donor site muscle–tendon unit. While EMD is influenced by electrochemical processes (i.e., the propagation of the action potential on the muscle membrane and excitation–contraction coupling), mechanical determinants such as the time required for the contractile element of muscle to stretch the passive series elastic components are important (Cavanagh and Komi, 1979). Therefore, changes in the mechanical properties of a muscle–tendon unit due to injury or training adaptations are likely to affect EMD. ACLR with the ST tendon autograft procedure leads to significant deficits in ST muscle volume and muscle length that persist beyond return to sport (Makihara et al. 2005; Nomura et al.2014). Also, although a tendon-like tissue can regrow in the ST tendon space, the recovery of the viscoelastic properties in the regenerated tendon takes beyond 12 months (Suydam et al. 2017). The reasons for the discrepancy between the present findings and those of the scientific literature are unclear. In addition to the possible contribution of measurement error and a lack of statistical power, it is possible that post-surgical anatomical adaptations contributed to the discrepant results. While not a part of the present study aims, post-testing ultrasound analysis of the participants revealed ST tendon-like tissue at the knee crease in only 2 out of the 11 ACLR limbs (*c.f.* Fig. 7). This suggests poor anatomical recovery of the donor muscle–tendon unit. ST muscle morphological changes (such as severe proximal retraction of the ST muscle tendon unit), along with the possibility of crosstalk from the surface EMG recording of the semimembranosus muscle may have impacted our findings. Nevertheless, future exploration into the association between the neuromechanical recovery and anatomical recovery after ST autografts seems warranted.

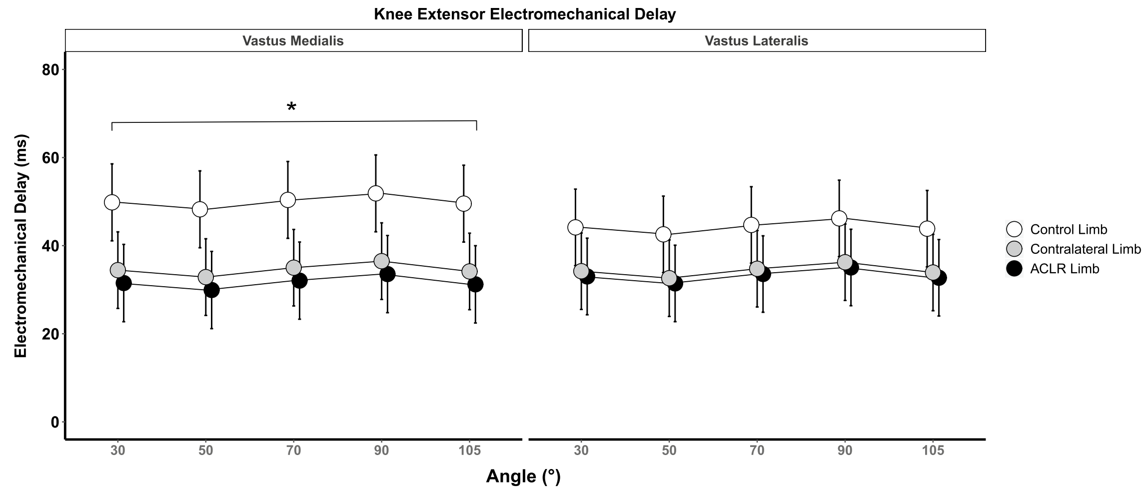

Fig. 5 Comparison of knee extensor electromechanical delay (EMD) between limb conditions and across joint angles. EMD of the vastus medialis (VM) was longer for the Control limb compared to the anterior cruciate ligament reconstructed (ACLR) limb and non-injured contralateral limb at all angles (* Control limb significantly different from ACLR limb and Contralateral limb ($P<0.05$)). Data are presented as the model effects and standard error of the effects

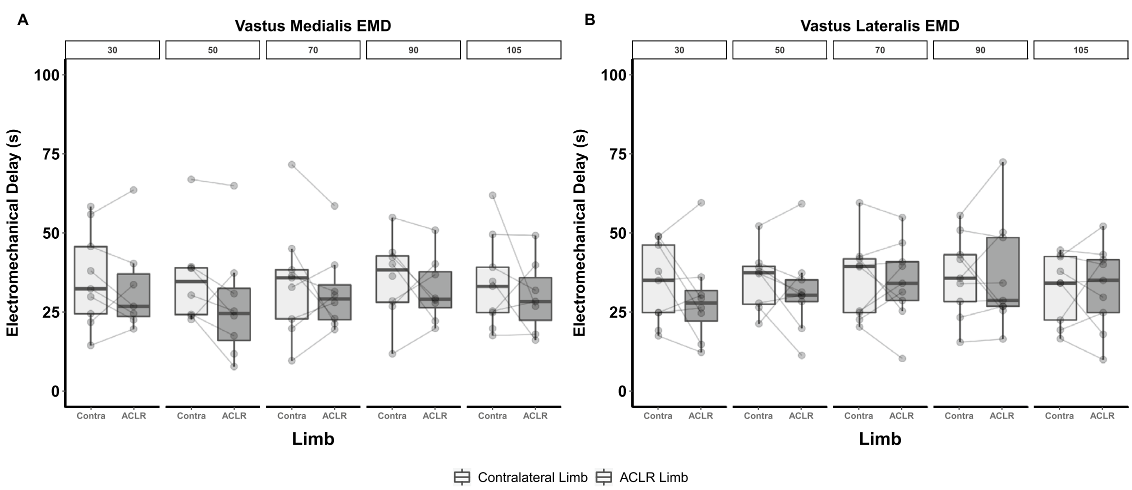

Fig. 6 Pairwise comparison of the anterior cruciate ligament reconstructed (ACLR) limb versus non-injured contralateral (Contra) limb across the tested joint angles for electromechanical delay (EMD) of the vastus medialis (Panel A) and the vastus lateralis (Panel B)

In agreement with Ristanis et al., a significantly greater EMD of the BF was observed in the ACLR limb compared to the contralateral limb (Ristanis et al. 2009). This may reflect detraining and inadequate rehabilitation of the ACLR limb hamstrings resulting in a more compliant BF muscle–tendon unit. Oppositely, this result may be interpreted as a positive training effect on the contralateral limb, since the ACLR limb and Control limb conditions were not different. EMD appears to be trainable, with studies showing decreases in EMD following resistance training programs (Kubo et al. 2001; Stock et al. 2016) and endurance training programs (Grosset et al. 2009). Kubo et al. found an 18% decrease in the EMD of the quadriceps following 12 weeks of isometric training at 70% MVC. This was accompanied by significant increases in quadriceps muscle volume, tendon stiffness, MVC, and rate of force development. Kubo et al. concluded that increased stiffness of tendon structures allowed for more effective transmission of muscle force, playing an important role in decreasing EMD (Kubo et al. 2001). While the mechanical and morphological properties of the muscle–tendon unit appear to be important determinants of EMD, in the present study, BF and ST tendon stiffness and architecture were not measured. Further, due to the observational study design, preinjury measurements were not obtained and so it is impossible to determine whether the greater BF EMD resulted from the ACLR surgery. More research is needed to differentiate the mechanisms affecting the change in EMD following ACLR with the ST tendon

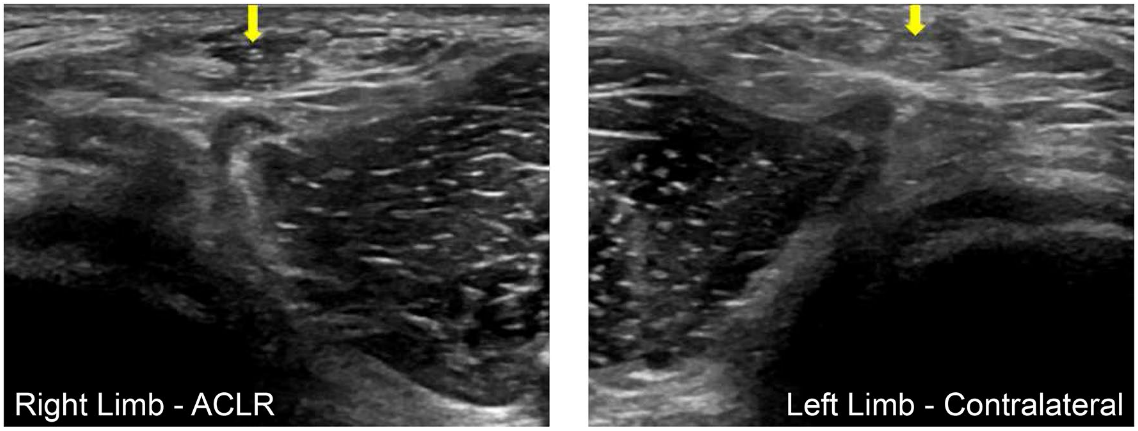

Fig. 7 Sample ultrasound image confirming no regeneration of tendon-like tissue in the semitendinosus tendon space at the knee crease of the anterior cruciate ligament reconstructed (ACLR) limb. Only 2 of the 11 ACLR limbs showed signs of tendon regeneration at this anatomical location

autograft procedure and prospective studies are warranted to determine the effectiveness of specific training methods on restoring knee flexor neural and mechanical function, including EMD. Finally, no differences were found between the ACLR limb and contralateral limb for EMD of the quadriceps. This was expected, as one of the major advantages of the ST tendon autograft over the bone-patellar tendon-bone autograft is reduced knee extensor mechanism comorbidities (Feller and Webster 2003).

Limitations

Although the methodology we applied for detecting onset of EMG signals and torque generation has been used in previous studies (Freddolini et al. 2015; Georgoulis et al. 2005; Ristanis et al. 2009; Zhou et al. 1995), there is no generally accepted and well-defined method for EMD calculation. Automatic detection methods may reduce the likelihood of bias, but manual identification methods have been found to be more accurate and detect onsets earlier (Tillin, Pain, & Folland, 2013). Nevertheless, our EMD values are in accordance with those employing similar automatic thresholds (Ristanis et al. 2009). Additionally, standardization of bipolar surface EMG electrode placement is challenging with respect to muscle innervation zones, which may affect EMD measurements. Future studies may benefit from using a 64-channel HD-EMG setup, where electrode placement can be closely controlled. Another limitation of this study was that the athletes had to perform a high number of maximal voluntary contractions throughout the testing protocol in order to test our hypotheses, which may have led to premature performance fatigability. However, we were working with high performance athletes for whom strength training was an integral part of their training regime. Furthermore, we randomized the order of joint angles so that possible fatigue effects across the group were controlled in our study design. Also, pilot results in an earlier study showed that the participants displayed sufficiently reliable performance for the maximal voluntary contractions used in the present experiment (coefficient of variation = 5%). Due to the small sample size, we were unable to control for sex, but sex-based differences in EMD were expected to be minimal (De Ste Croix et al. 2015; Hannah et al. 2015). Finally, owing to the observational study design, we were unable to collect preinjury data or control for the rehabilitation program that was implemented for the injured athletes.

Conclusion

In conclusion, a greater EMD was found for the BF in ACLR athletes with ST tendon autograft surgery despite unrestricted return to competition and a post surgery time of 1–3 years. These results are suggestive of persistent impairments in the neuromechanical and morphological properties of the knee flexors, which may compromise knee joint integrity by delaying the transfer time of muscle tension to the tibia. Future research should consider employing the neuromuscular assessments used in this study prospectively to identify if comorbidities arising from the ST autograft technique are trainable and to develop specific rehabilitation interventions to restore neuromuscular control of the knee flexors.

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Authors' contributions All authors contributed to the conception and design of the study. NM was responsible for data collection and preparing the first draft of the manuscript. All authors read, edited, and approved the final manuscript.

Compliance with ethical standards

Conflict of Interest The authors declare that they have no conflict of interest.

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