

Gait perturbation response in chronic anterior cruciate ligament deficiency and repair

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Abstract

Objective. To determine how chronic anterior cruciate ligament deficient and surgically repaired subjects react to unexpected forward perturbations during gait as compared to healthy controls.

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

Background. The ability of an anterior cruciate ligament injured individual to react and maintain equilibrium during gait perturbations is critical for the prevention of reinjury. No studies have investigated how these individuals respond to unexpected perturbations during normal gait.

Methods. An unexpected forward perturbation was induced upon heel strike using a force plate capable of translational movement.

Results. Prior to surgery, the anterior cruciate ligament subjects exhibited a greater knee extensor moment in response to the perturbation compared to healthy controls. Following surgery, the anterior cruciate ligament injured subjects exhibited a static knee position and a sustained knee extensor moment throughout stance in response to the perturbation as compared to controls.

Conclusions. These data suggest that chronic anterior cruciate ligament deficient subjects rely heavily on knee extensor musculature to prevent collapse in response to an unexpected perturbation. This same reactive response was more pronounced 3 months following surgery.

Relevance

The results suggest that, prior to and following surgery, chronic anterior cruciate ligament injured subjects respond differently than healthy controls to an unexpected perturbation during gait. Anterior cruciate ligament injured or repaired subjects do not reduce or avoid vigorous contraction of the quadriceps muscles when responding to gait perturbations.

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1. Introduction

Walking, although an extremely complex motor task, is one of the most common and repetitive non-contact movements. Human gait is considered relatively unstable due to a small base of support, long single support phase, and because two-thirds of the body's mass is lo-

cated in the upper torso (Winter et al., 1990). Few studies have been conducted that quantify reactive gait alterations due to unexpected gait perturbations (Ferber et al., 2002; Nashner, 1980; Tang et al., 1998). Nashner (1980) and Tang et al. (1998) incorporated a moveable platform into a walkway to simulate unexpected forward perturbations during gait. Results from these two studies indicate that a reactive strategy to gait perturbations in healthy individuals is to generate distal to proximal muscle activity patterns as well as longer burst durations and higher magnitudes of muscle

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electromyographic (EMG) activity in comparison to unperturbed gait. Ferber et al. (2002), using a similar paradigm, determined the lower extremity joint kinematic and kinetic patterns of healthy individuals in response to an unexpected forward perturbation. They reported that the hip produced a large extensor moment during early stance and was most important in maintaining control of the upper body, and preventing collapse of the lower extremity, as an initial response to an unexpected perturbation. However, later in stance, a large knee extensor moment became the dominant contributor to prevent collapse of the lower extremity and maintaining dynamic equilibrium (Ferber et al., 2002).

No studies have investigated how individuals with an injured anterior cruciate ligament (ACL) respond to unexpected gait perturbations. During an unexpected gait perturbation, the ability of an ACL injured individual to react and maintain equilibrium may be compromised due to changes in neuromuscular reprogramming as a result of injury and/or repair (Berchuck et al., 1990; Devita et al., 1997; Ferber et al., 2002). It has been hypothesized that rupture of the ACL leads to muscle adaptations and subsequent neuromuscular reprogramming that serve to stabilize the knee and prevent injury during non-perturbed gait (Andriacchi, 1983; Berchuck et al., 1990; Birac et al., 1991; Devita et al., 1997; Ferber et al., 2002; Roberts et al., 1999; Rudolph et al., 1998; Wexler et al., 1998). It has also been suggested that time since injury may play an important role in the type of gait adaptation observed in ACL injured patients (Berchuck et al., 1990; Devita et al., 1997; Wexler et al., 1998). Individuals who had recently suffered ACL injury exhibited a sustained knee extensor moment throughout stance compared to non-injured control subjects (Devita et al., 1997). 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. This has been termed a “quadriceps avoidance” pattern, possibly serving to reduce anterior tibial translation during gait (Andriacchi, 1983; Berchuck et al., 1990; Birac et al., 1991; Wexler et al., 1998). However, other investigators have been unable to reproduce the quadriceps avoidance phenomenon in chronic ACL deficient patients (Ferber et al., 2002; Roberts et al., 1999; Rudolph et al., 1998). Ferber et al. (2002) suggested that development of a quadriceps avoidance pattern is less common than previously reported. It was suggested that subjects adapt to chronic ACL deficiency through alterations in hip joint moment and power patterns in an effort to stabilize the knee joint and help prevent excessive anterior tibial translation (Ferber et al., 2002).

The time between injury and surgery may influence the type of gait pattern observed in ACL reconstructed subjects. Devita et al. (1997) examined the gait patterns

of ACL injured subjects 3 and 5 weeks post-surgically. The subjects exhibited a sustained knee extensor moment, a more flexed hip and knee joint position, and a significantly reduced and prolonged hip extensor moment throughout stance 3 weeks post-surgically. These distinctive joint moment patterns were still evident 5 weeks post-surgery but were more similar to the control group. Ferber et al. (2002) investigated chronic ACL injured subjects 3 months following reconstructive surgery and reported that these subjects exhibited a knee moment pattern similar to the 5 week post-surgery subjects investigated by Devita et al. (1997). Ferber et al. (2002) also reported that the ACL repaired subjects were significantly more flexed at the knee and hip compared to controls. These authors concluded 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.

It is important to determine how an ACL injured individual reacts to unexpected gait perturbations, especially since individuals often encounter obstacles or perturbations during gait. The purpose of this study was to determine the effect of unexpected forward perturbations (FP) during gait on lower extremity joint kinematic and kinetic patterns in chronic ACL deficient subjects prior to and 3 months following surgical repair and in healthy controls. This information may aid in the rehabilitation of ACL deficient and reconstructed patients and help to prevent re-injury due to unexpected perturbations during gait.

It has been shown that healthy control subjects exhibit a more flexed knee and hip in response to an expected FP (Ferber et al., 2002). As a protective response to the unexpected FP, it was hypothesized that the pre-surgical ACL deficient limb would exhibit greater knee and hip joint flexion positions in comparison to the uninjured controls during the FP. It was also hypothesized that the pre-surgical limb would exhibit greater knee and hip extensor moments compared to healthy controls in response to the FP. Since it has been reported that a flexed hip and knee position is common following ACL surgical repair, it was hypothesized that the ACL repaired limb would demonstrate a sustained knee extensor moment, greater hip extensor moment, and greater hip and knee flexion positions compared to pre-surgical values and controls in response to the FP.

2. Methods

2.1. Participants

Twenty subjects participated in this investigation. Ten (5 males and 5 females) ACL deficient individuals were compared with 10 (5 males and 5 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 (SD 9.1) yr, 79.1 (SD 13.8) kg, and 166.1 (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, no to minimal joint swelling, and no pain during ambulation. No episodes of knee joint “giving way” were reported by any subject after surgery.

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

The control subjects were tested one time while 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 3 times per week. Prior to participation, each subject signed a consent form approved by the University’s Human Subjects Compliance Committee Institutional Review Board.

2.2. Protocol

Subjects walked along a 5 m wooden walkway in which a force plate, capable of translational movement, was embedded. When preset, the force plate moved forward or backwards a distance of 10 cm at a velocity of 40 cm/s upon heel contact. The selected velocity was based on previous literature reporting heel velocities during realistic slip movements when a person is walking on a slippery surface (Strandberg and Lanshammar, 1981).

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. Joint kinematic and kinetic data were collected while the subjects walked along the walkway for a 5 s period,

which included the step prior to and following contact with the force plate.

Data were recorded from the ACL subjects injured limb and the control subject’s right limb. A total of 36 trials were collected consisting of 12 FP, 12 non-perturbation, and 12 backward perturbation trials. The backward and non-perturbation trials, along with the random order of trials, were used to help prevent possible accommodation and anticipation of the FP condition. Subjects were not allowed to practice the perturbation trials.

There was a small risk that the subjects could fall when their balance was perturbed. To minimize the risk, the subjects wore a harness attached to an overhead track and were provided a handrail to grasp if needed.

2.3. Instrumentation

A 6-degree of freedom custom-built 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 (F_z), antero-posterior (F_x), and medio-lateral (F_y) ground reaction forces. Using a feedback electric circuit, the F_z forces also served as trigger signals to initiate the force plate movement when the signal registered approximately 40 N (~8% of body weight). Kinetic data were recorded at 1200 Hz for a 5-s duration via an Associated Measurement Laboratory (AMLAB) data acquisition system (AMLAB Inc., Sydney, Australia). Prior to analysis, kinetic data were low-pass filtered between 4 and 10 Hz using a 4th 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 m³ volume). Five 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 register plate movement and 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 4 cameras synchronized with the AMLAB system. Each marker was then digitized for the entire collection period including the stride before and after the stance phase on the force plate. The digitized position data for all markers were then low-pass filtered

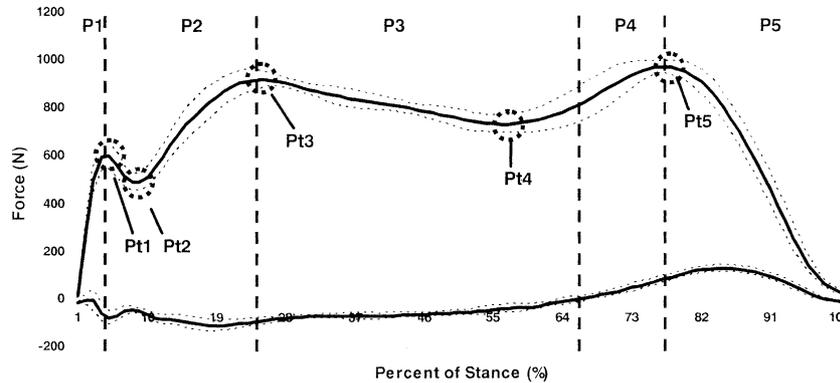


Fig. 1. Selection of five discrete points (dashed circles) and partitioning the stance phase into five phases (dashed vertical lines) according to discrete vertical (top trace) and anterior/posterior (bottom trace) kinetic events.

between 4 and 8 Hz using a 4th order dual-pass Butterworth filter. Optimal filter frequencies were determined for each force signal based on power spectral analyses wherein 80% 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 (1959) 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 (EAI) 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 analysis

The variables of interest were hip and knee joint moments, powers, and angles. Prior to analysis, each trial was partitioned for the stance phase of the gait cycle (heel strike to toe off), interpolated as a percent of stance, and an ensemble average was created by averaging the 12 trials. For the purpose of analyzing the temporal relationship between time-series curves, each ensemble average curve was divided into five phases (P)

and five discrete points (Pt) that were selected according to discrete 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 mid-stance (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, respectively. Comparisons were made for differences in knee and hip joint moments, powers, and angles during each of the five phases and at each of the five discrete points. Values reported are the averages of all variables 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 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 angles for each of the five phases and five discrete points.

3. Results

The onset of force plate movement occurred at 3.1 (SD 0.2)% of stance (approximately 29.10 (SD 0.19) ms after heel strike or at Point 1) and ended at 59.8 (SD 2.5)% of stance (approximately 543.21 (SD 0.24) ms after onset or at the end of Phase 3). There were no differences ($P > 0.05$) in total time of stance between the control (977.14, SD 58.33 ms), ACL deficient (962.33, SD 77.00 ms), and ACL reconstructed (907.20, SD 72.24 ms) groups.

3.1. Joint moments

Table 1 presents a comparison of ACL deficient, ACL reconstructed, and control hip and knee joint moments for P1-5 and Pt1-5 of total stance as well as the total joint EAI for stance. No significant ($P > 0.05$) differences in hip EAI were observed between groups but the ACL reconstructed subjects demonstrated significantly ($P < 0.05$) greater knee EAI compared to controls (Table 1).

The control knee moment curve exhibited a flexor–extensor–flexor pattern through early, mid-, and late stance, respectively (Table 1, Fig. 2A). The ACL deficient knee moment paralleled the control knee moment curve throughout stance but demonstrated a significantly ($P < 0.05$) smaller flexor moment during the latter half of early stance (Pt2, P2) and a significantly ($P < 0.05$) greater extensor moment during the early part of mid-stance (Pt3, P3; Table 1, Fig. 2A). The ACL reconstructed knee demonstrated a near net zero ($P < 0.05$) moment during the latter half of early stance (Pt2, P2) and a significantly ($P < 0.05$) greater extensor moment during mid-stance (Pt3–Pt4) compared to controls and pre-surgical values (Table 1, Fig. 2A). During the latter half of stance (P4, Pt5), the ACL reconstructed knee produced a sustained knee extensor moment significantly different ($P < 0.05$) compared to the control and pre-surgical values (Table 1, Fig. 2A).

The control, ACL reconstructed, and ACL deficient hip moments paralleled one another throughout stance ($P > 0.05$). The hip extensor moments rose sharply after heel strike and then rapidly decreased throughout the remainder of stance for all groups (Table 1, Fig. 2B).

3.2. Joint kinematics

Table 2 presents a comparison of ACL deficient, ACL reconstructed, and control hip and knee joint position values for P1-5 and Pt1-5 of total stance. The ACL deficient, ACL reconstructed, and control knee curves paralleled one another throughout stance ($P > 0.05$) and remained in a relatively static position until late stance when a sharp increase in the amount of knee flexion was observed (Table 2, Fig. 3A).

The control group hip curve moved steadily from a flexed position to extension from early to mid-stance after which it followed a flexion-extension pattern until toe-off (Table 2, Fig. 3B). The ACL deficient hip curve followed a similar pattern but was significantly ($P < 0.05$) more flexed during early stance (P1–P2) and most of mid-stance compared to controls (Pt3–Pt4; Table 2, Fig. 3B). Similarly, the ACL reconstructed hip curve was significantly ($P < 0.05$) more flexed during early stance (P1–P2) and the first half of mid-stance (Pt3–Pt4) compared to the control group (Table 2, Fig. 3B). No significant ($P > 0.05$) differences were observed between the ACL reconstructed and ACL deficient hip position throughout stance (Table 2, Fig. 3B).

3.3. Joint powers

Table 3 presents a comparison of ACL deficient, ACL reconstructed, and control hip and knee joint powers for P1-5 and Pt1-5 of total stance. The ACL deficient, ACL reconstructed, and control knee power curves were similar to one another ($P > 0.05$) and demonstrated undulating patterns of power production and absorption throughout stance (Table 3, Fig. 4A).

Table 1

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

Phase (P)/ Point (Pt)	Stance partition					
	Knee			Hip		
	CON	ACLD	ACLR	CON	ACLD	ACLR
P1	-0.26 (0.06)	-0.19 (0.06)	-0.21 (0.06)	0.49 (0.11)	0.46 (0.12)	0.51 (0.14)
Pt1	-0.38 (0.09)	-0.34 (0.08)	-0.33 (0.06)	0.80 (0.15)	0.89 (0.16)	0.89 (0.14)
Pt2	-0.27 (0.07)	-0.17 (0.05)*	-0.12 (0.07)*	0.59 (0.14)	0.62 (0.12)	0.57 (0.15)
P2	-0.14 (0.10)	0.05 (0.07)*	0.06 (0.01)*	0.55 (0.17)	0.53 (0.14)	0.45 (0.17)
Pt3	0.22 (0.15)	0.50 (0.25)*	0.38 (0.06)*	0.14 (0.14)	-0.07 (0.30)	-0.03 (0.09)
P3	0.02 (0.14)	0.36 (0.17)*	0.33 (0.16)*	-0.21 (0.17)	-0.28 (0.15)	-0.31 (0.15)
Pt4	0.09 (0.02)	0.13 (0.05)	0.17 (0.05)*	-0.28 (0.19)	-0.25 (0.18)	-0.32 (0.13)
P4	-0.22 (0.11)	-0.10 (0.05)	0.01 (0.05)*.#	-0.14 (0.16)	-0.19 (0.20)	-0.29 (0.11)
Pt5	-0.27 (0.13)	-0.16 (0.15)	0.03 (0.01)*.#	-0.12 (0.21)	-0.15 (0.03)	-0.24 (0.13)
P5	-0.09 (0.06)	-0.06 (0.05)	-0.02 (0.05)	-0.02 (0.07)	0.01 (0.15)	-0.01 (0.08)
EAI	8.39 (2.66)	14.79 (7.66)	15.28 (8.70)*	22.66 (7.25)	23.61 (10.38)	19.40 (5.46)

[†] Positive values indicate extensor moments, negative values indicate flexor moments (Nm/kg).

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

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

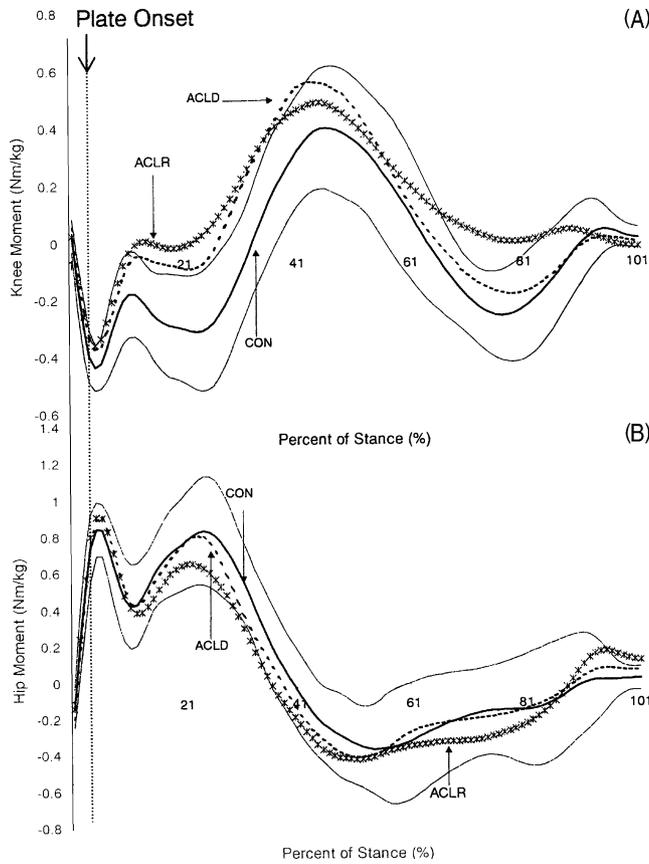


Fig. 2. Knee (A) and hip (B) joint moments for anterior cruciate ligament deficient (ACLD), anterior cruciate ligament reconstructed (ACLR), and control (CON) subjects. Positive values indicate extensor moments, negative values indicate flexor moments. Solid thick line and thin lines are mean and 1 SD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

The control and ACL deficient hip produced similar ($P > 0.05$) amounts of power during early stance, and absorbed similar ($P > 0.05$) amounts of power during mid-stance. Similar ($P > 0.05$) amounts of power were

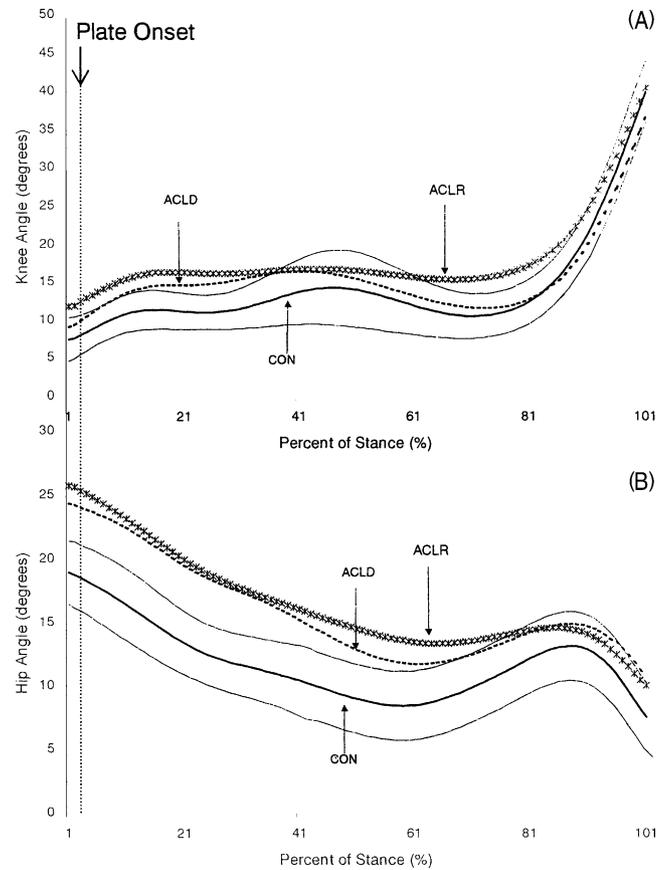


Fig. 3. Knee (A) and hip (B) joint angles for anterior cruciate ligament deficient (ACLD), anterior cruciate ligament reconstructed (ACLR), and control (CON) subjects. Positive and negative values are flexed and extended positions. Solid thick line and thin lines are mean and 1 SD for CON, dashed thick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

generated during late stance (Table 3, Fig. 4B). The ACL reconstructed hip power curve generally paralleled the controls and ACL deficient curve but the ACL reconstructed hip produced significantly ($P < 0.05$) more

Table 2

Mean (SD) of knee and hip joint angles[†] for control (CON), anterior cruciate ligament deficient (ACLD), and anterior cruciate ligament reconstructed (ACLR) subjects ($n = 10$)

Phase (P)/ Point (Pt)	Stance partition					
	Knee			Hip		
	CON	ACLD	ACLR	CON	ACLD	ACLR
P1	8.53 (1.86)	10.23 (2.46)	12.23 (2.24)	18.74 (2.06)	24.15 (4.27)*	25.21 (3.00)*
Pt1	9.46 (1.56)	11.27 (2.87)	13.14 (3.34)	18.15 (2.34)	23.78 (4.28)*	24.65 (3.22)*
Pt2	10.47 (2.22)	12.96 (3.84)	14.54 (3.69)	17.55 (2.18)	23.12 (4.04)*	23.69 (3.41)*
P2	11.76 (2.00)	14.89 (3.67)	15.45 (5.23)	14.02 (2.04)	19.65 (3.00)*	20.01 (3.90)*
Pt3	13.67 (2.44)	16.52 (2.29)	16.77 (2.22)	12.84 (2.00)	15.66 (4.22)*	16.51 (4.22)*
P3	12.92 (2.16)	15.01 (2.34)	15.38 (5.71)	9.31 (1.35)	13.36 (3.18)*	14.43 (2.81)*
Pt4	11.9 (2.32)	13.49 (2.04)	14.78 (5.46)	7.96 (1.44)	11.71 (2.44)*	13.71 (2.06)*
P4	10.74 (2.33)	12.56 (4.56)	14.76 (5.09)	10.09 (1.29)	12.93 (2.13)	13.74 (1.94)
Pt5	11.61 (2.47)	13.09 (2.74)	15.52 (3.08)	11.21 (1.48)	13.71 (3.77)	14.30 (2.92)
P5	22.23 (2.69)	21.78 (2.45)	24.28 (3.57)	11.31 (5.44)	13.85 (3.44)	13.86 (3.52)

[†] Positive values indicate flexion, negative values indicate extension (°).

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

Table 3

Mean (SD) of knee and hip joint powers[†] for control (CON), anterior cruciate ligament deficient (ACLD), and anterior cruciate ligament reconstructed (ACLR) subjects ($n = 10$)

Phase (P)/ Point (Pt)	Stance partition					
	Knee			Hip		
	CON	ACL D	ACLR	CON	ACL D	ACLR
P1	-0.42 (0.24)	-0.41 (0.15)	-0.37 (0.19)	0.56 (0.22)	0.39 (0.16)	0.71 (0.15)*.#
Pt1	-0.66 (0.45)	-0.76 (0.25)	-0.60 (0.31)	0.90 (0.27)	0.78 (0.45)	1.22 (0.55)*.#
Pt2	-0.30 (0.17)	-0.28 (0.11)	-0.29 (0.17)	0.79 (0.26)	0.67 (0.35)	0.98 (0.13)*.#
P2	-0.01 (0.07)	-0.03 (0.02)	-0.05 (0.07)	0.61 (0.21)	0.57 (0.26)	0.63 (0.25)
Pt3	0.15 (0.17)	-0.06 (0.77)	-0.01 (0.10)	0.05 (0.15)	-0.21 (0.24)	0.12 (0.12)
P3	-0.07 (0.10)	-0.27 (0.16)	-0.12 (0.11)	0.08 (0.12)	-0.12 (0.15)	-0.01 (0.14)
Pt4	-0.13 (0.15)	-0.05 (0.05)	-0.09 (0.07)	0.33 (0.22)	0.16 (0.13)	0.16 (0.14)
P4	-0.19 (0.12)	-0.16 (0.14)	-0.06 (0.12)	0.25 (0.17)	0.37 (0.15)	0.26 (0.13)
Pt5	-0.49 (0.26)	-0.16 (0.15)	-0.21 (0.17)	0.11 (0.17)	0.37 (0.24)	0.25 (0.19)
P5	-0.26 (0.15)	-0.10 (0.08)	-0.12 (0.18)	0.05 (0.09)	0.10 (0.05)	0.23 (0.15)*

[†] Positive values indicate power generation; negative values indicate power absorption (W/kg).

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

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

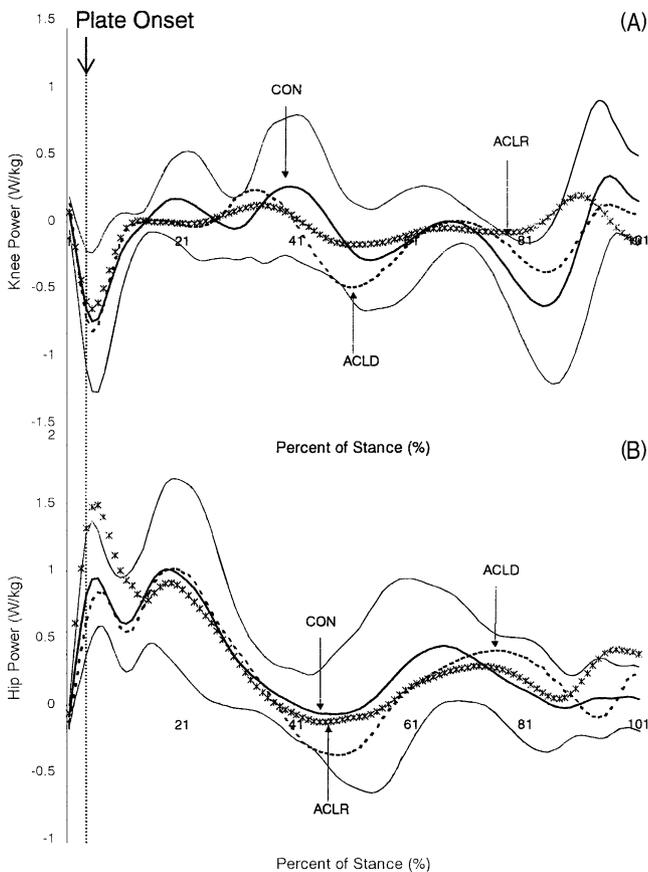


Fig. 4. Knee (A) and hip (B) joint powers for anterior cruciate ligament deficient (ACLD), anterior cruciate ligament reconstructed (ACLR), and control (CON) subjects. Positive and negative values are energy generation and absorption by the muscles. Solid thick line and thin lines are mean and 1 SD for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

power during early stance compared to controls and pre-surgical values (P1–Pt2; Table 3, Fig. 4B).

4. Discussion

The purpose of this study was to determine the effect of unexpected FP during gait on lower extremity joint mechanics in chronic ACL deficient subjects prior to and 3 months following surgical repair compared to healthy controls. It was hypothesized that as a protective response, the pre-surgical injured limb would exhibit a greater knee extensor moment compared to healthy controls in response to the FP. In support of the hypothesis, the ACL deficient subjects demonstrated a significantly greater knee extensor moment near mid-stance as compared to the control group (Fig. 2A). These data suggest that chronic ACL deficient subjects rely more heavily on knee extensor musculature to prevent vertical collapse in response to an unexpected perturbation compared to healthy individuals.

Previous investigations have demonstrated that ACL deficient subjects demonstrated a quadriceps avoidance pattern during non-perturbed gait and not while performing activities that place relatively large demands on knee extensor muscles compared to normal walking (Berchuck et al., 1990; Rudolph et al., 1998). In addition, Rudolph et al. (1998) suggested that knee control strategies become more pronounced as the demand on the knee increases. Berchuck et al. (1990) reported that chronic ACL deficient subjects did not exhibit a quadriceps avoidance gait pattern while running or ascending stairs. They suggested that the increased demand of the quadriceps muscles during these activities prohibited a quadriceps avoidance gait pattern. However, the ACL deficient subjects in that study exhibited a significantly greater knee extensor moment during stair ascent compared to controls, suggesting a possible compensatory response to ACL deficiency (Berchuck et al., 1990). Ferber et al. (2002) tested the response of healthy sub-

jects to an unexpected FP and postulated that the FP was an activity that placed a relatively large demand on the knee joint extensor muscles to maintain vertical support compared to non-perturbed gait. The results of this investigation support those of Berchuck et al. (1990) and Rudolph et al. (1998) and suggest that ACL deficient subjects do not reduce or avoid vigorous contraction of the quadriceps muscles during activities that place relatively large demands on knee extensor muscles.

It was hypothesized that the ACL deficient injured limb would exhibit a greater hip extensor moment compared to healthy controls in response to the FP. Contrary to this hypothesis, no significant differences were observed in hip moment or power production patterns between ACL deficient and control subjects during stance (Figs. 2B and 4B). Previous studies have suggested that ACL injury leads to neuromuscular reprogramming of the knee and hip muscles that stabilize the knee to prevent injury during gait (Andriacchi, 1983; Berchuck et al., 1990; Birac et al., 1991; Devita et al., 1997; Ferber et al., 2002; Roberts et al., 1999; Rudolph et al., 1998; Wexler et al., 1998). In addition, Ferber et al. (2002) suggested that the primary role of the hip muscles in healthy subjects was to maintain control of the upper body during the FP. It is possible that the neuromuscular alterations due to ACL injury may not manifest themselves in the hip joint muscles in response to an unexpected FP since their primary role was to maintain control of the upper body.

It was hypothesized that the ACL deficient subjects would demonstrate a more flexed position at the knee and hip as a protective response to the unexpected FP. In support of the hypothesis, the ACL deficient subjects exhibited greater knee and hip joint flexion throughout most of stance in response to the unexpected FP. A more flexed knee joint position possibly necessitated a greater net knee extensor moment to prevent vertical collapse. Devita et al. (1997) hypothesized that a more flexed position demands a greater knee extensor moment to prevent collapse of the body during the stance phase of non-perturbed gait. The results from this investigation support those of Devita et al. (1997) and suggest ACL deficient subjects respond to unexpected perturbations by assuming a more flexed knee and hip position to prevent collapse in the response to the FP.

Following surgical repair, it was hypothesized that the ACL repaired limb would demonstrate a sustained knee extensor moment in response to the FP. In support of this hypothesis, the ACL repaired group demonstrated a net zero knee moment during Phase 2 of early stance and a sustained knee extensor moment for the remainder of stance (Fig. 2A). The net zero knee moment observed during early FP stance was most likely the result of co-activating knee extensor and flexor muscles in response to the unexpected FP. Thus, these muscles produced near equal torque at the knee joint

and possibly increased knee joint stability during the FP. During early FP stance, the ACL repaired subjects also demonstrated relatively little knee power production (Fig. 4A) and a negligible change in knee angle (Fig. 3A). These data also support the premise that opposing knee muscles were co-activated possibly to maintain knee joint stability during early FP stance.

During the remainder of stance, the ACL repaired group demonstrated a significantly greater and sustained knee extensor moment throughout most of stance as compared to controls (Fig. 2A). The ACL repaired subjects appeared to react to the FP by producing a sustained knee extensor moment, possibly due to increased quadriceps activity, and produced a relatively static knee angle in an effort to stabilize the knee. Pre-surgically, these subjects exhibited a significantly greater knee extensor moment and this pattern was more apparent following surgery. These data suggest that following reconstructive surgery, ACL repaired subjects continue to rely heavily on knee extensor musculature in response to an unexpected FP.

It was hypothesized that the ACL repaired subjects would exhibit a greater hip extensor moment and greater hip and knee flexion. In partial support of this hypothesis, the hip generated significantly more power (Fig. 4B) but no differences in hip moment values were observed (Fig. 2B) during early FP stance as compared to control and pre-surgical values. In addition, the ACL repaired subjects exhibited significantly more hip flexion during early and mid-stance as compared to controls (Fig. 3B). With no change in the hip moment, an increase in hip power production must result from a greater angular velocity that can be observed in Fig. 3B. Ferber et al. (2002) reported that the muscles surrounding the hip are most important in maintaining control of the upper body and preventing collapse as an initial response to the FP. Since the ACL repaired subjects were already in a more flexed hip position, it is possible that a greater angular velocity of hip flexion and concomitant greater power generation was necessary to control the upper body in response to the FP.

Torry et al. (2000) 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 investigations. The ACL reconstructed subjects in the present study exhibited no to minimal knee joint effusion but it is possible that some effusion was present but was not noticeable to the principal investigator. However, Torry et al. (2000) 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. (2000). 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.

Specific limitations and delimitations in this study are recognized. Biomechanical adaptations to chronic ACL injury and reconstructive surgery depend on several factors including rehabilitation protocol, patient compliance, and surgical procedure. The present results may apply only to chronic ACL injured individuals who demonstrate similar characteristics as patients involved in this study. As well, the subjects involved in the present investigation would be classified as non-copers since each reported at least one episode of giving way prior to surgery (Rudolph et al., 1998). ACL injured subjects who are able to cope with their injury may not respond to an unexpected perturbation in a similar manner. It is also possible that since each participant was tested immediately prior to and 3 months following surgery, a learning effect of the perturbation response may have been present. However, the relatively long period of time between testing sessions reduced this possibility. In addition, it is possible that the subjects anticipated the FP condition. However, it has been reported that healthy controls, when undergoing a similar experimental protocol, did not demonstrate any significant differences between the randomized non-perturbation trials and a set of 12 blocked non-perturbation trials (Ferber et al., 2002). Subjects were also not allowed to practice the FP trials and the backward and non-perturbation trials, along with the random order of trials, were used to help prevent possible accommodation and anticipation of the FP condition.

4.1. *Clinical relevance*

ACL injured subjects exhibited a significantly greater knee extensor moment prior to and following reconstructive surgery compared to healthy controls. This pattern suggests that quadriceps-induced excessive anterior tibial translation may be potentiated in response to an unexpected FP. It is suggested that clinicians focus on knee flexor strengthening to prevent possible re-injury if ACL injured subjects are exposed to an unexpected perturbation. Following surgery the ACL subjects in this study demonstrated a near net zero knee moment in response to the FP during early stance. This strategy was indicative of knee stiffening possibly to increase knee joint stability. Knee joint stiffening may be a beneficial strategy to avoid injury in response to an unexpected FP. However, this response was significantly different from the reaction of the uninjured subjects and may represent an accommodation to surgery. Since the ACL repaired subjects exhibited lower extremity joint kinematic and kinetic patterns significantly different from healthy controls and pre-surgical values, a “nor-

mal” reactive gait pattern had not yet been established 3 months following surgery. Future investigations involving ACL repaired subjects 6 months following surgery may demonstrate that FP reactive patterns more similar to controls have been established.

It is also recommended that clinicians focus on perturbation training to restore response patterns with the goal of preventing re-injury as a result of unexpected perturbations. Chmielewski et al. (2002) investigated the effect of a perturbation training program on the walking patterns of acute ACL injured individuals. It was reported that perturbation training enhances dynamic knee stability during non-perturbed gait by altering the coordinative patterns of muscles that affect anterior tibial translation. Future research involving perturbation training in chronic ACL deficient and surgically repaired subjects could determine whether this intervention strategy would restore normal joint reactive patterns and whether or not these new reactive patterns prevent re-injury.

5. Conclusions

The ACL deficient subjects demonstrated a greater knee extensor moment than the controls in response to an unexpected FP that may have been necessary to prevent collapse. Following surgery, these subjects produced a sustained knee extensor moment and more flexed knee hip joint kinematic patterns during the stance phase of perturbed gait. These data suggest that the ACL injured subjects did not respond to an unexpected perturbation in a manner similar to healthy controls prior to or following surgical repair.

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