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Abstract

Purpose

Bone–patellar tendon–bone (BPTB) graft harvest for anterior cruciate ligament reconstruction alters patellar tendon properties, which inflict poor quadriceps neuromuscular function. BPTB autografts are associated with higher rates of posttraumatic osteoarthritis, which in turn is associated with pathological gait. The purpose of this study was to investigate the latency between the time of peak quadriceps activity and the peak knee flexion moment during gait, between those with BPTB grafts (*n* = 23) and other graft types (hamstring autograft or allografts, *n* = 54), 5 ± 2 months and 2 yr (25 ± 3 months) after anterior cruciate ligament reconstruction. We hypothesized that longer latencies would be observed in the BPTB graft group in the involved limb. We expected latencies to shorten over time.

Methods

Knee moments and quadriceps EMG were collected during gait, and vastus medialis, vastus lateralis, rectus femoris (RF), and quadriceps latencies were calculated. Linear mixed-effects models were used to compare latencies between graft types and over the two time points.

Results

The main effects of graft type were observed for vastus medialis (*P* = 0.005) and quadriceps (*P* = 0.033) latencies with the BPTB graft group demonstrating longer latencies. No main effects of graft type were observed for vastus lateralis (*P* = 0.051) and RF (*P* = 0.080) latencies. Main effects of time were observed for RF latency (*P* = 0.022).

Conclusions

Our hypothesis that the BPTB graft group would demonstrate longer extensor latency was supported. Contrary to our second hypothesis, however, latency only improved in RF and regardless of graft type, indicating that neuromuscular deficits associated with BPTB grafts may persist 2 yr after surgery. Persistent deficits may be mediated by changes in the patellar tendon's mechanical properties. Graft-specific rehabilitation may be warranted to address the long-term neuromechanical deficits that are present after BPTB graft harvest.

Anterior cruciate ligament (ACL) injury and reconstruction (ACLR) are reported at an increasing rate in high-level athletes[1,2]. ACLR most commonly involves the harvest of a bone–patellar tendon–bone (BPTB) or combined semitendinosus–gracilis tendon (hamstring) autograft, or a soft tissue allograft from a cadaveric donor. The use of a BPTB graft may be one of the best current surgical procedures for young athletes returning to high-level sports[3], but graft harvest from the patellar tendon also comes with a cost. The BPTB graft inflicts direct surgical insult to the extensor mechanism (patellar tendon) of the knee. As a result, BPTB graft harvest is associated with high levels of anterior knee pain[4,5], prolonged quadriceps weakness[6,7], and increased risks of posttraumatic osteoarthritis (PTOA) development compared with alternative graft types[8–10]. These factors can negatively influence an athlete's ability to return to sport and long-term quality of life.

The possible underlying sources of graft site morbidities after BPTB graft harvest include the additional surgical trauma and structural and mechanical alterations of the patellar tendon after surgery[11,12]. In the patellar

tendon after graft harvest, the mechanical properties of the central region of the tendon (where the BPTB graft is harvested) are altered[12,13]. Such changes must be considered over the course of rehabilitation because the latency in force production from the series elastic component of the patellar tendon contributes to the electromechanical delay of the quadriceps[14,15]. In the Achilles tendinopathy literature, tendons with reduced mechanical properties, such as higher strains and lower stiffness, also demonstrated longer electromechanical delay in the triceps surae[16]. Prolonged electromechanical delay can contribute to poor coordination of moment production at the knee joint, resulting in a clinical presentation that is commonly referred to as poor neuromuscular control[17,18]. Mechanical and morphological alteration within the extensor mechanism may play a role in the known graft site morbidities and pathological knee mechanics observed during athletic activities or gait after ACLR[19–22].

Current evidence indicates that aberrant gait mechanics and poor cyclical loading at the knee joint after ACLR contribute to PTOA development[19,22,23]. Quadriceps weakness and poor function are more profound early after ACLR in those who receive BPTB grafts compared with other graft types[6,7] and are associated with aberrant gait mechanics linked with PTOA[24,25]. The use of a BPTB graft is also associated with lower knee joint angles and moments compared with other graft types and healthy controls[8,26,27]. Quadriceps neuromuscular function during gait is diminished after ACLR[28,29] and may be further altered after BPTB graft harvest because of direct surgical insult to the extensor mechanism and contributing to altered gait biomechanics. Traditional methods of quantifying quadriceps neuromuscular function using electromechanical delay testing[30], or burst superimposition testing using a dynamometer[31], cannot, however, be used during functional activities such as gait.

A recent study by Ito et al.[28] introduced a novel method for quantifying the quadriceps muscles' neuromuscular function by using data from motion capture and electromyography (EMG) during gait. During the weight acceptance phase of gait, net internal knee extension moments produced primarily via quadriceps forces are equal and opposite to the net external knee flexion moments computed using inverse dynamics.
The quadriceps muscles activate to balance external moments, while accounting for latency in force production. Gait analysis using motion capture and simultaneous collection of quadriceps EMG activation patterns can be used to analyze temporal aspects of signals. No previous method has quantified the timing of EMG activity relative to moment output during a functional movement such as gait. The calculation of extensor latency is the first metric, to our knowledge, to quantify the timing-based force transferring ability of the extensor mechanism during gait, which may reflect the underlying neuromuscular deficit directly responsible for aberrant movement patterns. Hence, the purpose of this study was to investigate the latency between peak quadriceps muscle activity and peak external knee flexion moment (extensor latency) during gait between those with BPTB grafts and other graft types that do not involve direct surgical insult to the extensor mechanism (hamstring or soft tissue allografts), at baseline (5 ± 2 months) and 2 yr after ACLR. We hypothesized that at baseline, the involved limb quadriceps would demonstrate longer latency in those with BPTB grafts versus other graft types. Given that there were no mechanical alterations to the extensor mechanism in the uninvolved limb, we also hypothesized that there would be no differences in the uninvolved limb between graft types. Finally, we also expected the latency to improve from baseline to 2 yr as the patellar tendon has had a longer time to heal since surgery.

METHODS

Participants

This study was a secondary analysis of a cohort from a randomized control trial registered at clinicaltrials.gov (NCT01773317) and approved by the University of Delaware Institutional Review Board[32]. Of the 80 participants recruited for the randomized control trial, 77 participants who completed motion capture data collection at either (*n* = 13) or both (*n* = 64) the baseline (5 ± 2 months) and 2-yr (25 ± 3 months) time

points were included in this analysis (Table 1). All participants completed ethics board approved written informed consent forms.

Participants in this study sustained unilateral ACL injury before receiving primary ACLR. They were required to be participating in at least 50 h of level I or II sports[33] per year at the time of injury. At baseline, they demonstrated full and symmetric knee range of motion, trace or less effusion[34], at least 80% quadriceps strength index, and ability to hop on each leg without pain and initiated a running progression program. Exclusion criteria included any concomitant grade III ligament injury, a full thickness chondral lesion larger than 1 cm^2 indicated during surgery, or a history of serious injury or surgery to either lower extremity. Participants were randomized into two training groups: secondary prevention exercises and secondary prevention exercises plus perturbation training as part of the original randomized control trial. Our previous work has shown that the addition of perturbation training does not influence gait mechanics[20,21,35] or clinical or functional outcomes[36,37] after ACLR. The time points included in this study are from the pretraining (5 ± 2 months after ACLR) and at 2-yr follow-up as described by White et al. [32], in which further detail on the randomized control trial can be found. For this study, we have termed their first testing time point as the "baseline" time point because the focus of this analysis was not to investigate the influence of training on the outcomes of interest. Participants in the present study were divided into two groups, BPTB graft (*n* = 23) and other (*n* = 54) graft types (Table 1). The participants who received hamstring autografts (*n* = 37) or allografts (*n* = 17) were grouped into a single group for the purpose of this study because the primary objective was to compare the influence of graft harvest in the extensor mechanism on extensor latency during gait (see Supplemental Table 1, Supplemental Digital Content 1, Independent sample *t*-test between the involved knees of the hamstring autograft and allograft groups at the baseline time point, https://links.lww.com/MSS/C682).

Biomechanical testing

Gait biomechanics were assessed at both the baseline and the 2-yr time points. Gait kinetics and EMG were collected. Participants walked across a 10-m walkway over a built-in force plate (1080 Hz; Bertec Corporation, Columbus, OH) at self-selected gait speeds until five successful trials were obtained (Table 1). A successful trial was defined as a trial where the participant walked within 5% of their self-selected gait speed with no evidence of targeting or double striking of the force plate. Biomechanics data were collected using an eight-camera system (120 Hz; Vicon, Oxford, UK) and retroreflective markers placed on lower extremity joints and anatomical landmarks[20,21,32,35,38,39]. Force plate data were low-pass filtered at 25 Hz, and marker trajectories were low-pass filtered at 6 Hz. Knee joint kinetics were calculated using inverse dynamics with commercial software (Visual3D; C-Motion, Germantown, MD). Event markers were placed at initial contact, peak knee flexion moment (pKFM), and the time from initial contact to pKFM was reported as pKFM occurrence in milliseconds.

TABLE 1 - Participant characteristics at each time point by graft type.

Variables	**Baseline**		**2 yr**	
Graft Type	**BPTB (*n* = 21)**	**Other (*n* = 52)**	**BPTB (*n* = 19)**	**Other (*n* = 48)**
Age (yr)	19.3 ± 3.2	22.9 ± 8.8	19.9 ± 3.1	24.3 ± 7.8
Body mass index ($kg \cdot m^{-2}$)	26.1 ± 3.5	26.0 ± 3.3	26.6 ± 3.7	26.3 ± 3.1
Gait speed ($m \cdot s^{-1}$)	1.52 ± 0.1	1.54 ± 0.1	1.52 ± 0.1	1.54 ± 0.1
Time from surgery to gait analysis (wk)	28.8 ± 7.8	22.1 ± 7.3	109.2 ± 17.1	109.3 ± 11.7
Quadriceps strength index (%)	86.0 ± 4.6	93.1 ± 9.3	104.2 ± 13.7	100.5 ± 15.8
Sex (women:men)	13:8	22:30	13:6	19:29
Mechanism of injury (contact:noncontact)	4:17	25:27	3:16	22:26
Training group (secondary prevention:secondary prevention + perturbation)	10:11	25:27	10:9	23:25
Medial meniscal status (partial meniscectomy:meniscal repair:none) [a]	1:3:12	12:9:30	1:3:10	11:8:28
Lateral meniscal status (partial meniscectomy:meniscal repair:none) [a]	7:2:7	17:6:28	7:2:5	16:6:25

Data are presented as mean ±SD.

aMeniscal status does not have complete data due to missing operative reports.

EMG analysis and variables of interest

EMG electrodes were placed bilaterally on the three superficial quadriceps muscles, vastus medialis (VM), vastus lateralis (VL), and rectus femoris (RF) muscles[20,21,32,35] following the guidelines of the surface EMG for noninvasive assessment of muscles. EMG activity was normalized to maximum voluntary isometric contractions, and quadriceps EMG activity as a single extensor group was calculated using the mean activation between VM, VL, RF, and estimated VL activity (mean activation between VM and VL) as described by Ito et al., [28]. EMG signals were collected at 1080 Hz (MA-300 EMG System; Motion Lab Systems, Baton Rouge, LA), and data were high-pass filtered using a second-order Butterworth filter at 30 Hz, rectified, and low-pass filtered at 6 Hz to create a linear envelope. Peak extensor EMG activities were identified as the highest peak maintained over a 2.8-ms (three frames at 1080 Hz) window between initial contact and pKFM. The time values between initial contact and peak quadriceps, VM, VL, and RF activity were calculated and reported as Quad, VM, VL, and RF occurrence. The time values between time of peak Quad, VM, VL, and RF occurrence and pKFM occurrence were then calculated and reported as Quad, VM, VL, and RF latency, our primary variables of interest[28]. The average of five trials was calculated and reported. As a secondary purpose, extensor EMG occurrence and pKFM occurrence were analyzed separately to assess the underlying variable responsible for the difference in latencies observed between graft types (i.e., whether quadriceps are activating earlier or whether moment is produced later if a longer latency was observed).

Statistical analysis

A linear mixed-effects model using an unstructured covariance matrix was used to investigate the fixed effects of graft type (BPTB vs other), time point (baseline vs 2 yr), and the interaction between graft type and time point on the variables of interest (Quad, VM, VL, and RF latency) for the involved and uninvolved limbs separately (α = 0.05). Individual participants were included as random effects, and the model was adjusted for covariates, which may influence gait biomechanics and neuromuscular control (sex, training group, mechanism of injury, age, body mass index, time from surgery, and walking speed) as fixed effects. The interaction between training group and time was assessed to identify whether the training group that the participants were allocated in as part of the original randomized clinical trial influenced latency. EMG and pKFM occurrence were also evaluated in both limbs using the same statistical approach as a secondary purpose. Statistics were performed using R[40] (package: lme4 [41]). Demographic variables are presented for the complete sample (Table 1).

RESULTS

Involved limb extensor latency

Main effects of graft type were observed for VM latency (P = 0.005) and Quad latency (P = 0.033) after accounting for covariates. The BPTB graft group displayed longer latencies over the two time points compared with the other graft type group (Fig. 1, Table 2). The difference by graft type for VM latency (mean difference = 15.5 ms) also exceeded previously identified meaningful interlimb difference (11.7 ms)[28]. No main effects of graft type were observed for VL (P = 0.051) and RF latency (P = 0.080) (Fig. 1, Table 2). A main effect of time was observed only for RF latency (P = 0.022), characterized by shorter latency at the 2-yr time point regardless of graft type. No other interaction or main effects of time were observed (Table 2).

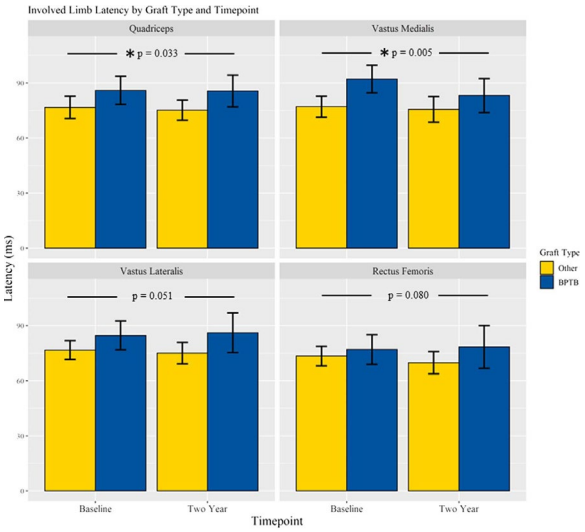

FIGURE 1: After adjusting for covariates, the main effect of graft type was observed for quadriceps and VM latency in the involved limb across both time points. The involved limb of the BPTB graft group had longer latency compared with the involved limb of the hamstring or allograft group collapsed across time points. A main effect of time was observed for RF latency characterized by shorter latency at the 2-yr time point compared with the baseline time point regardless of graft type. No other effects of time were observed. Error bars indicate 95% confidence intervals. *$P < 0.05$.

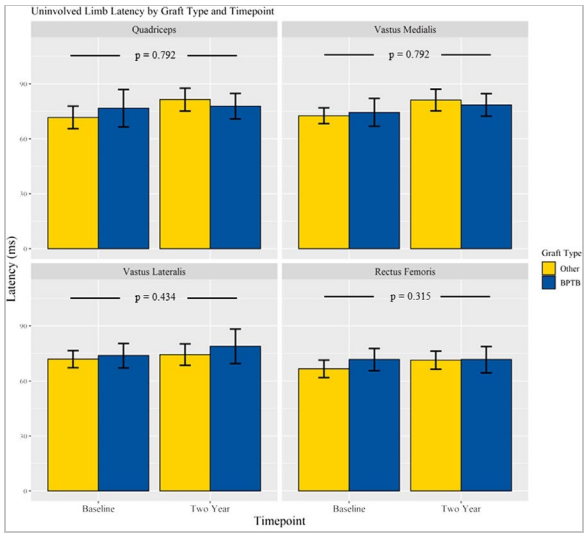

FIGURE 2: No main or interaction effects of time or graft type were observed in any of the latency values in the uninvolved limb. Error bars indicate 95% confidence intervals.

TABLE 2 - Involved limb latency.

	Quad Latency		**VM Latency**		**VL Latency**		**RF Latency**	
Variables of Interest	***F***	***P***	***F***	***P***	***F***	***P***	***F***	***P***
Sex (women:men)	0.428	0.516	1.463	0.230	0.151	0.699	3.349	0.071
Training group (secondary prevention|secondary prevention + perturbation)	1.081	0.303	1.844	0.179	0.596	0.443	1.554	0.217
Mechanism of injury (contact|noncontact)	3.727	0.058	1.467	0.230	3.553	0.063	3.933	0.051
Age (yr)	0.483	0.490	0.414	0.522	0.002	0.960	0.702	0.405
Body mass index (kg·m^{-2})	1.998	0.161	0.840	0.362	0.561	0.456	0.840	0.362
Time from surgery (wk)	0.084	0.772	0.107	0.744	1.527	0.219	6.201	0.014*
Walking speed (m·s^{-1})	0.554	0.460	0.702	0.405	0.004	0.948	0.260	0.612
Time point (baseline|2 yr)	0.114	0.736	0.000	0.993	1.445	0.232	5.444	0.022*
Graft type (BPTB|Other)	4.763	0.033*	8.425	0.005**	3.933	0.051	3.163	0.080
Time point × training group	2.549	0.116	0.965	0.329	2.365	0.129	0.086	0.771
Time point × graft type	0.176	0.676	0.831	0.365	0.442	0.508	2.707	0.105

The main effects of graft type were observed for Quad and VM latency characterized by a longer latency in the BPTB compared with the other graft type group. The main effect of time was also observed for RF latency characterized by shorter latency in the later time point regardless of graft type.

*$P < 0.05$.

**$P < 0.01$.

***$P < 0.001$.

Uninvolved limb extensor latency

No interaction or main effects of time or graft type were observed as expected for all four latencies (Table 3, Fig. 2).

TABLE 3 - Uninvolved limb latency.

	Quad Latency		**VM Latency**		**VL Latency**		**RF Latency**	
Variables of Interest	***F***	***P***	***F***	***P***	***F***	***P***	***F***	***P***
Sex (women:men)	0.034	0.854	0.065	0.800	0.031	0.862	1.708	0.195
Training group (secondary prevention|secondary prevention + perturbation)	0.000	0.998	2.131	0.149	0.826	0.367	0.056	0.813
Mechanism of injury (contact|noncontact)	0.581	0.449	0.740	0.393	0.658	0.420	0.000	0.992
Age (yr)	0.355	0.554	0.065	0.800	0.073	0.787	0.008	0.929
Body mass index (kg·m^{-2})	2.443	0.122	5.716	0.019*	1.415	0.237	0.028	0.867
Time from surgery (wk)	0.513	0.476	0.194	0.661	0.018	0.894	0.014	0.904
Walking speed (m·s^{-1})	3.637	0.062	4.411	0.039*	1.677	0.200	0.886	0.350

Time point (baseline|2 yr)	1.212	0.274	0.870	0.353	0.250	0.619	0.116	0.735
Graft type (BPTB|Other)	0.070	0.792	0.070	0.792	0.619	0.434	0.537	0.466
Time point × training group	0.819	0.369	4.808	0.032*	0.525	0.471	2.074	0.155
Time point × graft type	1.318	0.256	1.710	0.195	0.305	0.583	1.615	0.208

No effects of time or graft type were observed for variables in the uninvolved limb. Body mass index and walking speed were significant covariates for VM latency and a time point by training group interaction was also observed.:

*$P < 0.05$.

**$P < 0.01$.

***$P < 0.001$.

pKFM and extensor EMG occurrence

No interaction or main effects of time or graft type were observed for all occurrence measures in either limb (Tables 4 and 5).

TABLE 4 - Involved limb occurrence.

	Quad Occurrence		**VM Occurrence**		**VL Occurrence**		**RF Occurrence**		**pKFM Occurrence**	
Variables of Interest	***F***	***P***	***F***	***P***	***F***	***P***	***F***	***P***	***F***	***P***
Sex (women:men)	0.436	0.512	0.078	0.780	2.941	0.091	0.042	0.838	5.210	0.025*
Training group (secondary prevention|secondary prevention + perturbation)	0.262	0.611	0.905	0.345	0.112	0.739	1.976	0.164	0.188	0.666
Mechanism of injury (contact|noncontact)	3.188	0.079	0.980	0.325	2.458	0.121	2.934	0.091	0.025	0.874
Age (yr)	2.306	0.135	0.084	0.773	0.726	0.397	2.732	0.103	2.001	0.161
Body mass index ($kg \cdot m^{-2}$)	0.033	0.857	0.126	0.723	0.091	0.764	0.053	0.818	3.585	0.061
Time from surgery (wk)	0.019	0.890	0.149	0.700	0.713	0.401	5.724	0.019*	0.180	0.672
Walking speed ($m \cdot s^{-1}$)	2.094	0.154	7.599	0.008**	3.229	0.077	2.395	0.126	10.087	0.002**
Time point (baseline|2 yr)	0.169	0.682	0.128	0.722	0.227	0.635	3.559	0.062	1.015	0.316
Graft type (BPTB|Other)	1.027	0.315	2.763	0.101	0.597	0.442	0.391	0.534	2.414	0.124
Time point × training group	3.061	0.085	1.326	0.254	3.533	0.065	0.343	0.560	0.346	0.559
Time point × graft type	0.394	0.533	0.402	0.528	0.894	0.348	3.823	0.055	0.201	0.655

No effects of time or graft type were observed in occurrence measures:

*$P < 0.05$.

**$P < 0.01$.

TABLE 5 - Uninvolved limb occurrence.

	Quad Occurrence		**VM Occurrence**		**VL Occurrence**		**RF Occurrence**		**pKFM Occurrence**	
Variables of Interest	***F***	***P***	***F***	***P***	***F***	***P***	***F***	***P***	***F***	***P***
Sex (male|female)	0.301	0.585	1.865	0.176	0.964	0.329	0.002	0.961	3.277	0.074
Training group (secondary prevention|secondary prevention + perturbation)	0.052	0.820	0.890	0.349	0.262	0.610	0.168	0.683	0.175	0.677
Mechanism of injury (contact|noncontact)	1.204	0.277	1.394	0.242	1.437	0.234	0.187	0.667	0.549	0.461
Age (yr)	3.300	0.075	2.332	0.131	3.297	0.073	2.241	0.139	7.371	0.008**
Body mass index ($kg \cdot m^{-2}$)	0.308	0.580	0.776	0.380	0.003	0.955	1.173	0.281	3.819	0.053
Time from surgery (wk)	0.267	0.607	0.024	0.878	0.065	0.799	0.151	0.699	0.448	0.505
Walking speed ($m \cdot s^{-1}$)	0.107	0.744	0.512	0.477	1.281	0.262	2.227	0.140	14.515	<0.001***
Time point (baseline|2 yr)	1.303	0.257	0.304	0.583	0.138	0.711	0.012	0.915	0.052	0.821
Graft type (BPTB|Other)	0.365	0.548	1.510	0.223	0.039	0.843	0.093	0.762	2.274	0.136
Time point × training group	0.079	0.779	2.913	0.093	0.106	0.746	0.881	0.351	0.416	0.521
Time point × graft type	0.309	0.580	0.090	0.765	2.203	0.143	0.022	0.883	2.421	0.124

No effects of time or graft type were observed in occurrence measures.

*$P < 0.05$.

**$P < 0.01$.

***$P < 0.001$.

DISCUSSION

Extensor latency

The purpose of this study was to investigate the latency between peak quadriceps muscle activity and pKFM during gait, between athletes with BPTB grafts compared with other graft types, 5 ± 2 months and 2 yr after ACLR. After adjusting for confounding variables, our hypothesis that latencies would be longer in the involved limb of the BPTB graft group compared with the other graft type group was supported for the VM and quadriceps as a single muscle group. The difference between graft types for VL and RF latency was not significant. Our findings indicate that extensor latencies are longer after ACLR in athletes who received a BPTB graft compared with those with hamstring autograft or soft tissue allografts.

There were no improvements in latency for the two variables (VM and quad latency) that were different in latency at the baseline timepoint. The hypothesis was that these values will improve over time, but we did not observe that. After adjustments, RF latency shortened over time, regardless of the graft type. The findings for the RF may be attributed to the RF being a biarticular quadriceps muscle (i.e., knee extensor and hip flexor), which may have a more complex use than simply accepting weight early on during gait. As expected, we did not observe any effects of graft type or time in the uninvolved limb. These findings indicate that prolonged extensor latency from BPTB graft harvest and altered quadriceps neuromuscular function may persist 2 yr after ACLR.

To our knowledge, this study is the first to investigate extensor latencies during gait between graft types after ACLR. Although the clinical influence of prolonged extensor latencies during gait is currently being investigated, the latency values we collected were comparable with validated measures of electromechanical delay in previous work, which reported values in the 30- to 90-ms range[14,42]. Although direct comparisons cannot be made due to the different methods in quantifying neuromuscular function of the quadriceps, our results indicate conflicting evidence compared with a study by Georgoulis et. al.[30] that found no differences in electromechanical delay regardless of BPTB graft harvest. Our findings may differ due to several factors, primarily testing position and task. Georgoulis et al. tested individuals seated at 90° of knee flexion, using an isometric contraction against a dynamometer. The patellar tendon is taut in this position compared with our testing position throughout weight acceptance in gait (approximately 0° to 20° of knee flexion). Electromechanical delay in the quadriceps is shorter when the knee is positioned further into flexion[17], and the pretensioned tendon may have masked the contribution of the latency from the series elastic component of the tendon, which needs to be stretched enough before muscle forces can be transferred through the tendon[43]. Additionally, ACL injuries are most prevalent in knee angles closer to extension[44,45], which is similar to the angles where we found prolonged extensor latency after BPTB graft harvest. This may indicate that quadriceps neuromuscular function during high-risk athletic maneuvers (cutting, jumping, landing, etc.) may be diminished for at least 2 yr after BPTB graft harvest, although further research is needed. Quantifying extensor latencies may help further understand the influence of BPTB graft harvest on gait mechanics, sport performance, and secondary injury risks.

Quadriceps occurrence and pKFM occurrence

There were null statistical findings between graft types for the secondary purpose examining timing of peak quadriceps activity and knee flexion moment. This may indicate that the longer latency observed in the involved limb of the BPTB graft group is mediated through a combination of late pKFM and early peak quadriceps activity. Alterations in gait mechanics due to quadriceps weakness after ACLR have been reported previously[46]. To compensate for an ACL injury with potential muscle weakness, high muscle cocontraction may be a gait adaptation strategy to maintain a near normal joint loading response[47]. Similarly, for patients after ACLR, a possible explanation for the differences in latency between graft types is a compensation by an upregulation in the central nervous system (i.e., early quadriceps activity) combined with a resultant delay

in moment production, hence the null findings with our current sample size in this study.
Early quadriceps activity may be a result of the neuromuscular system trying to synchronize quadriceps forces with external moments, while accounting for the diminished ability of the patellar tendon to transfer forces across the knee joint. Similar findings have been seen in the tendinopathy literature, where prolonged electromechanical delay, reflex times, and an upregulation of the central nervous system during a drop jump task were observed in the triceps surae muscle[16]. The clinical implications of the timing of quadriceps activity require further investigation; however, our findings may suggest a link between quadriceps neuromuscular function with the development of PTOA. Altered quadriceps neuromuscular function, especially in those who receive BPTB grafts, may influence gait mechanics and subsequent joint loading patterns associated with PTOA development[8–10].

Patients after ACLR using a BPTB autograft have been reported to walk with lower knee flexion angles and moments[8,26,27], and the cohort in this study presented with differences in sagittal plane mechanics between graft types consistent with the literature (see Supplemental Table 2, Supplemental Digital Content 2, Independent sample t-test between the involved knees of the 2 groups at the baseline time point, https://links.lww.com/MSS/C683). The group differences may be a consequence of the prolonged extensor latency we observed in the BPTB graft cohort. Altered activation patterns and temporal characteristics of gait mechanics must be considered to fully comprehend the underlying sources of aberrant gait biomechanics associated with OA. Long-term follow-up, including radiographic evidence, is necessary to quantify the implications of altered quadriceps activation patterns and temporal characteristics of gait mechanics after BPTB graft harvest.

Graft-specific rehabilitation

Currently, there are no established graft-specific rehabilitation protocols that target the altered neuromechanical properties of a tendon after graft harvest. Anterior knee pain[4,5] and prolonged quadriceps weakness[6,7] are common morbidities after BPTB graft harvest. Both factors may potentially alter neuromuscular function of the quadriceps during the early stages of rehabilitation. The findings in this study, however, show that extensor latency did not improve at 2 yr after ACLR, when both pain[6] and quadriceps strength asymmetry were resolved (Table 1). Altered mechanical properties of the patellar tendon long term may explain the lack of quadriceps latency improvement, which persists even after athletes have returned to sport, as all participants who completed 2-yr testing had returned to sport by that time[6,36,48].

There was no difference in the timing of pKFM occurrence between graft types, whereas latency was longer in the BPTB graft group, suggesting a potential upregulation of the central nervous system, resulting in minimally altered timing of pKFM. ACL injuries commonly occur within the first 30 to 40 ms from landing[45]. If prolonged extensor latency is also present among different functional tasks after ACLR, the 15-ms longer latencies we observed in the BPTB group (Table 6) may factor into the difference between a second injury and a safe landing in these athletes. A similarly diminished knee stabilization mechanism may also be observed in the semitendinosus muscle after hamstring autograft harvest, which may be a component of why reinjury rates continue to be high after ACLR regardless of autograft choice. Further investigation of extensor latency during high-risk maneuvers, however, is necessary to quantify such associations.

TABLE 6 - Latency and occurrence measures by graft type and time point.

Variables	Baseline		2 yr	
Group	BPTB (*n* = 21)	Other (*n* = 52)	BPTB (*n* = 19)	Other (*n* = 48)
Involved limb				
Quad latency (ms)	86.0 ± 18.9	76.7 ± 22.6	85.7 ± 21.2	75.2 ± 20.5
RF latency (ms)	77.0 ± 19.7	73.4 ± 20.0	78.4 ± 28.6	69.8 ± 22.6

VL latency (ms)	84.6 ± 19.3	76.7 ± 19.3	86.0 ± 26.4	74.9 ± 22.1
VM latency (ms)	92.1 ± 18.5	77.0 ± 21.5	83.2 ± 22.7	75.6 ± 25.9
pKFM occurrence (ms)	142.1 ± 13.9	137.5 ± 17.2	138.0 ± 16.6	134.6 ± 15.1
Quad occurrence (ms)	56.3 ± 21.4	61.0 ± 26.2	52.9 ± 24.4	59.9 ± 24.1
RF occurrence (ms)	65.1 ± 24.3	64.1 ± 20.6	59.6 ± 29.7	64.8 ± 24.9
VL occurrence (ms)	57.5 ± 22.3	60.8 ± 23.7	51.9 ± 28.2	59.6 ± 27.3
VM occurrence (ms)	50.0 ± 21.2	60.4 ± 24.8	54.8 ± 21.5	59 ± 27.8
Uninvolved limb				
Quad latency (ms)	76.7 ± 24.9	71.7 ± 23	77.8 ± 17.1	81.4 ± 23.6
RF latency (ms)	71.6 ± 14.9	66.6 ± 17.8	71.6 ± 17.6	71.3 ± 18.8
VL latency (ms)	73.8 ± 16.3	71.9 ± 17.7	78.9 ± 23.2	74.3 ± 22.0
VM latency (ms)	74.4 ± 18.6	72.6 ± 16.1	78.5 ± 15.1	81.2 ± 22.2
pKFM occurrence (ms)	141.4 ± 15.4	135.5 ± 15.2	136.2 ± 16.1	138.0 ± 13.6
Quad occurrence (ms)	65.9 ± 29.2	65.3 ± 23.5	54.5 ± 24.7	61.0 ± 23.9
RF occurrence (ms)	69.8 ± 22.7	68.9 ± 22.3	64.9 ± 23.6	66.4 ± 23.4
VL occurrence (ms)	67.6 ± 23.2	63.6 ± 20.0	61.9 ± 23.2	59.2 ± 29.4
VM occurrence (ms)	67.0 ± 23.1	63.0 ± 20.2	55.0 ± 22.9	59.5 ± 19.1

Data are presented as mean ±SD.

Graft-specific rehabilitation may be necessary to improve outcomes after ACLR. Developing graft-specific rehabilitation will require investigation of the efficacy of interventions involving neuromuscular control to promote central nervous system upregulations to account for prolonged latency. Additionally, a specific tendon loading protocol may be necessary to promote and accelerate tendon healing to improve the graft site tendon's ability to efficiently transfer forces[49]. The healing process within the graft site tendon's morphology (cross-sectional area, thickness, etc.) suggests that poor tendon quality may persist up to 6 yr after graft harvest[11,30,50,51]. The tendon's mechanical properties responsible for force transmission is also altered long term in both the hamstring[11] and patellar tendons[12,13] after graft harvest. Longitudinal studies assessing mechanical properties and morphology of the graft site tendon are needed to establish a better baseline understanding of the contribution of the patellar tendon to knee function after graft harvest.

Limitations

The cohort in this study cleared stringent inclusion criteria before enrollment. The participants in this study are the highest-level athletes with desires to return to sport, indicating that the generalizability to the everyday athlete or levels 3 and 4 athletes may not be perfect. It is, however, expected that the patients who do not clear our inclusion criteria may present with larger deficits than presented in this study. Future research is indicated to further understand the generalizability of our findings in a more heterogeneous population. Graft harvest techniques and physical therapy treatment received before enrollment were not controlled because surgery was not performed by a single surgeon, and rehabilitation before enrollment varied between each participant. Tendon structure at the time of enrollment may have been variable within the BPTB graft group, which may have altered the extensor latency values.

CONCLUSIONS

Prolonged VM and quadriceps latency is present in athletes 2 yr after ACLR with a BPTB graft compared with those who receive a hamstring autograft or soft tissue allograft. The graft site tendon's force transferring ability may be altered long term. Interventions to promote and accelerate healthy patellar tendon healing along with neuromuscular control exercises to compensate for the prolonged latency in the quadriceps muscles may be necessary. This study provides evidence to support the need for further understanding the influence of

the patellar tendon graft on quadriceps function and consider the need of a graft-specific rehabilitation protocol after ACLR using BPTB grafts.

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