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Abstract

Purpose

Asymmetrical gait mechanics after anterior cruciate ligament reconstruction (ACLR) are associated with the development of posttraumatic knee osteoarthritis. Current measures of gait mechanics have focused heavily on peak magnitudes of knee kinematics, kinetics, and joint contact forces but have seldom considered the rate of knee loading, cumulative knee load, or the timing of motor input surrounding peaks. The purpose of this study was to introduce and describe novel metrics of gait using temporal characteristics of kinetics and EMG to identify neuromuscular deficits of the quadriceps in patients after ACLR.

Methods

Gait mechanics were assessed 6 months (n = 145) and 24 months (n = 116) after ACLR. External knee flexion rate of moment development (RMD) and knee flexion moment impulse (KFMI) leading up to the time of peak knee flexion moment (pKFM), peak RMD between initial contact to pKFM, and cumulative KFMI were calculated. Extensor latencies from the quadriceps, vastus medialis, vastus lateralis, and rectus femoris (time of pKFM – time of peak EMG activity) during the weight acceptance phase of gait were also calculated. Paired-sample t-tests (α = 0.05) were performed between limbs at both time points.

Results

Slower RMD, smaller KFMI, and longer extensor latencies in the involved compared with uninvolved limb were observed across all measures at 6 months ($P < 0.005$). At 24 months, RMD_{peak} was slower, and $KFMI_{50ms}$, $KFMI_{100ms}$, and $KFMI_{total}$ were lower in the involved limb ($P < 0.003$), but no other asymmetries were found.

Conclusions

Slower RMD, smaller KFMI, and prolonged extensor latencies may characterize neuromuscular deficits underlying aberrant gait mechanics early after ACLR. RMD, KFMI, and extensor latencies during gait should be considered in the future to quantify asymmetrical movement patterns observed after ACLR and as markers of recovery.

Anterior cruciate ligament (ACL) injury and ACL reconstruction (ACLR) increases the risk of developing posttraumatic knee osteoarthritis (OA) ([1–3]). Recent evidence suggests that inadequate loading of the involved knee during the first 50% of the stance phase in gait may contribute to OA development ([4–7]). Studies have focused heavily on asymmetries in the peak magnitude of gait kinetics, kinematics, and joint contact forces to identify pathological gait after ACLR ([8–13]). The temporal characteristics of knee kinetics and EMG leading up to the peaks, such as the knee flexion rate of moment development (RMD), knee flexion moment impulse (KFMI) ([14,15]), or timing of motor input from the quadriceps relative to

knee flexion moment, however, have not been rigorously evaluated. This article introduces novel approaches to identify pathological gait mechanics during the weight acceptance phase of gait through evaluating the temporal characteristics of knee kinetics and EMG data from the quadriceps muscles in patients after ACLR.

Articular cartilages in the knee (both tibiofemoral and patellofemoral joints) are biphasic materials compromised by both fluids and collagen matrix, meaning their response to load is viscoelastic in nature, or rate dependent ([16]). Although studying the magnitude of the peak knee joint moment or contact force may detect the asymmetries in instantaneous load applied through the knee joint, the context surrounding how the load reached that point, such as how long the joint was under load, or how quickly the joint was loaded, cannot be explained. Slow gait speed after ACLR is associated with biomarkers of collagen breakdown ([17]), and magnetic resonance imaging confirmed that trochlear cartilage degeneration has also been observed in those who walked slower after ACLR ([18]). In the later study, although gait speed was associated with an indicator of early OA (i.e., T2 relaxation time), peak knee kinetic and kinematic measures were not. Part of the explanation for these findings may be due to peak kinetic and kinematic measures of gait not capturing the context surrounding the way the articular surfaces in the knee joint were loaded leading up to the peaks (i.e., RMD or KFMI during weight acceptance). In addition to our current knowledge on the peak magnitude of knee gait mechanics, quantifying the timing, rate, and motor input that results in the altered peak knee gait mechanics can give us a better understanding of the context surrounding the asymmetries in gait mechanics after ACLR.

Quadriceps function is diminished after ACLR and plays a key role in both the magnitude and timing of knee gait mechanics ([19,20]). Altered function is likely because external knee flexion moment is primarily balanced by quadriceps forces during the stance phase of gait. Because of the commonly observed quadriceps inhibition ([21]), decreased corticospinal excitability ([22,23]), and increased electromechanical delay ([24]), patients after ACLR may walk with greater temporal mismatch between their motor inputs from the quadriceps and joint kinetics in their involved knees. Measuring the temporal characteristics and calculating the latency between quadriceps EMG activity and external knee flexion moment during gait will allow clinicians and researchers to quantify a patient's neuromuscular capabilities after ACLR. Measures of this latency may be used as a tool to identify individual patients and patient subgroups (graft type, sex, age, activity level, etc.) with greater levels of neuromuscular control deficits to target future interventions. This study aimed 1) to introduce and describe thoroughly novel metrics of gait by calculating sagittal plane knee RMD, KFMI, and temporal disagreement between quadriceps EMG and kinetic outputs and 2) to investigate how the novel metrics present between the involved and the uninvolved limbs 6 and 24 months after ACLR.

METHODS

Participants in this study were enrolled in one of three IRB-approved studies at the University of Delaware between February 2006 and November 2020. All gait data were collected and analyzed using the same methodology (described below). Given the novel and explorative nature of the clinical question, cross-sectional data from three longitudinal cohort studies were combined in this analysis to emphasize the content validity of this investigation by capturing an inclusive cohort of those who undergo ACLR (e.g., high level athletes to nonathletes) and maximizing sample size for statistical power. All participants sustained primary unilateral ACL injury before receiving ACLR, were between ages 13 and 55 yr, and had not sustained a secondary injury before either one of their gait data collections.

Participants were excluded if they had sustained an osteochondral defect greater than 1 cm^2 or any combined grade 3 ligament injury identified at surgery (Table 1). Informed consent was obtained from all participants.

TABLE 1 - Participant demographics at both time points.

Variables	**6 Month (*N* = 145)**	**24 Month (*N* = 116)**
	Mean ± SD or Distribution	Mean ± SD or Distribution
Height (m)	1.73 ± 0.09	1.73 ± 0.09
Weight (kg)	80.0 ± 14.8	81.9 ± 15.6
BMI	26.6 ± 3.6	27.1 ± 3.8
Age (yr)	24.3 ± 9.1	25.7 ± 8.8
Gait speed ($m \cdot s^{-1}$)	1.56 ± 0.13	1.56 ± 0.12
KOS-ADLS (%)	93.4 ± 7.2 [a]	96.5 ± 5.2 [b]
IKDC (%)	81.2 ± 10.8 [c]	93.7 ± 10.1 [b]
Sex (%)	43% women, 57% men	39% women, 61% men
Mechanism of injury	81 noncontact, 46 contact, 18 unknown	70 noncontact, 33 contact, 13 unknown
Graft type	47 allograft, 56 hamstring, 39 bone-patellar tendon-bone, 3 unknown	36 allograft, 48 hamstring, 27 bone-patellar tendon-bone, 5 unknown

[a]N = 143 due to missing data.
[b]N = 113 due to missing data.
[c]N = 131 due to missing data.
BMI, body mass index; KOS-ADLS, Knee Outcome Survey Activities of Daily Living Scale; IKDC, International Knee Documentation Committee.

Motion capture data collection and analyses

Motion capture gait analyses were performed at 5.9 ± 1.4 and 24.7 ± 2.3 months after ACLR. Data from 145 participants at the 6-month time point and 116 participants at the 24-month time point were included in this analysis. One hundred and six participants had longitudinal data captured and included at both time points. Gait kinematic, kinetic, and EMG data were collected at each time point. Participants were asked to walk across a 10-m walkway over a built-in force plate (Bertec Corporation, Columbus, OH), at self-selected gait speeds (Table 1). Trials recorded at speeds within 5% of their self-selected gait speeds ([9,10,13,25,26]) were included for analysis. Retroreflective markers were placed on the pelvis, lower extremity joints, and anatomical landmarks, as described in our previous work ([27]). An eight-camera system (Vicon, Oxford, UK) tracking 39 markers was used to collect kinematic data at 120 Hz. Force plate data were collected at 1080 Hz with initial contact and terminal stance determined using a 20-N threshold. External knee flexion moment was calculated throughout the weight acceptance phase of gait, using a commercial software (Visual3D, C-Motion, Germantown, MD), via inverse dynamics and normalized to body mass and height ($N \cdot kg^{-1}$). Event markers for initial contact, terminal stance, peak knee flexion angle (pKFA), and peak knee flexion moment (pKFM) were created, and the time from initial contact to terminal stance was determined as stance time, the time from initial contact to pKFA as pKFA occurrence, and the time from initial contact to pKFM as pKFM occurrence, all reported in milliseconds (Fig. 1).

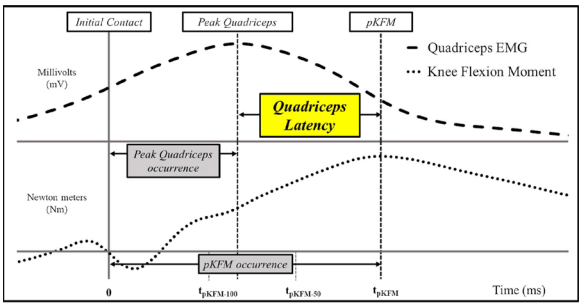

FIGURE 1: Linear envelope of quadriceps EMG curve (top) and knee flexion moment curve (bottom) plotted against time (ms) during the weight acceptance phase of gait. The waveforms in this graph are from a representative trial (scaled to fit the axes) used in this analysis. The *y*-axis represents the magnitude of each signal scaled appropriately to fit the figure. On the *x*-axis: initial contact = 0 ms. $t_{pKFM-100}$ = 100 ms before pKFM, $t_{pKFM-50}$ = 50 ms before pKFM, t_{pKFM} = time of pKFM.
Peak quadriceps occurrence = time from initial contact to peak quadriceps activity. pKFM occurrence = time from initial contact to pKFM. Quadriceps latency = pKFM occurrence − quadriceps occurrence.
*VM, VL, and RF EMG curves take similar shapes.

RMD

Sagittal plane RMD leading up to pKFM during the stance phase of gait was calculated by taking the slope of the knee flexion moment curve against time in seconds and reported in Newtons per kilogram per seconds ($N \cdot kg^{-1} \cdot s^{-1}$). The peak instantaneous RMD (RMD_{peak}) was determined as the steepest identified slope between initial contact and pKFM. Average RMD during the last 50 ms (RMD_{50ms}) and between 50 and 100 ms (RMD_{100ms}) before pKFM were also calculated (Fig. 1).

KFMI

KFMI was calculated by taking the area under the knee flexion moment curve and reported in newton milliseconds per kilogram ($N \cdot ms \cdot kg^{-1}$). Intervals of interest were similar with RMD and taken during the last 50 ms leading up to pKFM ($KFMI_{50ms}$) and between 50 and 100 ms ($KFMI_{100ms}$) before pKFM (Fig. 1). The cumulative positive KFMI ($KFMI_{total}$) during the weight acceptance phase of gait was also calculated by taking the area under the curve between the time of pKFM and the time of *x*-intercept of the knee flexion moment curve. In the case where multiple *x*-intercepts were found between initial contact and pKFM, the intercept closest to pKFM was chosen to determine the intervals.

EMG analysis and extensor latency

EMG electrodes were placed bilaterally on the vastus medialis (VM), vastus lateralis (VL), and rectus femoris (RF) muscles, after shaving and abrading the skin using alcohol and gauze to lower skin impedance and improve electrode adhesion. Signals were collected at 1080 Hz (MA-300 EMG System; Motion Lab Systems, Baton Rouge, LA), and data were high-pass filtered (2^{nd} order Butterworth, 30 Hz), rectified, and low-pass filtered (6 Hz) to create a linear envelope and normalized to their maximum voluntary isometric contractions (MVIC). MVIC-normalized EMG data from the three superficial quadriceps were then used to estimate the quadriceps activity as a single knee extensor muscle group (EMG_{Quad}) for each time point throughout the stance phase of gait using the following formula:

$$\mathrm{EMG_{Quad}(\%MVIC)} = \frac{\mathrm{EMG_{VM}} + \mathrm{EMG_{VL}} + \mathrm{EMG_{RF}} + \mathrm{EMG_{VI}}}{4},$$

where EMG_{VM} is the vastus medialis EMG, EMG_{VL} is the vastus lateralis EMG, EMG_{RF} is the rectus femoris EMG, and EMG_{VI} is the vastus intermedius EMG, all calculated as a percent of their MVIC. EMG_{VI} was estimated using the mean activity between the EMG_{VM} and the EMG_{VL} as described in previous musculoskeletal models of the knee joint during gait ([28]). Peak quadriceps, VM, VL, and RF EMG activity were determined using the highest peak maintained over a 2.8-ms (three frames at 1080 Hz) window between initial contact and pKFM. The time from initial contact to peak quadriceps, VM, VL, and RF activity were calculated and reported as peak quadriceps, VM, VL, and RF occurrence in milliseconds (Fig. 1). If a second peak was observed after initial contact, the peak with the higher magnitude was chosen to represent peak quadriceps, VM, VL, and RF activity. We also considered peak EMG activity before initial contact in the rare case (1.9% of all trials) where no peaks were found after initial contact (i.e., downward slope from initial contact to pKFM). The temporal agreement between the motor input and the kinetic output was calculated by measuring the time between peak quadriceps, VM, VL and RF occurrence and pKFM occurrence to quantify neuromuscular function of the quadriceps. The outputs from this measure were reported as quadriceps, VM, VL, and RF latency (Fig. 1). An average of five trials for each variable, per limb were calculated and reported for each participant. All temporal calculations were performed using custom scripts in Visual3D.

Statistical analysis

Separate paired samples *t*-tests ($\alpha = 0.05$) were run between the involved and the uninvolved limbs at 6- and 24-month time points for our primary variables of interest: RMD_{peak}, RMD_{50ms}, RMD_{100ms}, $KFMI_{50ms}$, $KFMI_{100ms}$, $KFMI_{total}$, quadriceps latency, VM latency, VL latency, and RF latency. Statistically significant findings were also compared with Bonferroni adjusted α values ($\alpha = 0.05/10 = 0.005$) to account for multiple comparisons. Asymmetries were also compared against meaningful interlimb differences (MILD; see Supplemental Table 1, Supplemental Digital Content, Inter-class correlations (ICC) and meaningful interlimb difference [MILD] values, https://links.lww.com/MSS/C513) calculated using established methodology ([5,29]). Seven secondary variables of interest (stance time, pKFA occurrence, pKFM occurrence, peak quadriceps occurrence, peak VM occurrence, peak VL occurrence, and peak RF occurrence) were also compared using the same statistical methods. Statistical analyses were performed using SPSS version 25.0 (IBM Corporation, Armonk, New York). Analyses comparing variables of interest over time were not performed because the purpose of this study was to establish means and asymmetries at each time point while maximizing sample size. *A priori* power analysis was not performed given the explorative nature of the study.

RESULTS

RMD

RMD_{peak}, RMD_{50ms}, and RMD_{100ms} all demonstrated statistically significant differences ($P < 0.001$) between limbs at the 6-month time point characterized by slower RMD in the involved versus uninvolved limbs at each time interval (Table 2). At the 24-month time point, RMD_{peak} was the only RMD variable that presented with a statistically significant difference between limbs ($P < 0.001$) characterized by slower RMD_{peak} in the involved limbs, whereas RMD_{50ms} and RMD_{100ms} were not different between limbs (Table 3). No asymmetries exceeded MILD (see Supplemental Table 1, Supplemental Digital

Content, Inter-class correlations (ICC) and meaningful interlimb difference [MILD] values, https://links.lww.com/MSS/C513).

TABLE 2 - Statistically significant differences between limbs characterized by slower RMD and smaller KFMI were observed in all three time intervals for each parameter at the 6-month time point.

Variables	**Side**	**Mean ± SD**	**Mean Difference (95% CI)**	**Significance (*P*)**	**Effect Size (Cohen's *d*)**
RMD peak ($N \cdot kg^{-1} \cdot s^{-1}$)	IN	15.60 ± 4.89	−2.16 (−2.79 to −1.53)	<0.001**	−0.560 [a]
	UN	17.76 ± 5.58			
RMD 50 ms ($N \cdot kg^{-1} \cdot s^{-1}$)	IN	3.14 ± 1.20	−0.39 (−0.59 to −0.20)	<0.001**	−0.326 [b]
	UN	3.54 ± 1.20			
RMD 100 ms ($N \cdot kg^{-1} \cdot s^{-1}$)	IN	6.02 ± 2.42	−1.29 (−1.63 to −0.94)	<0.001**	−0.612 [a]
	UN	7.31 ± 2.69			
KFMI 50 ms ($N \cdot ms \cdot kg^{-1}$)	IN	19.19 ± 7.91	−5.47 (−6.70 to −4.24) [c]	<0.001**	−0.729 [a]
	UN	24.66 ± 8.15			
KFMI 100 ms ($N \cdot ms \cdot kg^{-1}$)	IN	2.65 ± 4.10	−2.09 (−2.72 to −1.46) [c]	<0.001**	−0.544 [a]
	UN	4.74 ± 4.23			
KFMI Total ($N \cdot ms \cdot kg^{-1}$)	IN	23.58 ± 10.11	−7.27 (−8.90 to −5.63) [c]	<0.001**	−0.729 [a]
	UN	30.85 ± 10.83			

Mean differences in all three impulse measures exceeded meaningful interlimb difference (MILD) (Supplemental Table 1, Supplemental Digital Content, https://links.lww.com/MSS/C513) (n = 145).
*P < 0.05.
**P < 0.005 (Bonferroni adjusted α = 0.05/10 = 0.005).
[a]Medium effect size >0.5.
[b]Small effect size >0.2.
[c]Exceeded meaningful interlimb difference.
95% CI, 95% confidence interval; IN, involved limb; UN, uninvolved limb.

TABLE 3 - Statistically significant differences between limbs were observed for RMD_{peak} characterized by slower loading in the involved vs uninvolved limbs, and KFMI in all three time intervals characterized by smaller impulse at the 24-month time point.

Variables	**Side**	**Mean ± SD**	**Mean Difference (95% CI)**	**Significance (*P*)**	**Effect Size (Cohen's *d*)**
RMD peak ($N \cdot kg^{-1} \cdot s^{-1}$)	IN	16.33 ± 4.78	−1.38 (−1.96 to −0.80)	<0.001**	−0.435 [a]
	UN	17.71 ± 5.16			
RMD 50 ms ($N \cdot kg^{-1} \cdot s^{-1}$)	IN	3.31 ± 1.23	0.00 (−0.18 to 0.18)	0.893	−0.004
	UN	3.31 ± 1.10			
RMD 100 ms ($N \cdot kg^{-1} \cdot s^{-1}$)	IN	6.84 ± 2.47	−0.33 (−0.67 to 0.02)	0.051	−0.175
	UN	7.16 ± 2.68			
KFMI 50 ms ($N \cdot ms \cdot kg^{-1}$)	IN	22.89 ± 7.79	−1.57 (−2.61 to −0.53)	0.003**	−0.280 [a]
	UN	24.46 ± 7.50			

KFMI 100 ms ($N \cdot ms \cdot kg^{-1}$)	IN	4.08 ± 4.62	−1.05 (−1.72 to −0.38)	0.002**	−0.288 [a]
	UN	5.12 ± 4.56			
KFMI total ($N \cdot ms \cdot kg^{-1}$)	IN	28.46 ± 10.70	−2.54 (−4.08 to −1.00)	0.001**	−0.304 [a]
	UN	31.00 ± 10.52			

No statistically significant findings were observed for RMD_{50ms} and RMD_{100ms} (n = 116).
*P < 0.05.
**P < 0.005 (Bonferroni adjusted α = 0.05/10 = 0.005).
[a]Small effect size >0.2.
95% CI, 95% confidence interval; IN, involved limb; UN, uninvolved limb.

KFMI

$KFMI_{50ms}$, $KFMI_{100ms}$, and $KFMI_{total}$ all demonstrated statistically significant differences between limbs characterized by smaller impulse in the involved versus uninvolved limbs at both the 6- and the 24-month time points (P < 0.003) (Tables 2 and 3). Mean differences at the 6-month time point exceeded MILD values (see Supplemental Table 1, Supplemental Digital Content, Inter-class correlations (ICC) and meaningful interlimb difference [MILD] values, https://links.lww.com/MSS/C513) for all three variables; however, at the 24-month time point, none of the mean differences reached MILD values.

Extensor latency

Quadriceps latency, VM latency, VL latency, and RF latency all demonstrated statistically significant differences (P < 0.005) between limbs at the 6-month time point characterized by longer latency in the involved versus uninvolved limbs (Table 4). At the 24-month time point, none of the latency measures demonstrated statistically significant differences between limbs (Table 5). No asymmetries exceeded MILD (see Supplemental Table 1, Supplemental Digital Content, Inter-class correlations (ICC) and meaningful interlimb difference [MILD] values, https://links.lww.com/MSS/C513).

TABLE 4 - Statistically significant differences characterized by longer latency in the involved vs uninvolved limbs for all four latency measures were observed at the 6-month time point (n = 145).

Variables	**Side**	**Mean ± SD**	**Mean Difference (95% CI)**	**Significance (P)**	**Effect Size (Cohen's d)**
Quadriceps latency (ms)	IN	85.4 ± 24.2	6.79 (2.66–10.93)	0.002**	0.270 [a]
	UN	78.7 ± 24.2			
VM latency (ms)	IN	85.7 ± 26.0	7.40 (2.30–12.49)	0.005**	0.238 [a]
	UN	78.3 ± 24.8			
VL latency (ms)	IN	85.7 ± 23.4	7.17 (2.93–11.41)	0.001**	0.277 [a]
	UN	78.5 ± 23.0			
RF latency (ms)	IN	78.6 ± 24.7	9.63 (5.22–14.05)	<0.001**	0.358 [a]
	UN	68.9 ± 20.2			

*P < 0.05.
**P < 0.005 (Bonferroni adjusted α = 0.05/10 = 0.005).
[a]Small effect size >0.2.
95% CI, 95% confidence interval; IN, involved limb; UN, uninvolved limb.

TABLE 5 - No statistically significant differences were observed between limbs for all four latency measures at the 24-month time point (n = 116).

Variables	Side	Mean ± SD	Mean Difference (95% CI)	Significance (P)	Effect Size (Cohen's d)
Quadriceps Latency (ms)	IN	82.9 ± 22.9	2.20 (−2.20 to 6.95)	0.360	0.085
	UN	80.7 ± 22.7			
VM latency (ms)	IN	84.7 ± 25.6	0.78 (−3.88 to 5.44)	0.741	0.031
	UN	83.9 ± 21.8			
VL latency (ms)	IN	84.3 ± 22.7	3.36 (−1.11 to 7.83)	0.140	0.138
	UN	81.0 ± 23.6			
RF latency (ms)	IN	77.5 ± 23.3	3.26 (−1.68 to 8.21)	0.194	0.121
	UN	74.3 ± 22.8			

95% CI, 95% confidence interval; IN, involved limb; UN, uninvolved limb.

Secondary variables of interest

Stance time, pKFM occurrence, and peak quadriceps, VM, VL, and RF occurrences demonstrated statistically significant differences between limbs at the 6-month time point ($P < 0.05$). Stance time was shorter, pKFM occurrence happened later, and peak quadriceps occurrences happened earlier in the involved versus uninvolved limbs (Table 6). At the 24-month time point, stance time and pKFA occurrence demonstrated statistically significant differences between limbs (P = 0.016 and 0.014) characterized by longer stance time and earlier pKFA occurrence in the involved versus uninvolved limbs. Neither of these differences at 24 months, however, were statistically significant after Bonferroni corrections for multiple comparisons ($\alpha = 0.05/7 = 0.007$) (Table 7).

TABLE 6 - Statistically significant differences characterized by shorter stance time, later pKFM occurrence, and earlier peak quadriceps, VM, VL, and RF occurrences in the involved vs uninvolved limbs were observed at the 6-month time point.

Variables	Side	Mean ± SD	Mean Difference (95% CI)	Significance (P)	Effect Size (Cohen's d)
Stance time (ms)	IN	634.8 ± 47.4	−7.28 (−9.31 to −5.25)	<0.001**	−0.590 [a]
	UN	642.1 ± 43.7			
pKFA occurence (ms)	IN	142.9 ± 17.2	0.11 (−1.79 to 2.01)	0.908	0.010
	UN	142.7 ± 15.5			
pKFM occurrence (ms)	IN	140.7 ± 16.7	2.40 (0.76 to 4.04)	0.004**	0.241 [b]
	UN	138.3 ± 14.5			
Peak quadriceps occurrence (ms)	IN	55.2 ± 25.2	−4.46 (−8.29 to −0.62)	0.023*	−0.191
	UN	59.7 ± 25.0			
Peak VM occurrence (ms)	IN	55.0 ± 28.1	−4.99 (−9.98 to −0.01)	0.050*	−0.164
	UN	60.0 ± 26.9			

Peak VL occurrence (ms)	IN	55.0 ± 25.5	−4.77 (8.89 to −0.65)	0.024*	−0.190
	UN	59.8 ± 23.6			
Peak RF occurrence (ms)	IN	62.1 ± 26.3	−7.23 (−11.18 to −3.28)	<0.001**	−0.300 [b]
	UN	69.4 ± 23.5			

No statistically significant findings were observed for pKFA occurrence (*n* = 145).
**P* < 0.05.
***P* < 0.007 (Bonferroni adjusted α = 0.05/7 = 0.007).
[a]Medium effect size >0.5.
[b]Small effect size >0.2.
95% CI, 95% confidence interval; IN, involved limb; UN, uninvolved limb; pKFA, peak knee flexion angle.

TABLE 7 - Statistically significant differences characterized by shorter stance time and later pKFA occurrence in the involved vs uninvolved limbs were observed at the 24-month time point.

Variables	**Side**	**Mean ± SD**	**Mean Difference (95% CI)**	**Significance (*P*)**	**Effect Size (Cohen's *d*)**
Stance time (ms)	IN	635.9 ± 47.0	−2.47 (−4.48 to −0.47)	0.016*	−0.227 [a]
	UN	638.4 ± 44.5			
pKFA occurence (ms)	IN	138.6 ± 16.9	−2.29 (−4.11 to −0.47)	0.014*	−0.231 [a]
	UN	140.9 ± 15.6			
pKFM occurrence (ms)	IN	136.9 ± 16.0	−0.53 (−2.09 to 1.04)	0.507	−0.062
	UN	137.4 ± 15.5			
Peak quadriceps occurrence (ms)	IN	54.0 ± 26.4	−2.73 (−7.57 to 2.11)	0.266	−0.104
	UN	56.8 ± 24.6			
Peak VM occurrence (ms)	IN	52.2 ± 28.0	−1.31 (−6.19 to 3.58)	0.597	−0.049
	UN	53.5 ± 22.9			
Peak VL occurrence (ms)	IN	52.6 ± 26.9	−3.88 (−8.38 to 0.62)	0.090	−0.159
	UN	56.5 ± 26.3			
Peak RF occurrence (ms)	IN	59.4 ± 24.5	−3.79 (−8.74 to 1.17)	0.133	−0.141
	UN	63.2 ± 25.1			

Neither of these variables were statistically significant after Bonferroni corrections. No statistically significant findings were observed for the remaining variables of interest (*n* = 116).
**P* < 0.05.
[a]Small effect size >0.2.
95% CI, 95% confidence interval; IN, involved limb; UN, uninvolved limb; pKFA, peak knee flexion angle.

DISCUSSIONS

This study aimed to 1) introduce and describe novel metrics of gait by calculating sagittal plane knee RMD, KFMI, and temporal disagreement between quadriceps EMG and kinetic outputs and 2) to investigate how the novel metrics present between the involved and the uninvolved limbs 6- and 24 months after ACLR. Our findings suggest that individuals 6 months after ACLR walk with slower RMD, smaller KFMI, and prolonged extensor latencies in their involved versus uninvolved limbs, perhaps reflecting the known diminished quadriceps neuromuscular function early after ACLR ([21,22,24,30]). Although statistically significant asymmetries were found for stance time and KFMI 24 months after ACLR, no meaningful asymmetries based on MILD values presented herein were present between limbs at 24 months (Tables 3, 5, and 7). Sagittal plane knee RMD, KFMI, and extensor latency during the weight acceptance phase of gait should be considered in future studies to quantify asymmetrical movement patterns and investigate their association with posttraumatic knee OA and other outcomes after ACLR.

Slow and insufficient cumulative loading of the involved knee found in this study, in addition to the smaller magnitude of peak knee joint loading in patients 6 months after ACLR ([4,11,12]), may contribute to the degenerative process seen in knees after ACLR. Building hydrostatic pressure in articular cartilage is one of the key components of maintaining good cartilage health ([3,31]), and loading a joint is the primary method of building hydrostatic pressure in articular cartilage. Inadequate loading and slow loading rates of articular surfaces in the knee joint, however, may lead to less hydrostatic pressure build up during cyclical loading of cartilage ([32]); hence, slow knee joint loading combined with inadequate cumulative load during gait may be detrimental to cartilage health. In addition, the analysis of our secondary variables of interest identified shorter stance times in the involved versus uninvolved limbs, which may persist up to 24 months after ACLR (Tables 6 and 7). Cumulative time spent on the involved leg during a full gait cycle is also shorter and potentially contributing to insufficient cumulative loading of the knee joint after ACLR. Although confirmation of articular surface loading rate and alterations in hydrostatic pressure *in vivo* is necessary, further assessment of stance time and RMD at the knee joint and the association with degenerative changes after ACLR are warranted in future studies.

Altered quadriceps function early after ACLR may be contributing to our findings of asymmetrical knee joint loading and was captured in the prolonged extensor latencies 6 months, but not 24 months, after ACLR (Tables 4 and 5). Our analyses of secondary variables indicate that the prolonged latencies are a result of a combination between the involved limbs reaching peak quadriceps activity earlier and reaching pKFM later compared with their uninvolved limbs (Table 6). Multiple systems likely contribute to prolonged extensor latencies, such as muscle and tendon stiffness, quadriceps weakness and inhibition, knee loading strategy changes, and electromechanical delay of the extensor mechanism. Knee flexion moment calculations using inverse dynamics are also influenced by intersegmental dynamic forces (i.e., hip and ankle moments), which must be considered when interpreting extensor latencies. Further investigation of extensor latencies is needed to determine the exact mechanism behind these changes; however, the present findings add to evidence suggesting overall diminished quadriceps neuromuscular function after ACLR ([30]).

The novel aspect of the extensor latency measures, compared with previous methods, is the ability to quantify quadriceps neuromuscular function in a dynamic setting. In the past, quadriceps EMG in

relation to knee extensor output has been assessed primarily in an isolated setting (e.g., isokinetic dynamometer). To our knowledge, our measure of extensor latency is the first to quantify the timing of peak quadriceps activity in comparison with peak kinetic outputs at the knee joint during a functional task such as gait. This metric may be used in future studies to identify neuromuscular deficits underlying knee kinetic and kinematic alterations during gait. Extensor latency measures may also be adopted in other functional tasks such as drop jump or cutting tasks, where the relevance of quadriceps neuromuscular function may be targeted toward sport performance and secondary injury prevention after ACLR ([33,34]). Although clinical relevance needs further investigation, the novel method of quantifying quadriceps neuromuscular function should be considered in future studies to identify neuromuscular deficits contributing to asymmetrical movement patterns.

There are limitations to consider when interpreting the findings of this study. Our sample was a heterogeneous sample including a variety of patient demographics (e.g., graft type, meniscal involvement, activity level, and postoperative rehabilitation). This heterogeneity does, however, increase the generalizability of these results. Although statistically significant differences were found between limbs, asymmetries exceeding MILD values were only found in KFMI measures at the 6-month time point. Future assessments in a more homogeneous sample are necessary to identify which patient subgroups present with more pronounced asymmetries using our novel metrics. Some degree of association between the novel metrics and the traditional gait mechanics is expected as the values are generated from the same knee moment waveform. In the involved limb, the strength of the associations between the traditional and the novel metrics is, however, weak to moderate: Pearson's correlation coefficients between traditional measures of kinetics (pKFM) and our RMD outcomes in the ACLR limb were 0.202 for RMD_{peak} and pKFM, 0.717 for RMD_{100ms} and pKFM, and 0.749 for RMD_{50ms} and pKFM, indicating that the traditional and novel gait outcomes share 4%–56% variance. Therefore, the novel metrics proposed in this study evaluate unique aspects of gait that have not been closely investigated in the past, the full meaning of which needs to be further evaluated. Comparisons to healthy controls should also be considered as interlimb differences may not be sufficient in identifying changes that occur after ACLR in the uninvolved limb in addition to the involved limb ([11,35]). Follow-up with radiographic evidence and other functional outcomes measures are also necessary to identify the relationship between our novel metrics of gait and outcomes after ACLR.

Sagittal plane RMD is slower, KFMI is smaller, and extensor latencies are longer during the weight acceptance phase of gait in the involved versus uninvolved limbs in participants 6 months after ACLR. These participants also had shorter stance times on their involved limb, indicating less time spent on their involved limb in addition to the slower rate and smaller cumulative sagittal plane loading during gait. Our findings may be driven by altered neuromuscular function of the quadriceps as indicated by prolonged extensor latencies in the involved limbs, ultimately contributing to the high incidences of posttraumatic knee OA in patients after ACLR. The novel metrics of gait introduced in this study may provide insight into quadriceps dysfunction, posttraumatic knee OA development, and other outcomes after ACLR.

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