

Gait mechanics in chronic ACL deficiency and subsequent repair

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Abstract

Objective. To determine how normal gait patterns may change as a result of chronic anterior cruciate ligament deficiency and subsequent reconstructive surgery.

Design. Gait testing of 10 chronic anterior cruciate ligament deficient subjects prior to and 3 months following reconstructive surgery, and 10 uninjured controls.

Background. There is controversy whether persons with chronic anterior cruciate ligament deficiency develop a “quadriceps avoidance” pattern and how anterior cruciate ligament reconstructive surgery influences gait mechanics in these same individuals.

Methods. Gait analysis was employed to determine kinematic, kinetic, and muscle Electromyographic data.

Results. Prior to surgery, no anterior cruciate ligament deficient subject exhibited a quadriceps avoidance pattern. Following surgery, the subjects exhibited a significantly greater knee extensor moment during early stance as compared to the control group. Prior to and following surgery, anterior cruciate ligament deficient subjects demonstrated a significantly greater hip extensor moment possibly to reduce anterior tibial translation.

Conclusions. These data suggest that (1) development of a quadriceps avoidance pattern is less common than previously reported, (2) anterior cruciate ligament deficient subjects accommodate through alterations of hip joint mechanics, (3) surgical repair significantly alters lower extremity gait patterns, and (4) re-establishment of pre-injury gait patterns takes longer than 3 months to occur.

Relevance

The results suggest that chronic anterior cruciate ligament deficient subjects do not exhibit a quadriceps avoidance gait pattern. Surgical intervention significantly alters lower extremity gait mechanics in a population that has accommodated to anterior cruciate ligament deficiency. © 2002 Elsevier Science Ltd. All rights reserved.

Keywords: Anterior cruciate ligament; Quadriceps avoidance; Gait; Surgery

1. Introduction

It has been hypothesized that injury and subsequent repair of the anterior cruciate ligament (ACL) leads to alterations in lower extremity joint kinetics, kinematics, and energetic patterns during gait. These gait patterns may develop as a result of muscle adaptations and neuromuscular reprogramming, possibly in response to pain or instability, to stabilize the knee and to prevent re-injury during gait [1–3]. It has also been demon-

strated that acute (<1 month post-injury) ACL injured patients exhibit significantly different knee moment patterns during gait compared to chronic (>2 years post injury) ACL deficient subjects [1,2]. Individuals who had recently suffered ACL injury exhibited a sustained knee extensor moment throughout stance compared to non-injured control subjects [2]. This gait pattern may result from factors such as knee joint swelling, joint tissue derangement, or muscle inhibition due to pain. It has been suggested that, over time, ACL deficient individuals develop a sustained knee flexor moment during mid-stance (MS). This has been termed a “quadriceps avoidance” pattern, possibly serving to reduce anterior tibial shear during gait [1,3–6]. However, other investigations have found no evidence of a quadriceps

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avoidance gait pattern and suggest that knee moment patterns of chronic ACL deficient subjects resume pre-injury knee moment characteristics [7,8].

Noyes et al. [9] postulated that approximately one-third of ACL deficient patients are able to resume pre-injury activity levels, one-third compensate for the deficiency but have to modify some sport activities, and one-third have to discontinue many sport activities in light of poor knee function. Reconstructive surgery is often used to reestablish functional and mechanical stability of the knee in those ACL deficient patients who experience changes in lifestyle, episodes of giving way, or joint instability. However, factors such as the type of surgery and patient characteristics, as well as the compliance to and type of rehabilitation, may each play a significant role in the type of gait pattern developed following surgery.

Investigations involving ACL reconstructed subjects suggest that time since surgery may play an important role in the return of normal gait patterns [10–15]. Devita et al. [13] examined ACL reconstructed patients 3 weeks and 6 months post-surgically and reported a sustained knee extensor moment and a reduced but prolonged hip extensor moment pattern in ACL reconstructed subjects 3 weeks post-surgically. However, at 6 months following surgery, ACL reconstructed subjects demonstrated knee and hip moment patterns more similar to control group values suggesting that ACL reconstructed subjects can regain pre-injury gait characteristics over time. Bush-Joseph et al. [11] studied a group of ACL reconstructed subjects 8 months after surgery and reported only slight reductions in the peak knee extensor moment during gait. However, it was also reported that two ACL reconstructed subjects exhibited a quadriceps avoidance gait pattern. Timoney et al. [15] reported that at 10 months post-surgery, ACL reconstructed subjects walked with a significantly reduced knee extensor moment as compared to control subjects, suggesting that not all patients demonstrate a time-related return of normal gait patterns during the first year following ACL reconstructive surgery. Bulgheroni et al. [10] studied the gait patterns of ACL reconstructed subjects 2 years post-operatively and reported no significant differences in sagittal plane knee or hip moments suggesting that, given time, ACL reconstructed subjects can regain normal knee moment gait patterns.

The time between injury and surgery may also influence the type of gait pattern observed in ACL reconstructed subjects. Little is known about the gait mechanics of ACL injured subjects prior to and following surgical repair. Devita et al. [2] examined the gait patterns of ACL injured subjects 2 weeks after ACL injury, but before surgery, and 3 and 5 weeks post-surgically. The subjects exhibited a sustained knee extensor moment and a significantly reduced and prolonged hip extensor moment throughout stance prior to surgery,

and 3 weeks post-surgically. These distinctive joint moment patterns were still evident 5 weeks post-surgery but were more similar to the control group. However, those involved in that investigation were acute ACL subjects who were injured less than 1 month prior to data collection. It is possible that subjects who have sustained ACL injury 2 or more years prior to surgery (chronic ACL deficient) may develop gait patterns different than those tested in the acute stages of ACL injury.

Additional studies are necessary to either support or refute the development of a quadriceps avoidance gait pattern. The development of a quadriceps avoidance gait pattern has been described as a patient's tendency to reduce or avoid contraction of the quadriceps muscles. As such, significant alterations in gait mechanics may occur. As well, it is necessary to better understand the neurological and mechanical influences that chronic ACL deficiency and subsequent surgical repair have on the development of gait patterns. Pre- and post-surgical data from a chronic ACL deficient population has not been previously reported in the literature. Since this patient population has accommodated to ACL deficiency over several years, it is possible that these subjects may respond differently to surgical intervention. Therefore, the purpose of this investigation was to determine how normal gait patterns may change as a result of chronic ACL deficient prior to and 3 months following reconstructive surgery.

2. Methods

2.1. Subjects

Twenty subjects participated in this investigation. Ten (five males and five females) ACL deficient individuals were compared with 10 (five males and five females) healthy uninjured age and gender-matched control subjects. The mean age, body weight, and body height of the ACL deficient subjects were 27.7 yr (SD 9.1 yr), 79.1 kg (SD 13.8 kg), and 166.1 cm (SD 20.2 cm), respectively. The ACL deficient subjects had sustained an isolated unilateral ACL injury confirmed by an orthopedic surgeon and had sustained the injury more than 2 years prior to testing (mean = 5.7, SD 5.1 yr). Prior to surgery, all subjects exhibited full knee joint range of motion, no joint swelling, and no pain during ambulation. However, all subjects exhibited at least one episode of knee joint instability (“giving way”) prior to surgery which was the main impetus for undergoing reconstructive surgery. These subjects had a normal contralateral knee and had undergone arthroscopically assisted, endoscopic, bone-patellar-bone reconstruction using the central one-third of the patellar tendon. All subjects were compliant with a conservative

rehabilitation program and no subjects exhibited dysfunction at any other lower extremity joint. Following surgery, all subjects exhibited full knee joint range of motion, none to minimal joint swelling, and no pain during ambulation. No episodes of knee joint “giving way” were reported by any subject.

The mean age, body weight, and body height of control subjects were 24.4 yr (SD 3.1 yr), 67.2 kg (SD 10.7 kg), and 170.1 cm (SD 9.3 cm), respectively. No control subject had a history of lower extremity infirmity or pathology that may have affected the ability to perform the experiment.

The ACL deficient subjects were tested prior to and 3 months following reconstructive surgery. All subjects were physically active, participating in regular activity at least three times per week. Prior to participation, each subject signed a consent form approved by the University’s Human Subjects Compliance Committee Internal Review Board.

2.2. Procedures

The subjects walked along a 5 m wooden walkway in which a force plate was embedded. The subjects walked at a self-selected comfortable pace that was maintained throughout data collection via a metronome. Each subject began walking at a sufficient distance from the force plate so that the self-selected pace was attained prior to the foot of the test limb making contact with the center of the force plate. Muscle electromyographic (EMG), joint kinematic and kinetic data were collected for a 5 s period, which included the step prior to, during, and following contact with the force plate. Data were recorded from 12 trials for the control subject’s right limb and the ACL deficient subject’s injured limb.

2.3. Instrumentation

EMG data were collected using bipolar surface electrodes (DE-02, Delsys, Boston, MA, USA). The electrodes were placed on the skin overlying the muscle belly of the tibialis anterior, medial head of the gastrocnemius, biceps femoris, and vastus lateralis of the test limbs. To achieve an optimal EMG signal and low impedance ($<5\text{ k}\Omega$), three, 4 cm² areas of skin were sanded and cleaned, and electrode gel applied between the skin and electrodes in accordance to procedures outlined by De Luca et al. [16]. All raw EMG analog signals were on-line pre-amplified ($\times 7000$), analog filtered (20–7000 Hz), and converted into digital signals sampled at 1200 Hz for a 5-s duration via the Associated Measurement Laboratory (AMLAB) data acquisition system (AMLAB Inc., Sydney, Australia). Prior to data analysis, EMG signals were full-wave rectified and low-pass filtered at 6 Hz using a fourth order dual-pass Butterworth filter.

EMG data were normalized to the peak EMG muscle activity within a given trial.

A 6-degree of freedom custom force plate (Institute of Neuroscience Technical Service Group, University of Oregon, Eugene, OR, USA) equipped with strain gauges mounted underneath the four corners was used to measure the vertical (Fz), horizontal antero-posterior (Fx), and medio-lateral (Fy) ground reaction forces. Kinetic data were recorded at 1200 Hz for a 5-s duration via the AMLAB system. Prior to analysis, kinetic data were low-pass filtered between 4–10 Hz using a fourth order dual-pass Butterworth filter. Selected filter frequencies were determined for each force signal based on specifications from the manufacturer.

Kinematic data were collected using a PEAK Performance Technologies Real-Time Data Acquisition System (Peak Performance Inc., Denver, CO, USA). Four cameras were positioned 4 m from the sagittal plane along the progression plane of the subject’s gait path. The pre-determined criterion for tolerable error in space calibration was set at 0.2% (2 mm maximum error for a 1-m-long object). Five kinematic reflective markers were placed on the skin overlying the base of the fifth metatarsal, lateral malleolus, lateral condyle of the femur, greater trochanter of the femur, and acromion process of the scapula. A reflective marker was also placed on the force plate to serve as the point of reference for transformation of local center of pressure (CoP) coordinates to global kinematic coordinates. Kinematic data were collected at 120 Hz for a 5 s duration with each of the four cameras synchronized with the AMLAB system. Data were then digitized for the entire collection period. The digitized position data for all markers were then low-pass filtered between 4–8 Hz using a fourth order dual-pass Butterworth filter. Optimal filter frequencies were determined for each force signal based on power spectral analyses wherein 90% of the raw signal was retained after the filtering process. Linear and angular position, velocity and acceleration data were then calculated and exported for further analysis.

2.4. Inverse dynamics calculations

The magnitudes of the segmental masses along with their moments of inertia were estimated using data reported by Dempster [17] and individual subject anthropometric data. CoP was calculated from the ground reaction force data within the force plate local coordinate system. Joint moments were calculated through an inverse dynamics analysis using a custom written MATLAB (The MathWorks, Inc., Natick, MA, USA) computer program combining the anthropometric, kinematic, and kinetic data. Ankle, knee, and hip joint moments were expressed as a reaction moment to all external moments and represent the internal moments normalized to subject mass. All joint moments were

expressed as positive values for extensor and plantarflexor moments. Extensor angular impulse was calculated from the positive area under the joint moment curve. Joint powers were calculated as the product of the joint moments and angular velocities and normalized to subject mass.

2.5. Data reduction

For the purpose of analyzing the temporal relationship between time-series curves, ensemble average curves for the stance phase of gait were computed and each divided into five phases (P) and five discrete points (Pt) that were selected according to kinetic events determined from vertical and anterior/posterior ground reaction forces (Fig. 1). Phase 1 (P1) ranged from heel strike to initial loading (Pt1), phase 2 (P2) from Pt1 to first acceptance of full body weight (Pt3), phase 3 (P3) from Pt3 to MS, phase 4 (P4) from MS to second acceptance of full body weight (Pt5), and phase 5 (P5) from Pt5 to toe-off. Two other discrete points (Pt2, Pt4) denoted the troughs between Pt1 and Pt3 and between Pt3 and Pt5. Values reported are the means of all measured values during that specific phase or point.

2.6. Statistical analysis

Following surgery, the ACL deficient subjects were identified as ACL reconstructed subjects and were treated as an independent group. Two-way repeat measures ANOVAS (10×3 ; $\alpha = 0.05$) and a priori post-hoc tests were used to determine differences, if any, between the three groups (ACL deficient, ACL reconstructed, and control group) for joint moments, powers, and positions and muscle EMG activity for each of the five phases and five discrete points.

3. Results

There were no differences ($P > 0.05$) in total time of stance between the control (863.06, SD 77.27 ms), ACL deficient (865.08, SD 52.22 ms), and ACL reconstructed (853.22, SD 72.33 ms) groups.

3.1. Joint moments

Table 1 presents a summary of comparisons for ACL deficient, ACL reconstructed, and control group lower extremity joint moments for each of the five phases (P) and five discrete points (Pt) of total stance as well as the total joint extensor angular impulse for stance. The control group and ACL deficient knee moments paralleled each other throughout stance ($P > 0.05$). An initial knee flexor moment was observed in early stance, followed by a biphasic extensor–flexor–extensor moment pattern for early, mid-, and late stance periods, respectively (Table 1; Fig. 2A). The ACL reconstructed knee moment curve demonstrated a similar biphasic pattern but significantly greater extensor moment during the first half of MS (P2–P3) compared to the controls (Table 1; Fig. 2A; $P < 0.05$). During the latter half of stance (Pt4–P5), the ACL reconstructed subjects exhibited a significantly smaller flexor moment compared to the control and ACL deficient groups (Table 1; Fig. 2A; $P < 0.05$).

The control group hip extensor moment rose sharply in early stance and then decreased steadily until MS after which a flexor moment was observed (Table 1; Fig. 2B). The ACL deficient hip extensor moment was significantly greater during early stance (Pt1, Pt2, P2) and MS (Pt3–Pt4) periods and then switched to a significantly reduced flexor moment, compared to controls for the remainder of stance (P4, Pt5, P5; Table 1; Fig. 2B; $P < 0.05$). The ACL reconstructed hip extensor moment was significantly greater during early stance

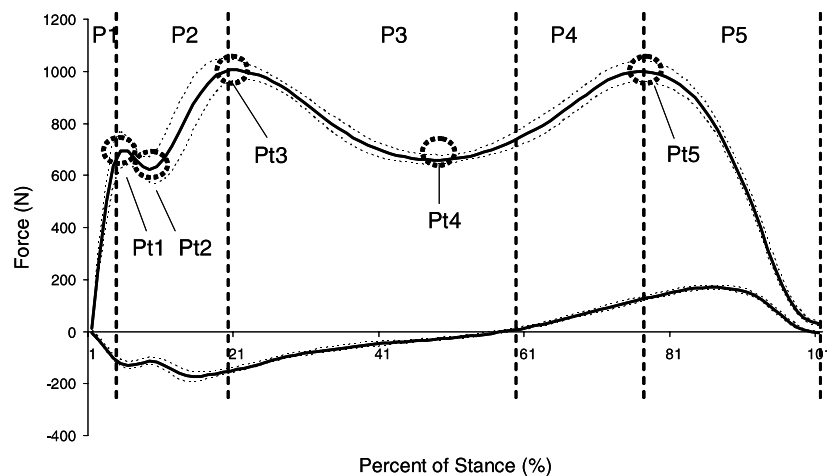


Fig. 1. Selection of five discrete points (dashed circles) and partitioning the stance phase into five phases (dashed vertical lines) according to discrete vertical (top trace) and anterior/posterior (bottom trace) kinetic events.

Table 1

Mean (SD) of knee and hip joint moments^a for control (CON), anterior cruciate ligament deficient (ACLD), and anterior cruciate ligament reconstructed (ACLR) subjects ($n = 10$)

Phase (P)/Point (Pt)	Knee			Hip		
	CON	ACL D	ACLR	CON	ACL D	ACLR
P1	-0.13(0.06)	-0.12(0.04)	-0.12(0.05)	0.30(0.11)	0.33(0.08)	0.37(0.08)
Pt1	-0.12(0.09)	-0.14(0.05)	-0.13(0.05)	0.39(0.17)	0.55(0.09)*	0.56(0.09)*
Pt2	-0.09(0.07)	-0.07(0.09)	-0.08(0.07)	0.38(0.18)	0.58(0.12)*	0.52(0.13)*
P2	0.09(0.06)	0.15(0.05)	0.20(0.03)*	0.24(0.13)	0.65(0.05)*	0.60(0.14)*
Pt3	0.17(0.11)	0.24(0.08)	0.35(0.06)*	0.16(0.14)	0.28(0.07)*	0.20(0.11)
P3	-0.08(0.12)	-0.02(0.05)	0.10(0.05)*	0.04(0.13)	0.21(0.07)*	-0.01(0.10)**
Pt4	-0.23(0.08)	-0.16(0.03)	-0.04(0.05)**	-0.01(0.12)	0.04(0.05)*	-0.10(0.09)**
P4	-0.41(0.09)	-0.33(0.08)	-0.12(0.10)**	-0.31(0.10)	-0.03(0.02)*	-0.17(0.10)*
Pt5	-0.44(0.07)	-0.33(0.05)	-0.11(0.08)**	-0.34(0.11)	-0.08(0.16)*	-0.20(0.08)
P5	-0.18(0.04)	-0.10(0.050)	-0.01(0.05)**	-0.07(0.05)	0.11(0.01)*	-0.01(0.10)
EAI	3.44(2.34)	6.97(3.57)	9.92(3.34)*	12.93(7.19)	16.14(2.05)*	11.36(5.8)

^a Positive values indicate extensor moments, negative values indicate flexor moments (Nm/kg).

* Significantly different than CON ($P < 0.05$).

** Significantly different than ACLD ($P < 0.05$).

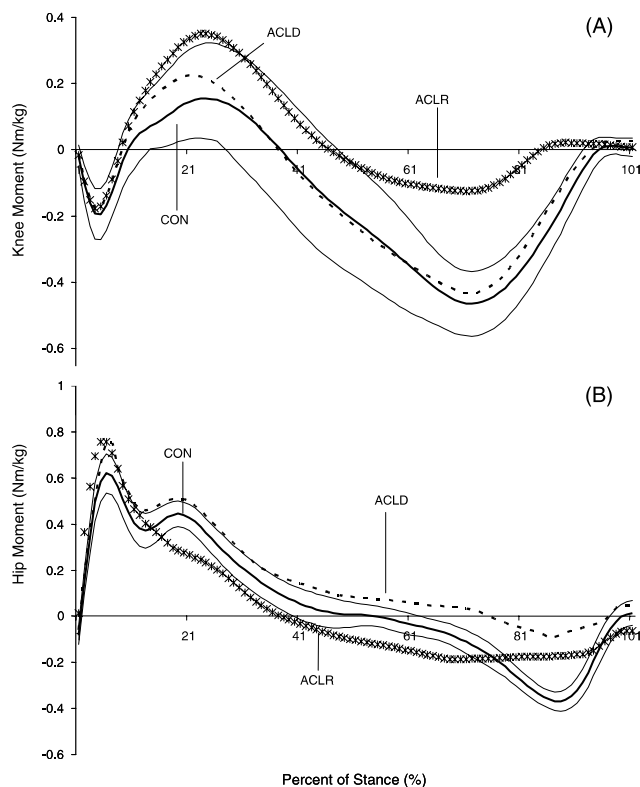


Fig. 2. Knee (A) and hip (B) joint moments for anterior cruciate ligament deficient (ACL D), anterior cruciate ligament reconstructed (ACLR), and control (CON) subjects. Positive values indicate extensor moments, negative values indicate flexor moments. Solid thick line and thin lines are mean and 1SD for CON, dashed thick line is mean of ACLD, and dashed hatches are mean of ACLR group.

(Pt1, Pt2, P2) and during the latter half of MS (P4) compared to controls (Table 1; Fig. 2B; $P < 0.05$). The ACL reconstructed subjects exhibited a significantly greater flexor moment during the first half of MS (P3, Pt4) compared to pre-surgical values (Table 1; Fig. 2B; $P < 0.05$).

3.2. Joint kinematics

Table 2 presents a summary of comparisons for ACL deficient, ACL reconstructed, and control group lower extremity joint position values for P1–5 and Pt1–5 of total stance. The ACL deficient and control group's knee position curves paralleled one another throughout stance and followed a flexion–extension–flexion pattern over early, mid-, and late stance, respectively (Table 2; Fig. 3A; $P > 0.05$). The ACL reconstructed knee paralleled control's throughout stance but was significantly more flexed during the latter half of stance (P3–Pt5; Table 2; Fig. 3A; $P < 0.05$). No significant differences were observed between the ACL reconstructed and ACL deficient knee position throughout stance (Table 2; Fig. 3A; $P > 0.05$).

The control group's hip position curve declined steadily from a flexed position to extension from early to MS respectively after which it followed a flexion–extension pattern from the latter half of mid- to late stance, respectively (Table 2; Fig. 3B). The ACL reconstructed and ACL deficient hip curve generally paralleled the controls curve but was significantly more flexed during early stance (P1, Pt1, Pt2, P2) and the first half of MS (Pt3–Pt4; Table 2; Fig. 3B; $P < 0.05$). No significant differences were observed between the ACL reconstructed and ACL deficient hip position throughout stance (Table 2; Fig. 3B; $P > 0.05$).

3.3. Joint powers

Table 3 presents a summary of comparisons for ACL deficient, ACL reconstructed, and control group lower extremity joint powers during the condition for P1–5 and Pt1–5 of total stance. The control group's knee power was undulating in nature until later in MS when the knee absorbed relatively large amounts of power

Table 2

Mean (SD) of knee and hip joint positions^a for control (CON), anterior cruciate ligament deficient (ACL D), and anterior cruciate ligament reconstructed (ACL R) subjects ($n = 10$)

Phase (P)/Point (Pt)	Knee			Hip		
	CON	ACL D	ACL R	CON	ACL D	ACL R
P1	8.55(2.23)	9.2(2.82)	11.34(3.68)	18.6(1.87)	23.95(3.34)*	24.89(3.49)*
Pt1	10.31(2.34)	10.56(3.17)	12.63(3.75)	17.94(1.88)	23.64(4.31)*	24.34(3.59)*
Pt2	10.86(2.58)	11.68(4.08)	13.31(4.85)	17.77(1.93)	23.47(4.28)*	24.11(3.56)*
P2	14.54(2.24)	16.23(4.62)	17.41(5.22)	14.68(1.05)	21.13(4.12)*	24.51(3.45)*
Pt3	16.13(2.14)	18.76(3.34)	19.77(5.35)	10.06(0.82)	17.76(3.87)*	18.16(3.13)*
P3	12.41(2.47)	14.52(3.18)	16.54(3.00)*	7.45(1.36)	12.9(3.02)*	14.14(2.64)*
Pt4	9.52(2.00)	11.4(2.51)	14.42(3.96)*	6.36(1.34)	10.35(3.36)*	12.41(2.78)*
P4	8.21(1.87)	9.04(2.55)	13.03(4.43)*	8.46(1.79)	11.01(3.04)	12.96(3.18)
Pt5	9.51(2.10)	10.01(3.73)	14.81(4.47)*	10.64(1.92)	12.47(3.00)	14.00(3.40)
P5	21.26(1.92)	19.82(4.47)	23.95(4.60)	10.95(3.84)	12.56(3.69)	13.68(3.79)

^a Positive values indicate flexion, negative values indicate extension (°).

* Significantly different than CON ($P < 0.05$).

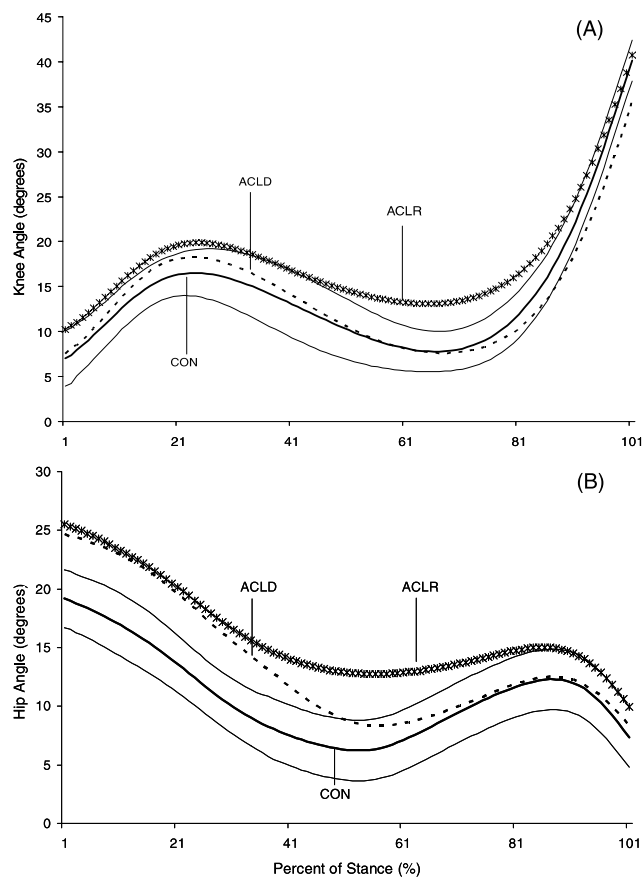


Fig. 3. Knee (A) and hip (B) joint moments for anterior cruciate ligament deficient (ACL D), anterior cruciate ligament reconstructed (ACL R), and control (CON) subjects. Positive and negative values are flexed and extended positions. Solid thick line and thin lines are mean and 1SD for CON, dashed thick line is mean of ACLD, and dashed hatches are mean of ACLR group.

until toe-off (Table 3; Fig. 4A). The ACL deficient knee power curve generally paralleled the control's curve until late stance when the ACL deficient knee absorbed significantly less power (P5; Table 3; Fig. 4A; $P < 0.05$).

The ACL reconstructed knee power curve generally paralleled the control and ACL deficient curves until late stance when the ACL reconstructed knee absorbed significantly less power compared to controls (Pt4–P5) and pre-surgical values (Pt5, P5; Table 3; Fig. 4A; $P < 0.05$).

The control group hip produced power during early stance after which power was absorbed for the first part of MS. Small amounts of power were then generated for the remainder of stance (Table 3; Fig. 4B). The ACL deficient and ACL reconstructed hip curve was similar to controls during early stance but differed significantly during MS when large amounts of power were generated (Pt3, P3; Table 3; Fig. 4B; $P < 0.05$). No significant differences were observed between the ACL reconstructed and ACL pre-surgical hip power values throughout stance (Table 3; Fig. 4B; $P > 0.05$).

3.4. Muscle EMG

Table 4 presents a summary of comparisons for ACL deficient, ACL reconstructed, and control group lower extremity muscle EMG values for P1–5 and Pt1–5 of total stance. Values expressed are normalized to the maximum within-trial EMG amplitude of that muscle and expressed as a ratio with maximum amplitude equal to 1.0. The control vastus lateralis muscle produced a large burst of EMG activity during early stance that steadily dropped and remained relatively low throughout the remainder of stance (Table 4; Fig. 5A). The ACL deficient and ACL reconstructed vastus lateralis EMG activity exhibited a significantly reduced rise to maximum EMG activity during early stance (Pt1, Pt2) compared to controls (Table 4; Fig. 5A; $P < 0.05$). No significant differences were observed between ACL reconstructed and ACL deficient vastus lateralis EMG activity throughout stance (Table 4; Fig. 5A; $P < 0.05$).

The control groups biceps femoris muscle response produced strong EMG activation during early stance,

Table 3

Mean (SD) of knee and hip joint powers^a for control (CON), anterior cruciate ligament deficient (ACL D), and anterior cruciate ligament reconstructed (ACL R) subjects ($n = 10$)

Phase (P)/Point (Pt)	Knee			Hip		
	CON	ACL D	ACL R	CON	ACL D	ACL R
P1	-0.40(0.19)	-0.35(0.19)	-0.32(0.18)	0.36(0.18)	0.26(0.17)	0.41(0.26)
Pt1	-0.37(0.18)	-0.46(0.19)	-0.38(0.23)	0.51(0.32)	0.47(0.28)	0.66(0.24)
Pt2	-0.26(0.06)	-0.19(0.17)	-0.24(0.13)	0.49(0.32)	0.47(0.25)	0.62(0.20)
P2	0.06(0.06)	0.16(0.10)	0.15(0.06)	0.46(0.25)	0.56(0.027)	0.48(0.29)
Pt3	-0.03(0.10)	-0.13(0.10)	-0.16(0.14)	-0.33(0.22)	0.61(0.26)*	0.41(0.17)*
P3	0.06(0.04)	0.03(0.06)	-0.11(0.05)	-0.09(0.04)	0.35(0.15)*	0.12(0.09)*
Pt4	0.22(0.05)	0.23(0.06)	0.04(0.08)*	0.12(0.07)	0.11(0.05)	0.05(0.05)
P4	-0.23(0.11)	-0.08(0.10)	-0.05(0.09)*	0.04(0.09)	0.17(0.06)*	0.11(0.10)
Pt5	-0.92(0.37)	-0.56(0.40)	-0.26(0.13)*,**	0.01(0.03)	0.22(0.16)*	0.13(0.10)
P5	-0.69(0.14)	-0.25(0.35)*	-0.03(0.03)*,**	0.39(0.08)	0.16(0.08)*	0.32(0.08)**

^a Positive values indicate power generation, negative values indicate power absorption (W/kg).

* Significantly different than CON ($P < 0.05$).

** Significantly different than ACL D ($P < 0.05$).

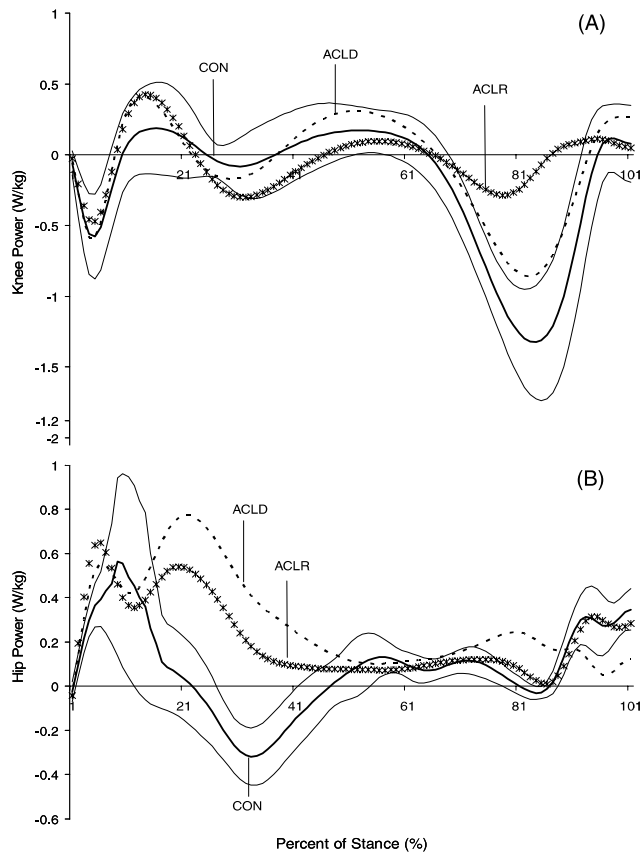


Fig. 4. Knee (A) and hip (B) joint moments for anterior cruciate ligament deficient (ACL D), anterior cruciate ligament reconstructed (ACL R), and control (CON) subjects. Positive and negative values are energy generation and absorption by the muscles. Solid thick line and thin lines are mean and 1SD for CON, dashed thick line is mean of ACL D, and dashed hatches are mean of ACL R group.

declined steadily for all of MS and the first part of late stance, after which another surge of activity was generated prior to toe-off (Table 4; Fig. 5B). The ACL deficient and ACL reconstructed biceps femoris EMG

activity produced significantly greater EMG activity during early stance (P1–P2) compared to controls (Table 4; Fig. 5B; $P < 0.05$). No significant differences were observed between ACL reconstructed and ACL deficient biceps femoris EMG activity throughout stance (Table 4; Fig. 5B; $P > 0.05$).

4. Discussion

4.1. Gait accommodations to ACL deficiency

Berchuck et al. [1] defined a quadriceps avoidance pattern as a sustained knee flexor moment throughout stance. In the present investigation, evidence of a quadriceps avoidance pattern was not observed, as chronic ACL deficient subjects exhibited no differences in knee moment characteristics compared to the control group (Fig. 2A). This finding is in contrast to previous investigations who have reported that ACL deficient patients injured for 2 or more years tend to develop a quadriceps avoidance gait pattern [1,3–6]. However, the results of the present investigations are consistent with other investigators who have reported that a quadriceps avoidance phenomenon does not develop in chronic ACL deficient patients [7,8].

Wexler et al. [3] found that 57% of their ACL deficient patients exhibited a quadriceps avoidance pattern while Birac et al. [5] reported an 80% quadriceps avoidance rate in ACL deficient subjects greater than 6 years post-injury. Since all ACL deficient subjects in the present investigation were similar in time since injury to those of Wexler et al. [3] and some ($n = 4$) fell within the >6 year period studied by Birac et al. [5] it is surprising that no ACL deficient individual involved in the current study exhibited a quadriceps avoidance gait pattern.

It has been suggested that inherent differences in the methodology used to calculate joint moments might

Table 4

Mean (SD) muscle EMG activity^a for control (CON), anterior cruciate ligament deficient (ACL), and anterior cruciate ligament reconstructed (ACLR) subjects ($n = 10$)

Phase (P)/Point (Pt)	BF			VL		
	CON	ACL	ACLR	CON	ACL	ACLR
P1	0.81(0.10)	0.94(0.03)*	0.93(0.04)*	0.80(0.07)	0.68(0.07)*	0.74(0.15)
Pt1	0.68(0.12)	0.92(0.08)*	0.90(0.08)*	0.95(0.03)	0.83(0.18)*	0.86(0.07)*
Pt2	0.63(0.12)	0.87(0.14)*	0.87(0.10)*	0.97(0.02)	0.88(0.07)*	0.89(0.07)*
P2	0.39(0.05)	0.50(0.06)*	0.53(0.10)*	0.73(0.10)	0.78(0.06)	0.73(0.09)
Pt3	0.32(0.08)	0.32(0.05)	0.40(0.19)	0.47(0.07)	0.48(0.10)	0.47(0.22)
P3	0.26(0.10)	0.31(0.13)	0.34(0.16)	0.35(0.15)	0.36(0.09)	0.37(0.19)
Pt4	0.36(0.04)	0.35(0.06)	0.33(0.20)	0.30(0.12)	0.32(0.12)	0.35(0.19)
P4	0.37(0.18)	0.35(0.15)	0.36(0.21)	0.30(0.04)	0.36(0.04)	0.32(0.20)
Pt5	0.21(0.06)	0.30(0.11)	0.35(0.22)	0.33(0.11)	0.42(0.08)	0.33(0.22)
P5	0.33(0.07)	0.27(0.10)	0.32(0.10)	0.30(0.13)	0.34(0.04)	0.31(0.15)

^a Muscle EMG activity normalized to maximum amplitude within NP condition.

* Significantly different than CON ($P < 0.05$).

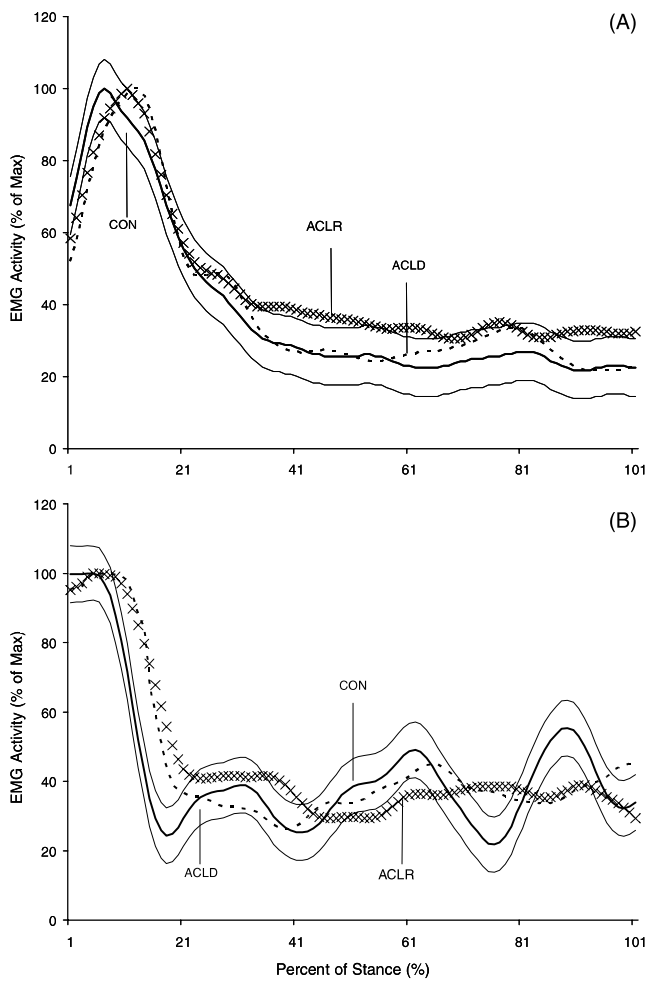


Fig. 5. Muscle EMG activity of anterior cruciate ligament deficient (ACL: dashed thick line), anterior cruciate ligament reconstructed (ACLR: dashed hatches), and control (CON: solid thick line and thin lines are mean and 1SD) subjects for the vastus lateralis (A), and biceps femoris (B).

serve to explain the variations in reports of the quadriceps avoidance pattern [7]. Roberts et al. [7] used Euler

angles and a 3-D analysis while others employed a simple linked segment model that assumes that flexion and extension occurs in a purely sagittal plane [1,3–6]. Euler angles use imbedded 3-D coordinate systems within each lower extremity joint and more accurately represent joint moment patterns along the 3-D planes of movement. Roberts et al. [7] contended that the different models themselves may partly account for the discrepancies in reported results. However, the present investigation also used a simple linked segment model but revealed no evidence of a sustained knee flexor moment or reduced knee extensor moment during the stance phase of gait.

No previous studies investigating the development of an ACL deficient quadriceps avoidance pattern has measured muscle EMG activity to help support or refute the existence of a sustained knee flexor moment. It has been reported that in healthy subjects, the vastus lateralis muscle exhibits a steady rise in muscle EMG activity during early stance [18,19]. Similar to those studies, the control and ACL deficient groups in the present investigation exhibited a steady rise in vastus lateralis EMG activity during early stance but the ACL deficient group exhibited significantly less vastus lateralis EMG activity compared to controls (Fig. 5A). However, differences in vastus lateralis EMG activity may be due to the method of EMG data normalization employed in this investigation. EMG data were normalized to the maximum within-trial amplitude (100%) and, thus, EMG amplitudes of the vastus lateralis muscle in the ACL deficient group relative to the control group were not available.

The reduced rise in vastus lateralis EMG activity exhibited by the ACL deficient group could be interpreted as a quadriceps avoidance gait pattern. However, since no differences in knee joint moment, power, or angle were observed between the ACL deficient and control groups, the differences in vastus lateralis EMG activity between the two groups are unlikely to be indicative of a quadriceps avoidance pattern. The reduced

vastus lateralis EMG activity can be interpreted as a reduced rise (slope) in vastus lateralis EMG activity toward maximum activation. The reduced slope could result from maximum vastus lateralis activation occurring slightly later in stance in the ACL deficient group compared to controls (Fig. 5A). It is possible that the later maximum activation and subsequent reduced rise in activation was a strategy to help reduce anterior tibial translation in the ACL deficient group.

Previous investigations reported that ACL deficient subjects were more flexed at the hip and knee joints and exhibited a greater hip extensor moment during stance [1,3,7]. In the present study, ACL deficient subjects demonstrated no significant differences from the control group subjects in knee joint position throughout stance, but produced significantly greater hip flexion throughout most of stance (Fig. 3B). The ACL deficient subjects also demonstrated a significantly greater and prolonged hip extensor moment compared to the control group (Fig. 2B). A more flexed hip position possibly demands a greater hip extensor moment early in stance to reduce and control the forward acceleration of the upper body. However, Devita et al. [2] suggested that a fundamental change in the length–tension relationship in hip extensor muscles of ACL deficient subjects during the stance phase of gait may occur as a result of an increased hip flexion position, possibly to help reduce anterior tibial translation. If knee position of the ACL deficient subjects in the present investigation remained unchanged throughout stance, a greater hip flexion position would alter the length–tension relationship of the hamstring muscles and possibly serve to reduce anterior tibial translation throughout stance.

Gait studies have demonstrated that peak anterior tibial translation occurred at or near full knee extension ($\sim 5^\circ$) during stance [20,21]. As well, Pandy and Shelburne [22,23] and Li et al. [24] reported that anterior tibial translation was greatest between full knee extension and 25° of knee flexion in both healthy and ACL deficient knee models. Furthermore, Pandy and Shelburne [21] indicated that patellar tendon force and the moment arm of the patellar tendon are similar in ACL deficient and healthy knees and suggest that function of the knee extensor mechanism is not altered by the loss of the ACL. In the present study, the knee is near full extension ($\sim 7\text{--}10^\circ$) at two points during stance: following heel strike and following MS (Fig. 6). It has also been demonstrated that the hamstring muscles, as a component of the hip extensor moment, are effective synergists to the ACL in reducing anterior tibial translation [20–22,25,26]. In the present investigation ACL deficient subjects demonstrated a significantly greater hip extensor moment (Fig. 2B), reduced rise in vastus lateralis EMG activity (Fig. 5A), and prolonged near-maximal biceps femoris EMG activity (Fig. 5B) compared to controls following heel strike. It is possible that the in-

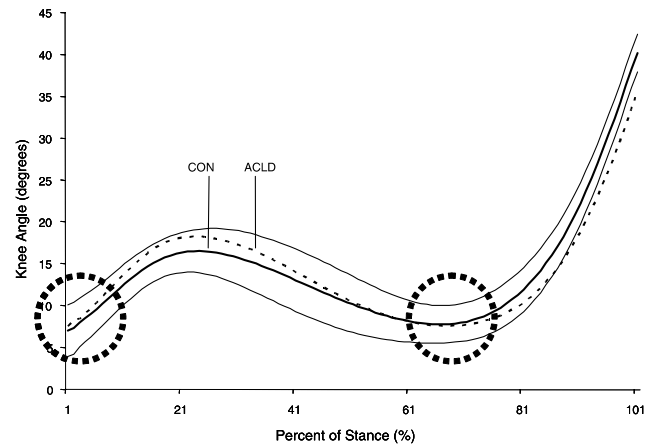


Fig. 6. Knee joint position curves for anterior cruciate ligament deficient (ACLD: dashed thick line) and control (CON: solid thick line and thin lines are mean and 1SD) subjects. Positive values indicate flexion, negative values indicate extension. Dashed circles indicate two times during stance when the knee joint is near full extension and anterior tibial translation is greatest.

crease in the hip extensor moment and reciprocal activity of the thigh muscles exhibited by ACL deficient subjects was necessary to reduce anterior tibial translation when shear force to the knee joint would be greatest. Following MS, control subjects demonstrated a hip flexor moment while ACL deficient subjects exhibited a prolonged hip extensor moment (Fig. 2B). It is possible that control subjects were able to produce a hip flexor moment following MS since the intact ACL was able to restrain the tibia posteriorly and it was therefore not necessary to generate a greater hip extensor moment. The prolonged hip extensor moment following MS exhibited by ACL deficient subjects may have been necessary in order to reduce anterior tibial translation at the second point of stance when anterior tibial translation is greatest.

Coinciding with the prolonged hip extensor moment following MS, ACL deficient subjects demonstrated significantly greater hip extensor power generation (Fig. 4B), a knee flexor moment (Fig. 2A), and significantly reduced knee flexor muscle power absorption as compared to the control group (Fig. 4A). It is possible that the reduction in knee power absorption in ACL deficient subjects was due to the bi-articular action of the hamstring muscles that were simultaneously generating hip extensor and knee flexor power possibly to stabilize the tibia when the knee was near full extension during stance.

It is also possible that the significantly more flexed hip position exhibited by the ACL deficient group resulted in the increased hip extensor moment. Devita et al. [2] hypothesized that a more flexed (crouched) position demands a greater hip extensor moment to prevent collapse of the body during the stance phase of gait. Other investigations have indicated that the injured limb

of an ACL deficient population exhibited greater anterior tibial translation during stance as compared to the contralateral, non-injured limb and compared to controls [24,27]. However, Beard and associates [28] reported no differences in anterior tibial translation during gait following reconstructive surgery compared to pre-surgical values. While several studies support the theory that ACL deficient subjects actively try to limit anterior tibial translation during gait [1–8,10–15,20–22], there remains little evidence to support its merits. Future studies investigating anterior tibial translation in ACL deficient and reconstructed population may shed more light on this important topic.

4.2. Gait accommodations to ACL reconstruction

Three months following surgery, several gait characteristics observed in the present study were significantly different compared to pre-surgical values. The ACL reconstruction subjects were approximately 3° more flexed at the knee and hip as compared to pre-surgical values, and approximately 5° more flexed than controls (Fig. 3). Devita et al. [13] reported that ACL reconstruction subjects walked with approximately 10° more flexion at the hip and knee joints 3 weeks after surgery but no differences were observed 6 months later compared to uninjured controls. Bush-Joseph et al. [11] reported no significant differences in knee flexion angle at MS 8 months after surgery. The results from this investigation are consistent with those findings and suggest that ACL reconstructed subjects may not yet be fully recovered 3 months after surgery but that over time, ACL reconstructed subjects may gradually regain a more erect posture during the stance phase of gait.

The ACL reconstructed subjects produced a significantly greater knee extensor moment during early stance compared to the control group and a significantly reduced knee flexor moment for the remainder of stance compared to the controls and pre-surgical values (Fig. 2A). To the authors' knowledge, this pattern has not been previously reported in any ACL reconstruction gait investigation. Devita et al. [2] demonstrated that ACL reconstruction subjects exhibited a sustained knee extensor moment throughout stance both pre- and post-surgically, although the 5 week ACL reconstruction knee moment pattern more closely resembled knee moment characteristics of healthy control subjects. Since the subjects involved in that study were less than 1 month post-injury, it is unknown how acute ACL injury influenced post-operative gait patterns. Devita et al. [13] further investigated ACL reconstruction subjects who were undergoing an accelerated rehabilitation protocol 3 weeks and 6 months post-operatively. No data regarding time between injury and surgery nor comparisons to pre-surgical data from the same subjects were reported. The results indicated that the ACL reconstruction group demon-

strated a sustained knee extensor moment throughout stance at 3 weeks post-operatively, a result similar to 3 week ACL reconstruction data previously reported [2]. Devita et al. [13] also demonstrated that ACL reconstruction subjects at 6 months post-surgery exhibited a biphasic knee extensor–flexor–extensor moment pattern similar to healthy subjects but a significantly reduced positive extensor angular impulse. This suggests that after approximately 6 months, ACL reconstructed subjects can approach normal joint moment patterns but that more time may be needed to re-establish pre-injury gait characteristics. In the present investigation, ACL reconstructed subjects demonstrated a biphasic knee moment pattern although the pattern was significantly different in magnitude compared to controls and pre-surgical values (Fig. 2A). These data are consistent with those of Devita et al. [2,13] and further demonstrate a time-related trend toward re-establishment of pre-injury knee moment patterns but suggest that several months may be needed for this pattern to develop.

Timoney et al. [15] observed ACL reconstruction subjects between 9 and 12 months after surgery with a range of 1–66 months between injury and surgery. They found that ACL reconstructed subjects walked with a 64% reduction in knee extensor moment at MS compared with healthy controls. The knee extensor moment results reported by Timoney et al. [15] were lower than those reported in this investigation at 3 months post-surgery and those reported by Devita et al. [2,13] at 3 weeks post-surgery. It is possible that factors such as patient compliance, different rehabilitation protocols, and different times between injury, surgery, and data collection could account for the discrepancies between the results from this investigation and those of Timoney et al. [15] and Devita et al. [13].

It is also possible that intra-articular knee effusion may also serve to explain the discrepancies between various studies involving ACL reconstructed individuals. Torry et al. [29] suggested that intra-articular knee effusion significantly alters gait mechanics in healthy individuals and may be responsible for many gait adaptations reported in previous ACL injured investigations. And similarly, the ACL reconstructed subjects in the present study, exhibited no to minimal knee joint effusion which may also explain some of the alterations in the knee joint moment pattern as compared to pre-surgical and control group values (Fig. 2A). However, Torry et al. [29] reported that knee joint effusion resulted in a decrease in peak knee extensor moment while the ACL reconstructed subjects in this investigation exhibited an increase in the peak knee extensor moment (Fig. 2A). It is possible that an injured population may respond differently to knee joint effusion as compared to the healthy group investigated by Torry et al. [29]. It is also possible that the bone-patellar tendon-bone surgery itself may have resulted in alterations in knee joint

mechanics regardless of time between injury and surgery or knee joint or knee joint effusion.

The ACL reconstructed subjects exhibited a significantly altered hip moment pattern during early stance as compared to controls and pre-surgical values (Fig. 2B). These data are similar to previous investigations involving ACL reconstruction subjects who were less than 1 year post-surgery suggesting that the hip had not yet re-established normal joint moment characteristics at 3 months post-surgery [2,10,11,13,15]. As previously noted, Devita et al. [2] hypothesized that a more flexed (crouched) position, indicative of an ACL reconstructed population, demands a greater knee and hip extensor moment to prevent collapse of the body during the stance phase of gait. The significantly greater knee and hip extensor moment observed in the ACL reconstruction group during early stance could be the result of a more crouched gait position to prevent collapse.

5. Conclusion

The data from this study did not reveal a quadriceps avoidance gait pattern in the ACL deficient subjects. The ACL deficient subjects appeared to accommodate to chronic ACL deficiency through alterations of hip and ankle joint kinematic and kinetic and muscle power patterns during the stance phase of gait. Three months following surgery, these same subjects demonstrated a significantly different knee moment and were significantly more flexed at the knee and as compared to pre-surgical ACL deficient and control values. The ACL reconstructed group also exhibited a hip moment pattern significantly different from pre-surgical values. These data suggest that time since injury plays an important role in the adaptation of gait mechanics and must be considered when evaluating post-surgical ACL subjects. These data also suggest that ACL surgical repair significantly alters lower extremity gait patterns regardless of time since injury and that the re-establishment of pre-injury gait patterns takes longer than 3 months to occur.

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