GAIT PERTURBATION RESPONSE IN ANTERIOR CRUCIATE LIGAMENT DEFICIENCY AND SURGERY

by

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A DISSERTATION

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The purpose of this study was to investigate the gait patterns in persons with chronic ACL deficiency (ACLD: n=10) who subsequently undergo surgical repair, and to determine how these individuals respond to an unexpected forward perturbations (FP) compared to healthy controls (n=10). An unexpected FP was applied using a moveable force plate imbedded in a walkway. During non-perturbed (NP) gait, ACLD subjects exhibited similar knee moment patterns compared to healthy adults but appeared to accommodate to ACLD through alterations of hip and ankle joint kinematic, kinetic, and muscle power patterns. Three months following surgery, these same subjects demonstrated a significantly different knee moment pattern and were significantly more flexed at the knee and hip during NP gait. 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 surgery significantly alters lower extremity gait patterns and that the re-establishment of pre-injury gait patterns takes longer than 3 months to occur.

When healthy adults experienced an unexpected FP, the hip was favored in maintaining control of the upper body and in preventing collapse. In response to the same FP, ACLD subjects demonstrated a hip moment pattern similar to controls but a greater knee extensor moment. The increased knee moment pattern was more prevalent 3 months following surgery. These data suggest that ACLD and ACLR subjects rely more on knee extensor muscles for the prevention of collapse when reacting to an unexpected FP.

Bilateral accommodations to ACL injury and surgery during NP and FP gait were also examined. During NP gait, healthy adults demonstrated asymmetrical hip moment and power patterns whereas ACLD and ACLR subjects exhibited symmetrical hip but asymmetrical knee mechanics. In response to the FP, healthy adults exhibited lower extremity joint symmetry but the ACLD and ACLR group exhibited asymmetrical knee moment and power patterns. These findings suggest that ACL injury and surgery result in bilateral joint accommodations and that when investigating ACL injured populations, bilateral control population data should be used in addition to non-injured limb data.

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CHAPTER I

INTRODUCTION

Normal knee joint movements are accomplished via an intricate balance between passive ligamentous and active muscular components to maintain knee stability and prevent injury. The anterior cruciate ligament (ACL) is a critical passive component to normal knee function which acts to resist anterior rotatory motion of the tibia relative to the femur (Norkin & Levangie, 1992). Injury to the ACL is among the most frequent sports-related injuries. Some studies have estimated that 1 per 3000 people suffer ACL tears annually in the United States with an estimated cost for these injuries of almost a billion dollars per year (Fu et. al., 1999; Griffin et. al., 2000). Recent epidemiological data suggest that injury to the ACL alone accounts for 59% of all knee ligament injuries, with 70% of ACL injuries occurring in non-contact situations (Bollen, 2000).

Once the ACL is injured, the patient can experience knee joint instability due to the lack of ligamentous restraints and reduced joint position sense (Andriacchi et al., 1993; Berchuck et al., 1990; Cicotti et al., 1995; Lass et al., 1991; Tibone et al., 1993). Some patients are able to cope with the injury by re-educating the surrounding knee muscles to stabilize the knee in the absence of the ACL, while other patients require surgical intervention to replace the ruptured ligament with replacement tissue.

Often, the functional outcome among ACL deficient (ACLD) patients is related to the type of muscular and neural adaptations that occur in lower extremity muscles. It has been hypothesized that muscular accommodations and alterations in gait biomechanics, due to ACLD, results from of reprogramming of the locomotor process so that excessive anterior displacement of the tibia is prevented (Andriacchi et al., 1993; Berchuck et al., 1990; Cicotti et al., 1995; Devita et al., 1997, 1998; Hurwitz et al., 1997; Lass et al., 1991; Osternig et al., 2000; Tibone et al., 1993; Yack et al., 1994).

Adaptations to ACL Deficiency

It has been suggested that time since injury may play an important role in the type of gait adaptation observed in ACLD patients. Investigations involving acute (< 1yr) ACLD subjects are limited (Devita et al., 1997) and suggest that during the stance phase of gait, ACL injured limbs exhibit a sustained knee extensor moment compared to non-injured control subjects. Results from investigations involving chronic (> 2yr) ACLD subjects differ significantly from acute and suggest that chronic ACLD subjects develop a sustained knee flexor moment throughout stance (Andriacchi et al., 1993, Berchuck et al., 1990; Birac et al., 1991, Wexler et al., 1998). This type of gait pattern has been interpreted as a tendency to avoid or reduce the demand placed on the quadriceps and termed a "quadriceps avoidance gait" possibly serving to reduce anterior tibial shear. Birac et al. (1991) reported that 88% of subjects who were at least 6 years post-injury demonstrated the avoidance pattern compared to only 44% of subjects injured less than 1.5 years. Wexler et al. (1998) reported that 57% of chronic ACLD patients demonstrated a quadriceps avoidance gait pattern.

Other investigators have been unable to reproduce the quadriceps avoidance phenomenon in chronic ACLD patients (Roberts et al., 1999; Rudolph et al., 1998). Rudolph et al. (1998) investigated ACLD individuals and reported that no subject exhibited a quadriceps avoidance pattern but that the injured knee demonstrated a reduced extensor moment at peak knee flexion angle compared to the uninjured knee. Roberts et al. (1999) investigated chronic ACLD subjects for the specific purpose of corroborating the quadriceps avoidance pattern. No ACLD patient demonstrated a sustained knee flexor moment, a decreased knee extensor moment, or a reduction in quadriceps electromyographic (EMG) activity during the stance phase of gait, regardless of time since injury. Roberts et al. concluded that quadriceps avoidance, as a gait adaptation in ACLD patients, might be less common than previously reported.

Adaptations to ACL Reconstructive Surgery

Early clinical studies reported that approximately one-third of ACLD 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 (Noyes et al., 1983). Reconstructive surgery is sometimes used to reestablish functional and mechanical stability of the knee in those ACLD patients who experience changes in lifestyle, episodes of giving-way, or joint instability. However, factors such as the type of surgery, patient characteristics, compliance with rehabilitation protocols, and type of ACL reconstruction (ACLR) may all play significant roles in the type of gait pattern developed following surgery.

Investigations involving ACLR subjects are limited and suggest that time since surgery may play an important role in the return of normal gait patterns (Bulgheroni et al., 1997; Bush-Joseph et al., 2001; Cicotti et al., 1994; Devita et al., 1998; Ernst et al., 2000; Timoney et al.,

1993). Devita et al. (1998) reported that ACLR subjects examined 3 weeks post-surgically demonstrated a sustained knee extensor moment and a reduced but prolonged hip extensor moment pattern. However, at 6 months following surgery, the same ACLR subjects exhibited knee and hip moment patterns more similar to control group values, suggesting that ACLR subjects can regain pre-injury gait characteristics. It was also reported that, 8 months after surgery, ACLR subjects exhibited only slight reductions in the peak knee extensor moment during gait (Bush-Joseph et al., 2001). However, Timoney (1993) reported that, at 10 months post-surgery, ACLR subjects walked with a significantly reduced knee extensor moment compared to control subjects, suggesting that not all patients demonstrate a return of normal gait patterns following ACL reconstructive surgery during the first year.

The time between injury and surgery may also influence the type of gait pattern observed in ACLR subjects. Few comprehensive gait studies have investigated ACL injured subjects prior to and following surgical repair (Devita, 1997). Devita (1997) examined the gait patterns of ACL injured subjects before surgery (2 weeks after ACL injury) and at 3 and 5 weeks post-surgically. It was demonstrated that ACLR subjects exhibited a sustained knee extensor moment and a significantly reduced and prolonged hip extensor moment throughout stance both 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, ACL injured subjects involved in this investigation were acutely injured ACL patients who also exhibited a sustained knee extensor moment pre-surgically. It is therefore not known whether the post-surgical gait pattern exhibited by the ACLR subjects resulted from ACL injury, reconstructive surgery, or both. It is possible that subjects who had sustained ACL injury 2 or more years prior to surgery developed different gait patterns following surgery as the mechanical stress of ACL reconstructive surgery may cause the development of distinctive gait patterns. Additional studies

are needed to better understand the neurological and mechanical influences that ACLD and subsequent surgical repair have on the development of gait patterns. Furthermore, few studies have investigated the effects of unexpected gait perturbations on ACLD and ACLR individuals.

Response to Unexpected Gait Perturbations

It has been demonstrated that standing perturbations produce characteristic distal to proximal lower extremity muscle activity sequences in normal individuals (Nashner, 1980; Tang et al., 1998). Few investigations have been performed to determine muscle activation patterns during unexpected gait perturbations (Brady et al., 2000; Dietz et al., 1984; Eng, 1994; Gollhofer, 1986; Nashner, 1980, Tang et al., 1998). In these studies, the results indicated that a reactive strategy to gait perturbations in healthy individuals was to generate distal to proximal muscle activity patterns as well as longer burst duration and higher magnitude of muscle EMG activity as compared to the unperturbed condition (Nashner, 1980; Tang et al., 1998). Furthermore, it has been shown that individuals subjected to an unexpected forward slip perturbation during normal gait exhibited a significantly longer stride duration, a longer stride length, and increased ankle plantarflexion, knee extension, hip extension, and trunk extension as compared to normal gait kinematic patterns (Tang et al., 1998). However, no studies have calculated the joint moments associated with the unexpected slip perturbations or examined the kinematic, kinetic, or muscle activation patterns of ACLD subjects. Although kinematic information describes the movement of the body, it provides little insight into the cause of the movement since movement of a particular joint result from muscle contraction at that particular joint or contraction at other joints. Information derived from muscle EMG recordings can also provide additional insight into the cause of body movement. However, EMG recordings only provide information regarding

superficial muscle contractions and cannot reveal whether a muscle is contracting concentrically or eccentrically. Calculation of joint moments and power production provide more direct measures of joint and muscle actions and coordination between lower extremity joints.

Purpose of the Study

It has been suggested that specific gait adaptations as a result of ACL injury do occur and that these adaptations may depend on the time since injury and surgical repair. Timely reactive balance adjustments are particularly important during locomotion, as individuals rarely encounter level walking surfaces free of obstacles or perturbations. Therefore, the purpose of this study was twofold: 1) to test ACLD subjects prior to and 3 months following reconstructive surgery to determine how normal gait patterns may change as a result of chronic ACLD and subsequent surgical repair and 2) to determine the effect of an unexpected forward gait perturbation on lower extremity joint moments in ACLD subjects prior to and following reconstructive surgery as compared to healthy controls.

Four specific research questions were addressed in this study:

1. What was the effect of unexpected gait perturbations on lower extremity joint moments and muscle EMG patterns in healthy subjects?

2. What was the effect of chronic ACLD on the normal walking and in response to unexpected gait perturbations?

3. What was the effect of ACL surgery on the normal walking patterns and in response to unexpected gait perturbations?

4. What was the effect of ACL injury and subsequent surgery on bilateral joint accommodations?

One experiment was conducted to answer these questions. While subjects walked across a walkway, a forward perturbation was applied upon heel strike. Simultaneous measurements of lower extremity joint moments, powers, and kinematics and muscle EMG were recorded. Subjects consisted of healthy uninjured individuals and chronic ACLD subjects who were scheduled for surgical repair. The same ACLD surgical subjects were then reassessed three months post-operatively.

Bridge

The research question for the first study was: What is the effect of unexpected gait perturbations on lower extremity joint moments and muscle EMG patterns in healthy subjects? To answer this question, reactive balance responses to an unexpected forward perturbation were investigated in healthy uninjured young adults.

Chapter II summarizes the reactive balance responses of 10 young adults to unexpected forward perturbations during normal gait. This study provides the baseline body of knowledge regarding lower extremity joint moments, powers, angles, and lower extremity muscle EMG activation patterns for further comparison to injured ACL patients.

CHAPTER II

REACTIVE BALANCE ADJUSTMENTS TO UNEXPECTED PERTURBATIONS DURING HUMAN WALKING

Introduction

Human gait is the most common form of human movement, yet the underlying neurological and biomechanical processes by which movement occurs are complex and individualistic. Walking involves the integration of muscular contractions across different joints in an effort to initially lose, then regain dynamic equilibrium as the body is propelled through space. The inherent instability involved in human locomotion results from a relatively small base of support, long single support phase, and the observation that 2/3 of the body's mass is located in the head-arm-trunk (HAT) segments (Winter et al., 1990a).

Successful locomotion requires three essential elements: (a) the ability to generate and maintain fundamental locomotor patterns appropriate for moving toward an intended destination, (b) maintenance of basic dynamic equilibrium between a shifting center of mass (COM) and a constantly changing base of support (BOS), and (c) the ability to change locomotor patterns in response to external or internal inertial changes that threaten dynamic equilibrium (Shik & Orlavsky, 1976). While the first element is concerned with the generation of complex locomotor

patterns, the latter two are critical in the detection of potential threats to balance and the subsequent reaction to either foreseen or unexpected perturbations during normal gait.

Humans rarely encounter level walking surfaces free of obstacles or perturbations. The ability of an individual to react to external perturbations is critical in the prevention of injury due to joint trauma and/or falls. Patla (1993) suggested that two control mechanisms are necessary for the maintenance of dynamic equilibrium during gait: proactive and reactive. Proactive mechanisms can be defined as those acting in advance of a particular gait event to accommodate for potential forthcoming perturbations. During normal gait, proactive control is integrated within the locomotor pattern such that minimal disruptions in balance occur and the large HAT segment is maintained in an upright posture throughout the gait cycle. Proactive control mechanisms are also used when foreseen obstacles are presented in the path of an individual so that they may be avoided.

Often, individuals cannot anticipate external threats to dynamic equilibrium during gait and reactive mechanisms are required to act after the person experiences an unexpected perturbation. Examples of unexpected external perturbations include slipping on ice or stepping on an unstable surface. It has been demonstrated that reactive mechanisms serve to stabilize the body via feedback primarily from the somatosensory and vestibular systems (Nashner, 1980; Tang et al., 1998).

Relatively few investigations have studied reactive postural adjustments during gait in response to unexpected perturbations (Brady et al., 2000; Dietz et al., 1984; Eng, 1994; Gollhofer, 1986; Nashner, 1980, Tang et al., 1998). Nashner (1980) incorporated a moveable platform into a walkway to simulate unexpected perturbations during gait. Forward/backward translation, upward/downward rotation, and elevation/depression of the platform were employed as the external perturbation. Each perturbation was imposed at heel strike (HS), start of single support,

mid-stance (MS), or at the beginning of double support phase. Electromyographic (EMG) recordings from the gastrocnemius (GAS) and tibialis anterior (TA) muscles were measured along with lower extremity joint angles. Unexpected forward translation or downward rotation perturbations applied at HS produced increased TA EMG activity in the perturbed leg and unexpected backward translation or upward rotation produced increased GAS EMG activity. These recordings were similar to those obtained during standing perturbations (Nashner, 1977, 1980). Based on the observation that platform perturbations resulted in altered foot trajectory and stretching of the muscle, Nashner (1980) hypothesized that alterations in distal limb trajectories provided the principal sensory feedback input to the central nervous system to elicit reactive balance control strategies.

Dietz et al. (1984) supported the theory of distal limb control of dynamic equilibrium using a treadmill paradigm wherein unexpected perturbations were provided via a sudden acceleration or deceleration of a treadmill upon which subjects walked. Acceleration or deceleration of the treadmill at HS resulted in increased TA and GAS EMG activity, respectively, and deviation in ankle joint trajectory away from its normal path.

Using a similar paradigm to Nashner (1980), in that subjects were subjected to unexpected forward perturbations using a moveable platform, Tang et al. (1998) hypothesized that proximal muscles (hip and trunk) may play as important a role as do distal muscles (TA and GAS) in balance recovery. Tang et al. (1998) further hypothesized that muscles that contribute more to reactive balance control would demonstrate consistent activation patterns over repeated trials, earlier onset latencies, longer burst durations, and greater burst magnitudes of response. EMG recordings were taken from the TA, GAS, rectus femoris (RF), biceps femoris (BF), rectus abdominis (RA), and erector spinae (ES). Results indicated that hip and trunk muscles (RA, ES) did not play a significant role in reactive balance adjustments during perturbed gait at HS since

these muscles did not demonstrate more consistent activation, earlier onset latency, longer burst duration, or larger burst magnitude compared to distal leg and thigh muscles. Leg and thigh muscles (TA, GAS, RF, BF) did, however, demonstrate earlier onset, high magnitude, and relatively long duration of activity to satisfactorily attenuate the perturbation. It was therefore concluded that activity from leg and thigh muscles was the key to reactive balance control.

This theory of distal muscle control, however, has been disputed by other investigators (Winter, 1987, 1990a) who have espoused that proximal muscle activity is critical during gait for control of the HAT segment. Winter (1987, 1990a) estimated that an ankle moment about 8 times greater than that of the hip is needed to control the HAT segment due to the combined moments of inertia of the rest of the body. Furthermore, it was suggested that during early stance, as the HAT segment accelerates anteriorly, the ankle does not intervene but undergoes a small dorsiflexor moment, and it is the large hip extensor moment that serves to directly control the displacement of the HAT segments to maintain dynamic equilibrium. Winter (1987) also documented high intra-subject and inter-subjects variability of the hip moments across trials and testing times. It was postulated that the high variability in hip moments was necessary on a stride-to-stride basis in an attempt to control the HAT segment and that changes in hip moment patterns were equally matched by alterations in knee moment patterns. Winter (1987) hypothesized that such a deterministic trade-off between the hip and the knee indicated a stride dependent control of the HAT segment to maintain the total moment of support to prevent the body from collapsing due to gravitational forces.

There is controversy surrounding the postural control mechanisms in response to changes in dynamic equilibrium during gait and a paucity of literature serving to explain reactive balance mechanisms to unexpected perturbations. Of those investigations, only EMG and kinematic data have been presented and no studies have calculated the joint moments necessary for

understanding the relative joint contributions in maintaining dynamic equilibrium. Therefore, the purpose of this investigation was to determine the effect of unexpected forward perturbations during gait on lower extremity joint moments and muscle EMG patterns in healthy subjects.

It was hypothesized that the forward perturbation would result in a greater knee and hip joint flexion and greater ankle plantarflexion. As a result of the alterations in lower extremity joint positions, it was also hypothesized that the perturbed limb would demonstrate a greater knee and hip extensor moment and a reduced ankle plantarflexion moment and increased ankle, knee, and hip joint extensor muscle EMG activity.

Method

Subjects

Ten (5 males and 5 females) healthy young adults participated in the study. The mean age, body weight, and body height of subjects were 24.4 yr (± 3.1 yr), 67.2 kg (± 10.7 kg), and 170.1 cm (± 9.3 cm), respectively. All subjects were physically active, participating in regular activity at least 3 times per week. No subject had a prior history of lower extremity infirmity or pathology, or was suffering from any osteoarthritic or musculoskeletal disease at the time of testing that may have affected the ability to perform the experiment. Prior to participation, each subject signed a consent form (APPENDIX A) approved by the Human Subjects Compliance Committee at the University of Oregon.

Experimental Apparatus

All data were collected in the Motor Control Laboratory at the University of Oregon. Equipment available included a custom built platform system consisting of a hydraulically driven moveable force plate (0.61 m long x 0.17 m wide) incorporated into the center of a 5 m long wooden walkway (FIGURE 2.1). Translational force plate movement of 10 cm at a velocity of 40 cm/s was used to produce the unexpected forward perturbation. 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 & Lanshammar, 1981). A downward force on the force plate due to heel contact triggered the perturbation.

The force plate was equipped with a set of strain gauges mounted underneath the four corners of the plate to measure the vertical (Fz), horizontal antero-poterior (Fx), and medio-lateral (Fy) ground reaction forces. Using a feedback electric circuit, the Fz forces also served as trigger signals to initiate the force plate movement when the signal registered approximately 40 N (~ 8% of body weight or less). Because of the time required for the hydraulics system to propel the force plate, a delay of 20-40 ms occurred between actual registering of the Fz force and movement of the force plate. Thus, the true force plate onset time was determined using a custom written computer program in later analysis. During the forward perturbation condition, onset of force plate movement occurred at 3.1 ± 0.2% of stance (approximately 29.10 ± 0.19 ms after heel strike) and ended at 59.8 ± 2.5% of stance (approximately 543.21 ± 0.24 ms after onset).

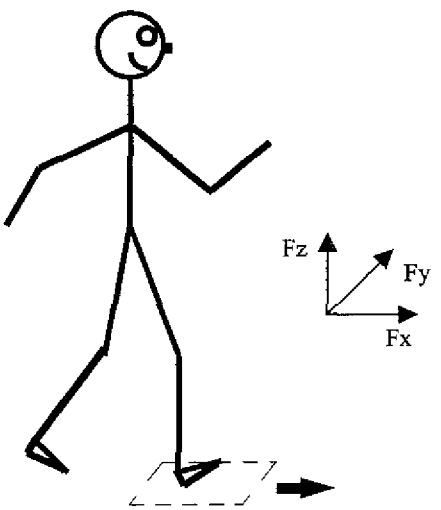

FIGURE 2.1 Schematic diagram of the setup of the moveable force plate on the walkway. The arrow indicates forward direction of force plate movement triggered by heel contact. The coordinate convention for the force plate is shown to the right of the figure.

Protocol

Subjects walked along a 5 m wooden walkway in which the moveable 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 EMG, joint kinematic and kinetic data were collected for a 5 sec period, which included the step prior to and following contact with the force plate, while the subjects walked along the walkway. Data were recorded from 48 trials using the subject's right limb. The first 12 trials consisted of "true control" non-perturbations (NP) trials to establish normal walking muscle activation patterns. Following the NP true control trials, 36 additional trials were performed consisting of 12 forward perturbations (FP), 12 NP "catch" trials, and 12 backward perturbations (BP) randomly ordered to prevent anticipation of the FP. BP trials were applied over a distance of 5 cm at a velocity of 10 cm/s and were used to help prevent possible accommodation and anticipation of the FP condition. Subjects were not allowed to practice the perturbation trials. It was assumed that randomization of the FP, BP, and NP trials prevented possible accommodation and anticipation of the FP condition.

There was a small risk that the subjects could fall when their balance was perturbed. Using a relatively mild perturbation speed and displacement minimized this risk. The risk was further reduced by requiring the subjects to wear a harness attached to an overhead track and by providing a handrail to grasp along the entire walkway. This study was approved by the University of Oregon Committee for the Protection of Human Subjects (Protocol # C2-74-00F).

Instrumentation

EMG Data

EMG data were collected using bipolar surface electrodes (DE-02, Delsys, Mass.). Eight electrodes were placed on the skin overlying the muscle belly of the tibialis anterior (TA), medial head of the gastrocnemius (GAS), biceps femoris (BF), and vastus lateralis (VL) of each limb. To achieve an optimal EMG signal and low impedance (< 5 kΩ), three, 2 in^2 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. (1997). All raw EMG analog signals were on-line pre-amplified (x 7000), analog filtered (20-7000 Hz), and then converted into digital signals sampled at 1200 Hz for a 5-sec 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 4^{th} order dual-pass Butterworth filter. EMG data for the FP and catch NP conditions were normalized to maximum EMG activity produced during the true control NP condition for each phase or discrete point and expressed as the NP:FP ratio.

Kinetic Data

A 6-degree of freedom custom-built force plate equipped with strain gauges mounted underneath the four corners was used to measure Fz, Fx, and Fy ground reaction forces (Institute of Neuroscience Technical Service Group, University of Oregon). Kinetic data were recorded at 1200 Hz for a 5-sec duration via the AMLAB data acquisition system. Prior to analysis, kinetic data were low-pass filtered between 4-10 Hz using a 4^{th} order dual-pass Butterworth filter.

Selected filter frequencies were determined for each force signal based on specifications from the manufacturer.

Kinematic Data

Kinematic data were collected using the PEAK Performance Technologies Real-Time Data Acquisition System (Peak Performance Inc., Colorado, USA). Four cameras were positioned along the progression plane of the subject's gait path (4 m from the sagittal plane). The pre-determined criterion for tolerable error in space calibration was set at 0.2% (i.e., 2mm 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 register plate movement and serve as the point of reference for transformation of local center of pressure (COP) coordinates to global kinematic coordinates. Three-dimensional data were collected at 120 Hz for a 5 sec duration with each of the 4 cameras synchronized with the AMLAB data acquisition system. Each marker was then digitized for the entire collection period that included the stride before and after the stance phase on the force plate. The digitized 3-D position data for all markers were then low-pass filtered between 4-8 Hz using a 4^{th} 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.

Inverse Dynamics Calculations

FIGURE 2.2 represents the free body diagram used for calculation of the inverse dynamics equations. The magnitude of the segmental masses along with their moments of inertia were estimated using data reported by Dempster (1959) and individual subject anthropometric data. Center of pressure was calculated from the ground reaction force data within the force plate's local coordinate system. The kinematic reference marker applied to the force plate served to spatially coordinate the local kinetic coordinate system with the global kinematic coordinate system. Joint moments were calculated through an inverse dynamics analysis using a custom written MATLAB (The MathWorks, Inc., Massachusetts) computer program (INVDYN4.m) combining the anthropometric, kinematic, and kinetic data using the following equations (1), (2), and (3). Joint moments were expressed as a reaction moment to all external moments and represent the internal moment produced by the muscles crossing the joint. All joint moments were expressed as positive values for extensor and plantarflexor moments. A positive knee extensor moment would therefore represent a moment produced by the quadriceps muscles. Extensor angular impulse (EAI) was calculated from the positive area under the joint moment curve and quantified the total contribution of a joint moment toward forward propulsion. Joint powers were calculated as the product of the joint moments and angular velocities. Positive power indicated that the joint moment was produced from concentric muscle actions in which energy is generated, whereas negative power indicates that the joint moment was produced from eccentric muscle actions in which energy was absorbed.

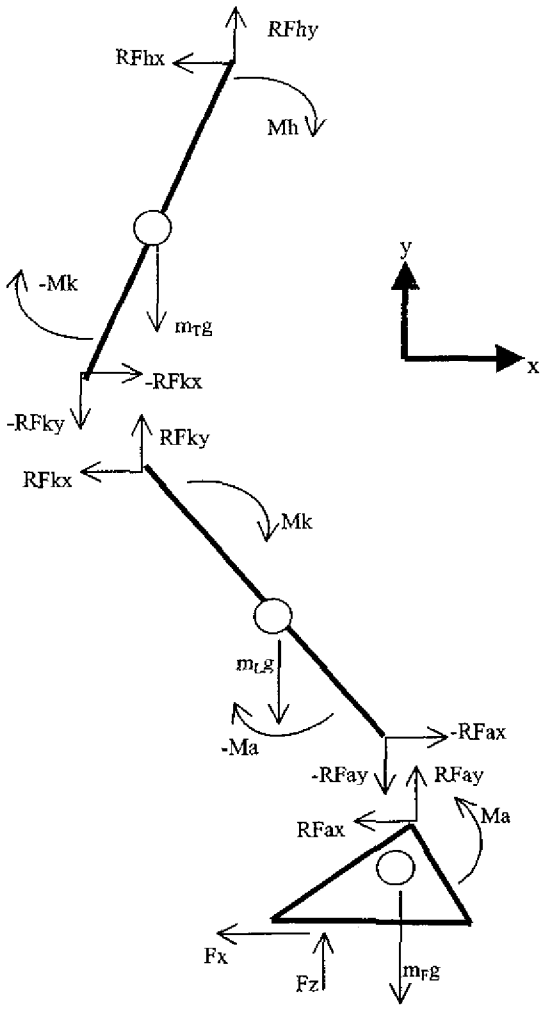

FIGURE 2.2 Freebody diagram of the lower extremity used for inverse dynamics calculations.

$M_a=(I_T*\alpha_F)+(F_y*CM_{Fx})+(F_x*(CP_y-CM_{Fy}))-(RF_{ay}*(CM_{Fx}-x_a))+(RF_{ax}*(CM_{Fy}-y_a))$ (1)

$M_k=(I_L*\alpha_L)+(-M_a)-(-RF_{ay}*(CM_{Lx}-x_a))+(-RF_{ax}*(CM_{Ly}-y_a))+(RF_{kx}*(CM_{Ly}-y_k))-(RF_{ky}*(CM_{Lx}-x_k))$ (2)

$M_h=(I_F*\alpha_T)-(-M_k)-(-RF_{ky}*(CM_{Tx}-x_k))-(-RF_{kx}*(CM_{Ty}-y_k))-(RF_{hx}*(CM_{Ty}-y_h))-(RF_{hy}*(CM_{Tx}-x_h))$ (3)

where

M – moment

I – moment of inertia of a segment

α - angular acceleration around the center of a segment

CM – center of mass

CP – center of pressure

RF – reaction force

Fx – anterior/posterior ground reaction force

Fz – vertical ground reaction force

g – gravity

F – foot

L – leg

T – thigh

a – ankle

k – knee

h – hip

Data Analysis

To obtain joint moment, kinematic, and EMG muscle activity measures of interest for the 12 FP, 12 "true control" NP block trials, and 12 NP catch trials, data analysis involved the following steps:

1. Partitioning of the stance phase of the gait cycle as defined from heel strike to toe off.
2. Interpolating joint (ankle, knee, hip) moment, power, position, and muscle (TA, GAS, BF, VL) EMG as percent stance phase for each condition.
3. Averaging the 12 trials for each condition into an ensemble average.
4. Dividing each ensemble average into 5 phases and selection of 5 discrete points according to discrete kinetic events determined from vertical and anterior/posterior ground reaction forces (FIGURE 2.3). Phase 1 (P1) was from heel strike to initial loading (Pt1), phase 2 (P2) was from Pt1 to first acceptance of full body weight (Pt3), phase 3 (P3) was from Pt3 to mid-stance (MS), phase 4 (P4) was from MS to second acceptance of full body weight (Pt5), and phase 5 (P5) was from Pt5 to toe off. Two other discrete points (Pt2, Pt4) were selected at the troughs between Pt1 and Pt3 and between Pt3 and Pt5.
5. Comparisons of each of the 5 phases and 5 discrete points for differences in average joint moments, powers, positions, and muscle EMG activity for the FP trials against the NP trials.

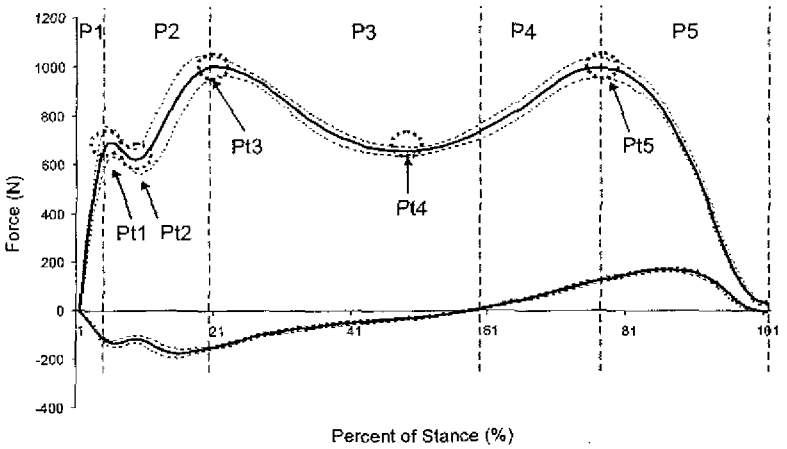

FIGURE 2.3. Selection of 5 discrete points (dashed circles) and partitioning the stance phase into 5 phases (dashed vertical lines) according to discrete vertical and anterior/posterior kinetic events.

Statistical Analysis

Two-way repeated measures ANOVAs (10 x 3) were used to determine differences, if any, between the three conditions,. The independent variables were 1) the 5 phases and 5 discrete points of stance and 2) condition (true control NP, catch NP, and FP). The dependent variables were joint 1) moment (ankle, knee, hip), 2) power, 3) position, and 4) muscle EMG magnitude (TA, GAS, BF, VL). A priori post-hoc tests were then performed to detect differences, if any, between conditions. A maximum α level of 0.05 was used to indicate statistical significance.

Results

This study was conducted to investigate the effect of unexpected FP during gait on lower extremity joint kinematics, moments, powers, and muscle EMG patterns in healthy subjects. In this section, descriptive measures of stance are presented first followed by lower extremity joint moments, joint kinematics, joint powers, and muscle EMG responses for 3 general aspects of stance: 1) early stance from heel strike to Pt3 including P1, Pt1, Pt2, and P2, 2) mid-stance from Pt3 to Pt5 including Pt3, P3, Pt4, and P4, and 3) late stance from Pt5 to toe-off including Pt5 and P5.

Descriptive Measures of Stance

Time

During the NP condition total time of stance (863.32 ± 77.27 ms) was significantly ($p<0.05$) less than the FP condition (977.59 ± 58.33 ms) and mid-stance occurred significantly ($p<0.05$) earlier (49.6 ± 2.4% of total stance) than FP (59.8 ± 2.6% of total stance). Total time of the 5 phases and location of the 5 discrete points is summarized in TABLE 2.1.

TABLE 2.1. Total Time and Percent of Stance of the 5 Phases and Location of the 5 Discrete Points during Stance Phase of Non-Perturbation (NP) and Forward Perturbation (FP) Conditions (n=10)

Stance Partition	NP		FP	
Phase (P)/ Point(Pt)	Time (ms)	Percent of Stance (%)	Time (ms)	Percent of Stance (%)
P1	0.00 - 51.78±9.84	0.00 - 6.34±1.21	0.00 - 48.85±11.10	0.00 - 5.02±1.33
Pt1	51.78±9.84	6.34±1.21	48.85±11.10	5.02±1.33
Pt2	86.30±11.39	10.22±1.32	87.93±15.04	9.64±1.54
P2	51.78 - 181.23±14.07	6.34 - 21.64±1.63	48.85 - 234.48±20.91	5.02 - 24.02±2.14
Pt3	181.23±14.07	21.64±1.63	234.48±21.10	24.02±2.16
P3	181.23 - 465.87±32.97	21.64 - 53.98±3.82	234.48 - 576.43±38.79	24.02 - 58.95±3.97
Pt4	422.87±12.69	49.22±1.47	513.65±18.26	52.61±1.87
P4	465.87 - 673.14±28.22	53.98 - 78.34±3.27	576.43 - 762.06 ±18.95	58.95 - 78.77±1.94
Pt5	673.14±28.22	78.34±3.27	762.06±34.68	78.77±3.55
P5	673.14 – 863.21±22.18	78.34 - 100.00	762.06 – 977.78±25.50	78.77 – 100.00

Trials

No significant ($p>0.05$) differences were found between the true control blocked NP trials and the randomized catch NP trials for any lower extremity variable.

Moment of Support

The results demonstrated an overall positive moment of support (Ms) and a significantly ($p<0.05$) reduced EAI (65.91 ± 5.61 Nm/kg) for the FP condition compared to the NP (75.5 ± 6.78 Nm/kg) condition (FIGURE 2.4).

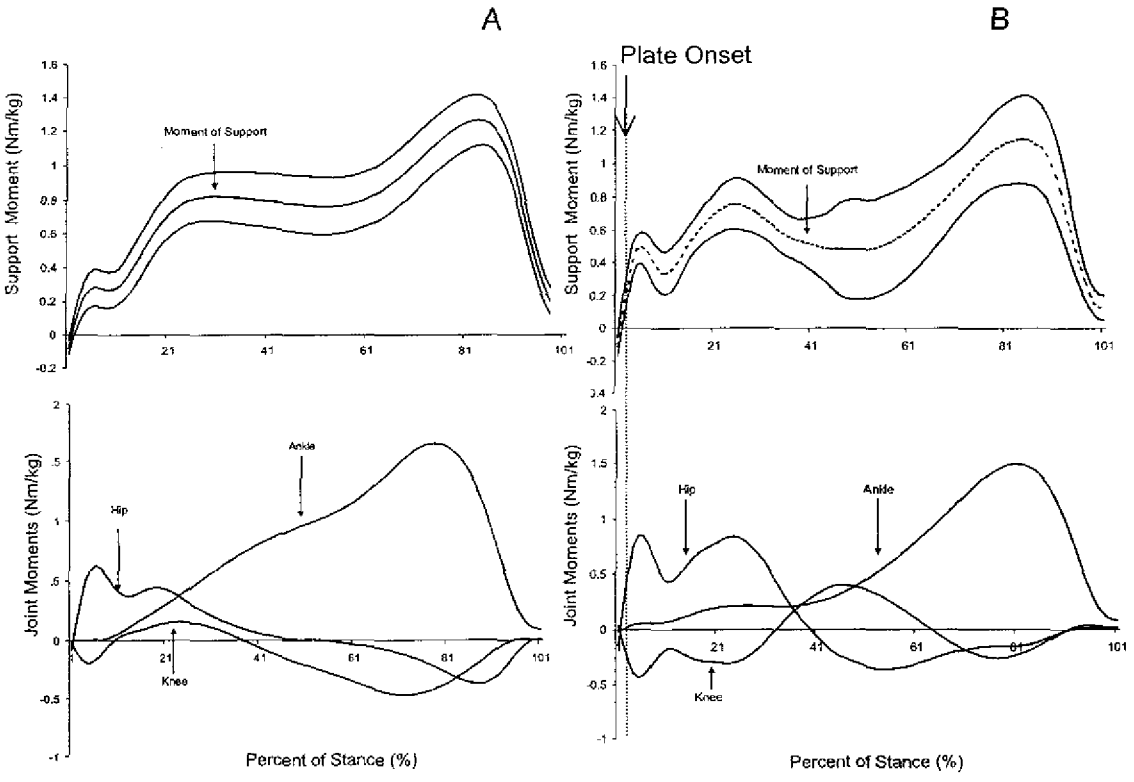

FIGURE 2.4. Moment of support (top) and overlay of individual joint moments (bottom) during NP (A) and FP conditions (B). Positive values are net extensor and plantarflexor moments and negative values are net flexor and dorsiflexor moments. Solid thick and thin lines are mean ± 1SD for the NP and FP moment of support.

Joint Moments

TABLE 2.2 presents lower extremity joint moments for each of the 5 phases (P) and 5 discrete points (Pt) of total stance during the NP and FP conditions as well as the total joint EAI for stance. The ankle exhibited significantly ($p<0.05$) less EAI while the knee and hip exhibited

significantly ($p<0.05$) more EAI during the FP condition compared to NP (TABLE 2.2; FIGURE 2.5A-C).

The ankle NP plantarflexor moment rose steadily from heel strike through mid-stance to Pt5 before declining rapidly during the latter half of P5 (TABLE 2.2; FIGURE 2.5A). The ankle FP plantarflexor moment remained relatively flat and was significantly ($p<0.05$) smaller in magnitude that NP through the latter part of early stance (P2) and the first half of mid-stance (Pt3, P3, Pt4; TABLE 2.2; FIGURE 2.5A). The ankle FP moment paralleled but was significantly ($p<0.05$) less than NP through the latter half of stance (P4, Pt5; TABLE 2.2; FIGURE 2.5A)

The knee NP generated an initial flexor moment in early stance and then followed a biphasic extensor-flexor-extensor moment pattern for early, mid-, and late stance periods, respectively (TABLE 2.2; FIGURE 2.5B). The knee FP produced a significantly ($p<0.05$) greater initial flexor moment than NP (P1, Pt1, Pt2) and, in contrast to NP, produced a flexor-extensor moment pattern during early (P2) and mid-stance (P2 – Pt4) periods. The knee FP moment paralleled NP for the remainder of stance although the knee produced a significantly ($p<0.05$) smaller flexor moment during the latter half of mid-stance (P4) and a significantly ($p<0.05$) greater extensor moment during late stance (Pt5, P5; TABLE 2.2; FIGURE 2.5B).

The hip NP extensor moment rose sharply in early stance and then decreased steadily until mid-stance after which a flexor moment was observed (TABLE 2.2; FIGURE 2.5C). The hip FP moment paralleled but was significantly ($p<0.05$) greater than NP following the onset of force plate movement (P1, Pt1, Pt2: TABLE 2.2; FIGURE 2.5C). In contrast to hip NP, the hip FP then produced an abrupt and significantly ($p<0.05$) greater extensor moment (P2, Pt3), which rapidly decreased and became a large flexor moment ($p<0.05$) through mid-stance (P3 – P4) that steadily declined through late stance (Pt5, P5; $p<0.05$; TABLE 2.2; FIGURE 2.5C).

TABLE 2.2. Mean (±SD) of Ankle, Knee, and Hip Joint Moments † for Non-Perturbed (NP) and Forward Perturbation (FP) Conditions (n=10)

Stance Partition	Ankle		Knee		Hip	
Phase (P)/ *Point*(Pt)	FP	NP	FP	NP	FP	NP
P1	0.03±0.02	0.01±0.02	-0.26±0.06*	-0.13±0.06	0.49±0.11*	0.30±0.11
Pt1	0.01±0.04	0.01±0.04	-0.38±0.09*	-0.12±0.09	0.80±0.15*	0.39±0.17
Pt2	0.01±0.04	0.01±0.05	-0.27±0.07*	-0.09±0.08	0.59±0.14*	0.38±0.18
P2	0.17±0.06*	0.22±0.14	-0.14±0.12*	0.09±0.06	0.55±0.17*	0.24±0.13
Pt3	0.26±0.13*	0.50±1.16	0.22±0.23	0.17±0.11	0.14±0.31	0.16±0.14
P3	0.57±0.24*	0.83±0.09	0.02±0.14*	-0.08±0.12	-0.21±0.19*	0.04±0.13
Pt4	0.77±0.21*	0.97±0.11	0.09±0.12*	-0.23±0.08	-0.28±0.19*	-0.01±0.12
P4	1.28±0.16*	1.36±0.09	-0.22±0.11*	-0.41±0.09	-0.14±0.16*	-0.31±0.10
Pt5	1.51±0.12*	1.65±0.08	-0.27±0.13*	-0.44±0.07	-0.12±0.21*	-0.34±0.11
P5	0.91±0.08	0.98±0.05	-0.09±.009*	-0.18±0.04	-0.02±0.16*	-0.07±0.05
EAI	58.51±9.634*	78.88±4.49	8.39±2.74*	3.44±2.23	22.64±7.26*	12.93±7.19

† Positive values indicate extensor and plantarflexor moments, negative values indicate flexor and dorsiflexor moments (Nm/kg)

* Significantly different than corresponding NP condition ($p<0.05$)

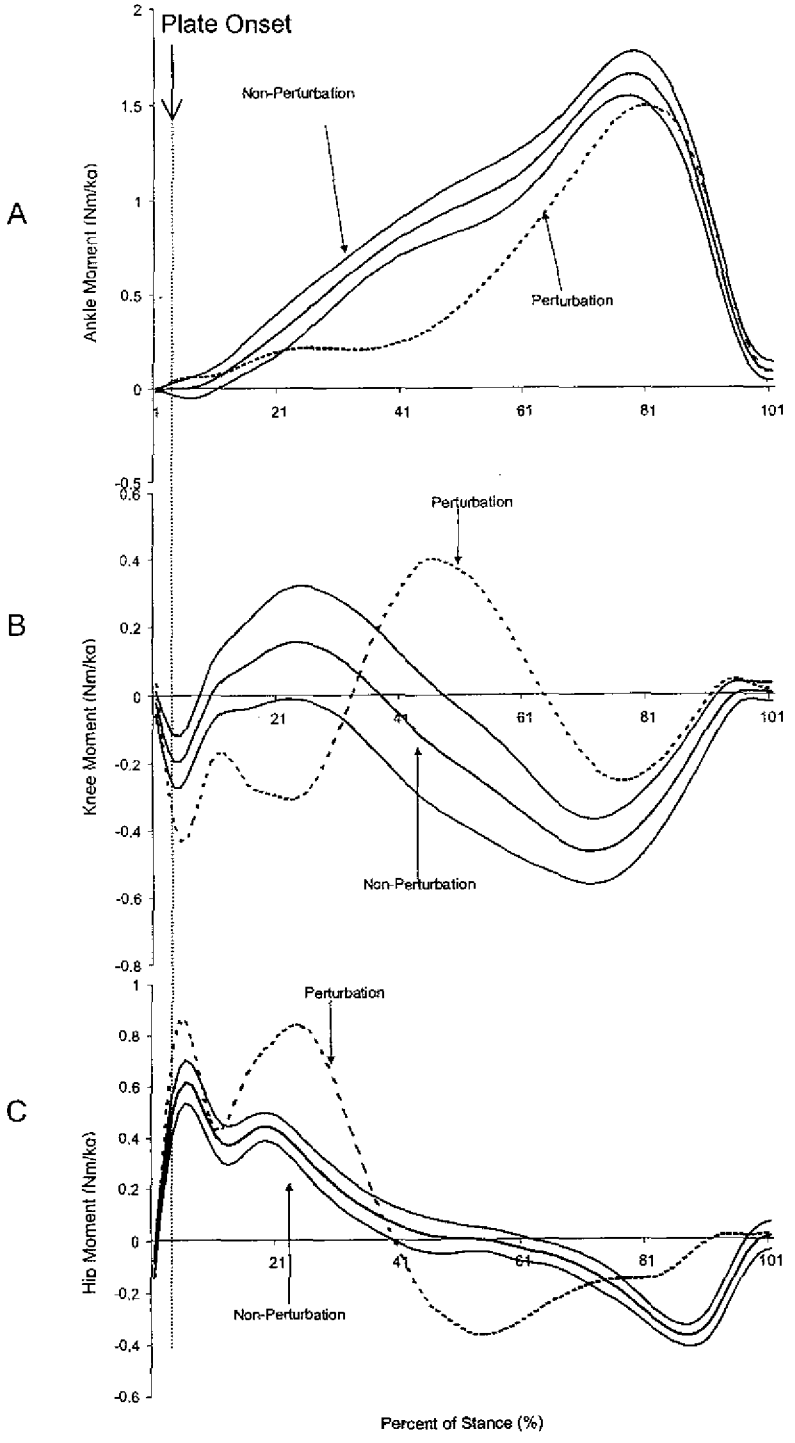

FIGURE 2.5. Ankle (A), knee (B), and hip (C) joint moments. Positive values indicate extensor and plantarflexor moments, negative values indicate flexor and dorsiflexor moments. Solid thick and thin lines are mean ± 1 SD for NP gait. Dashed thick line is mean of FP condition.

Joint Kinematics

TABLE 2.3 presents lower extremity joint position values during the NP and FP condition for P1-5 and Pt1-5 of total stance.

The ankle NP position curve followed a plantarflexion-dorsiflexion-plantarflexion pattern over early, mid-, and late stance periods, respectively (TABLE 2.3; FIGURE 2.6A). The mean ankle FP position curve generally paralleled the NP condition curve through the stance phase (TABLE 2.3; FIGURE 2.6A). However, the FP ankle position was significantly ($p<0.05$) more plantarflexed than NP during the early (Pt2, P2) and mid-stance (Pt3, P3) periods (TABLE 2.3; FIGURE 2.6A).

The knee NP position curve followed a flexion-extension-flexion pattern over early, mid-, and late stance periods, respectively (TABLE 2.3; FIGURE 2.6B). The knee FP position curve paralleled NP prior to and immediately following force plate translation after which, in contrast to NP, the knee position remained in a relatively static position (~15° flexion) until late stance (P2 – P4; TABLE 2.3; FIGURE 2.6B).

The hip NP position curve declined steadily from a flexed to extended position from early to mid-stance after which it followed a flexion-extension pattern from the latter half of mid-stance to late stance (TABLE 2.3; FIGURE 2.6C). The hip FP position curve generally paralleled the NP condition curve throughout stance except the FP hip position was significantly ($p<0.05$) more flexed during mid-stance (Pt3 – P4) compared to NP (TABLE 2.3; FIGURE 2.6C).

TABLE 2.3. Mean (±SD) of Ankle, Knee, and Hip Joint Positions † for Non-Perturbed (NP) and Forward Perturbation (FP) Conditions (n=10)

Stance Partition	Ankle		Knee		Hip	
Phase (P)/ Point(Pt)	FP	NP	FP	NP	FP	NP
P1	-3.79± 3.61	-4.66±1.32	8.53±1.86	8.55±2.23	18.74±2.15	18.6±1.95
Pt1	-3.95±3.31	-5.47±1.85	9.46±1.65	10.31±2.35	18.15±2.33	17.94±1.92
Pt2	-3.46±1.01*	-5.45±1.65	10.47±2.24	10.86±2.68	17.55±2.25	17.77±1.97
P2	-2.03±1.32*	-1.08±1.74	11.76±2.02*	14.54±2.24	14.02±2.04	14.68±1.22
Pt3	2.17±1.60*	5.99±1.33	13.67±2.49*	16.13±2.18	12.84±2.03*	10.06±0.84
P3	6.04±1.15*	9.88±2.65	12.92±2.21	12.41±2.44	9.31±1.48*	7.45±1.35
Pt4	10.91±1.48	8.71±2.78	11.9±2.36*	9.52±2.01	7.96±1.44*	6.36±1.37
P4	11.35±2.77	10.48±3.20	10.74±2.34*	8.21±1.92	10.09±1.29*	8.46±1.88
Pt5	10.28±2.75	9.18±0.19	11.61±2.54	9.51±2.17	11.21±1.55	10.64±1.92
P5	1.05±2.92	-1.18±3.74	22.23±2.77	21.26±1.96	11.31±5.42	10.95±3.84

† Positive values indicate flexion and dorsiflexion, negative values indicate extension and plantarflexion (°)

* Significantly different than corresponding NP condition ($p<0.05$)

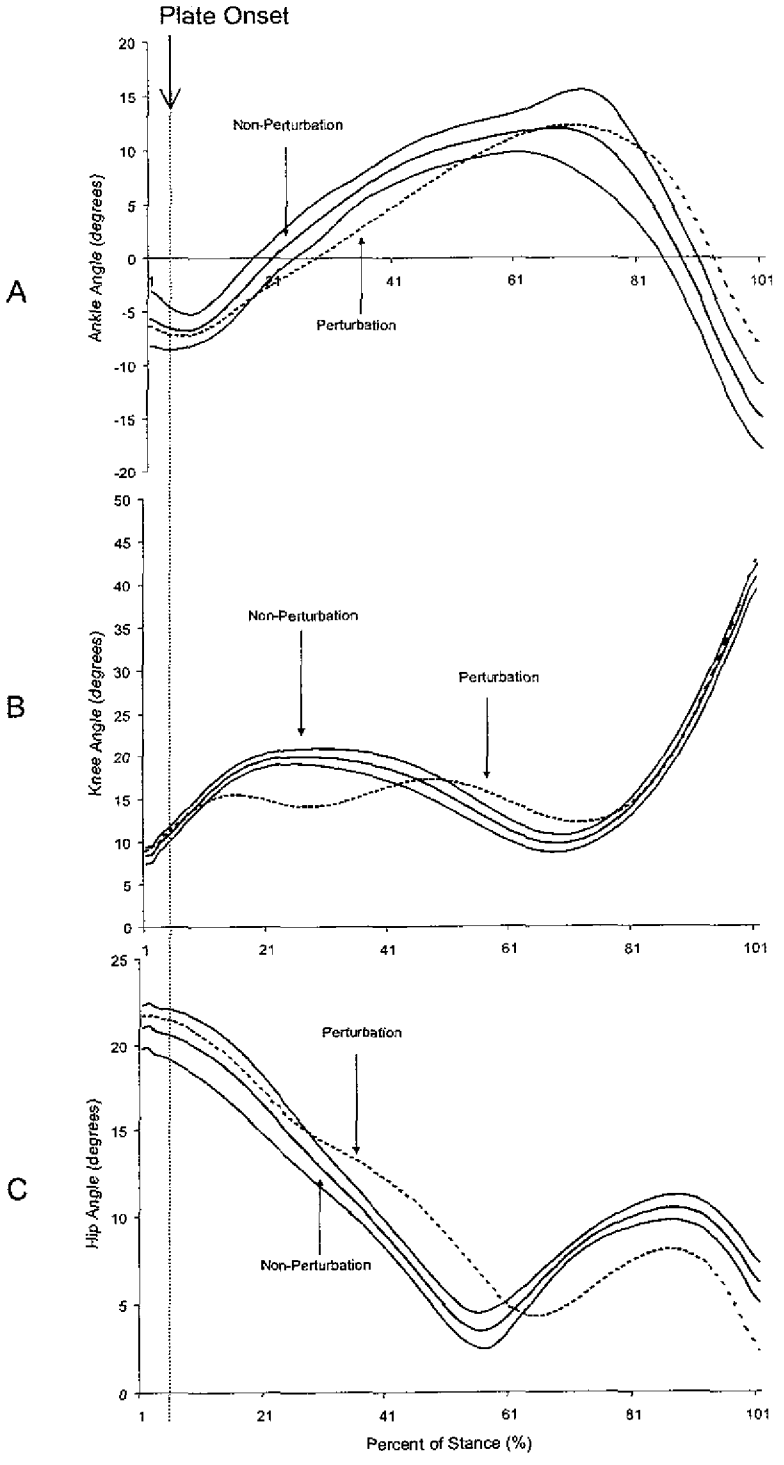

FIGURE 2.6. Ankle (A), knee (B), and hip (C) joint positions. Positive values indicate flexion and dorsiflexion, negative values indicate extension and plantarflexion. Solid thick and thin lines are mean ± 1 SD for NP gait. Dashed thick line is mean of FP condition.

Joint Power

TABLE 2.4 presents lower extremity joint powers during the NP and FP conditions for P1-5 and Pt1-5 of total stance.

The mean ankle NP ankle joint power curve revealed that small amounts of power were absorbed by the ankle during early stance and the first half of mid-stance after which the ankle sharply increased power generation until late stance (TABLE 2.4; FIGURE 2.7A). The mean ankle FP joint power curve generally paralleled NP over the course of stance. However, ankle FP absorbed significantly ($p<0.05$) less power during mid-stance (P2 - P4) and produced significantly ($p<0.05$) less power during late stance than ankle NP (Pt5, P5; TABLE 2.4; FIGURE 2.7A).

Knee NP and FP power curves differed markedly from one another. Both power curves were undulating in nature, with knee FP absorbing significantly ($p<0.05$) more power following onset of force plate movement (Pt1; TABLE 2.4; FIGURE 2.B7). During mid-stance, knee NP and FP power curves were prominently opposed to one another with knee FP generating power ($p<0.05$) early in mid-stance (P3) and absorbing power ($p<0.05$) late in stance (Pt4; TABLE 2.4; FIGURE 2.7B). During late stance (Pt5, P5), knee FP power absorption was significantly ($p<0.05$) less than knee NP (TABLE 2.4; FIGURE 2.7B).

The hip NP power curve exhibited power generation during early stance, power absorption during the first half of mid-stance followed by power generation for the latter half of mid-stance and late stance (TABLE 2.4; FIGURE 2.7C). The hip FP power curve differed markedly from the hip NP power curve as significantly ($p<0.05$) more power was generated by FP during early stance (P1 – Pt2) and throughout most of mid-stance (Pt3 – P4; TABLE 2.4;

FIGURE 2.7C). During late stance, (P5), the hip FP absorbed power in contrast ($p<0.05$) to power generation demonstrated in the NP condition (TABLE 2.4; FIGURE 2.7C).

TABLE 2.4. Mean (±SD) of Ankle, Knee, and Hip Joint Powers † for Non-Perturbed (NP) and Forward Perturbation (FP) Conditions (n=10)

Stance Partition	Ankle		Knee		Hip	
Phase (P)/ Point(Pt)	FP	NP	FP	NP	FP	NP
P1	0.06±0.23	0.01±0.008	-0.42±0.24	-0.40±0.19	0.56±0.22*	0.36±0.22
Pt1	0.08±0.42	0.002±0.19	-0.66±0.45*	-0.37±0.28	0.90±0.27*	0.51±0.32
Pt2	0.06±0.44	-0.02±0.18	-0.30±0.17	-0.26±0.20	0.79±0.26*	0.49±0.32
P2	-0.31±0.17*	-0.53±0.19	-0.10±0.07*	0.06±0.06	0.61±0.21	0.46±0.25
Pt3	-0.45±0.22*	-0.93±0.19	0.15±0.27	-0.03±0.11	0.05±0.20*	-0.33±.022
P3	-0.38±0.19*	-0.84±0.12	-0.07±0.11*	0.06±0.01	0.08±0.31*	-0.09±0.14
Pt4	-0.39±0.22*	0.94±0.51	-0.13±0.15*	0.22±0.05	0.33±0.03*	0.12±0.07
P4	0.16±0.81*	0.85±0.55	-0.19±0.12	-0.23±0.11	0.25±0.17*	0.04±0.09
Pt5	2.96±0.69	3.96±1.64	-0.49±0.26*	-0.92±0.07	0.11±0.27	0.01±0.13
P5	3.42±0.43*	4.14±0.81	-0.26±0.15*	-0.69±0.14	-0.05±0.01*	0.39±0.08

† Positive values indicate power generation, negative values indicate power absorption (W/kg)
* Significantly different than corresponding NP condition ($p<0.05$)

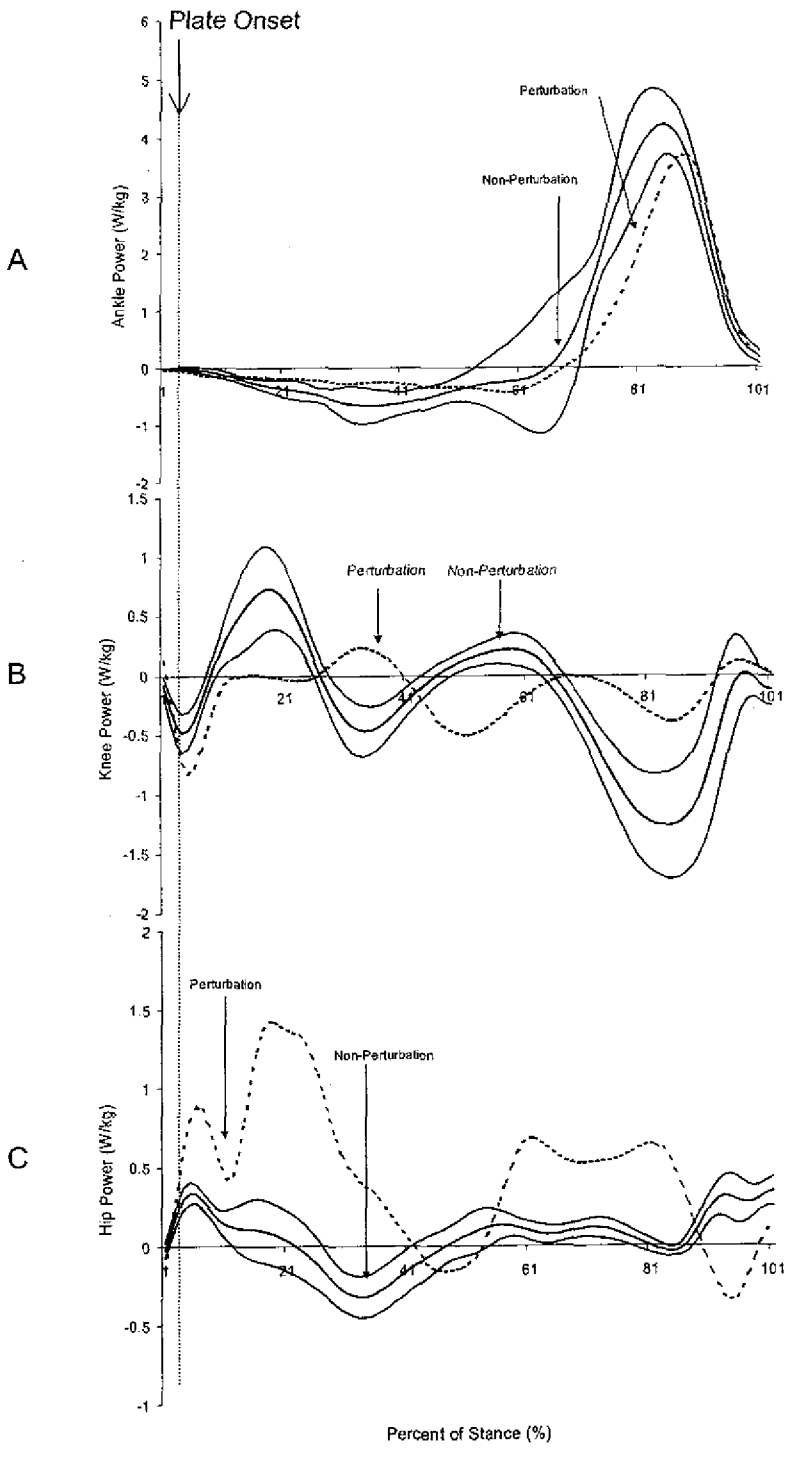

FIGURE 2.7. Ankle (A), knee (B), and hip (C) joint powers. Positive and negative values are energy generation and absorption by the muscles. Solid thick and thin lines are mean ± 1 SD for NP gait. Dashed thick line is mean of FP condition.

Muscle EMG

TABLE 2.5 presents lower extremity muscle EMG values during the NP and FP conditions for P1-5 and Pt1-5 of total stance. Values expressed are the FP:NP ratio for the corresponding phase or discrete point of stance.

The NP-TA EMG muscle response is characterized by strong activation during early stance followed by a rapid decrease to a low level for all of mid-stance and the first part of late stance, and then another surge of activity prior to toe-off (FIGURE 2.8A). Compared to NP, FP-TA activity was significantly ($p<0.05$) less during the first part of early stance (P1, Pt2) and significantly ($p<0.05$) greater during the latter half of early stance (P2) and most of mid-stance (Pt3, P3; TABLE 2.5; FIGURE 2.8A). FP-TA muscle activity then paralleled NP-TA activity for the remainder of stance.

The NP-GAS EMG muscle response is characterized by a steady rise in activity from heel strike through mid-stance and then a rapid decrease during late stance (FIGURE 2.8B). Compared to NP, FP-GAS produced significantly ($p<0.05$) more EMG activity during early stance (P1, Pt1) followed by significantly ($p<0.05$) less activity for the remainder of stance (P2 – P5; TABLE 2.5; FIGURE 2.8B).

The NP-VL EMG muscle response is characterized by a burst of activity during early stance that steadily drops and remains relatively low throughout the remainder of stance (FIGURE 2.8C). Compared to NP, FP-VL activity produced significantly ($p<0.05$) less EMG activity during most of early stance (P1-Pt2), followed by significantly ($p<0.05$) greater activity for the remainder of stance (P2 – P5; TABLE 2.5; FIGURE 2.8C).

The NP-BF EMG muscle response is characterized by strong activation during early stance followed by a steady decrease for all of mid-stance and the first part of late stance and a

surge of activity prior to toe-off (FIGURE 2.8D). Compared to NP, FP-BF EMG produced significantly ($p<0.05$) more EMG activity during early stance (P1 – P2) and most of mid-stance (P2 – Pt4) followed by reduced activity for the remainder of stance (P4 – P5; TABLE 2.5; FIGURE 2.8D).

TABLE 2.5. Mean (±SD) FP:NP Ratio of Muscle EMG Activity † (n=10)

Stance Partition	TA	GAS	BF	VL
Phase (P)/ Point(Pt)				
P1	0.87±0.09*	1.15±0.09*	1.24±0.12*	0.75±0.18*
Pt1	0.96±0.10	1.14±0.08*	1.26±0.08*	0.78±0.17*
Pt2	0.70±0.05*	1.01±0.04	1.17±0.06*	0.76±0.12*
P2	1.71±0.03*	0.92±0.03*	1.51±0.21*	1.23±0.07*
Pt3	1.63±0.15*	0.12±0.22*	1.39±0.05*	1.55±0.14*
P3	1.17±0.03*	0.80±0.03*	1.41±0.13*	1.51±0.14*
Pt4	0.97±0.03	1.10±0.12	1.28±0.07*	1.34±0.14*
P4	1.06±0.10	0.87±0.03*	0.66±0.05*	1.23±0.07*
Pt5	0.99±0.09	0.68±0.08*	0.74±0.08*	1.12±0.13
P5	0.98±0.03	0.85±0.06*	0.22±0.09*	1.04±0.01

† Values greater than 1.0 indicate FP EMG activity greater than NP condition, values less than 1.0 indicate FP EMG activity less than NP condition

* Significantly different than NP condition ($p<0.05$)

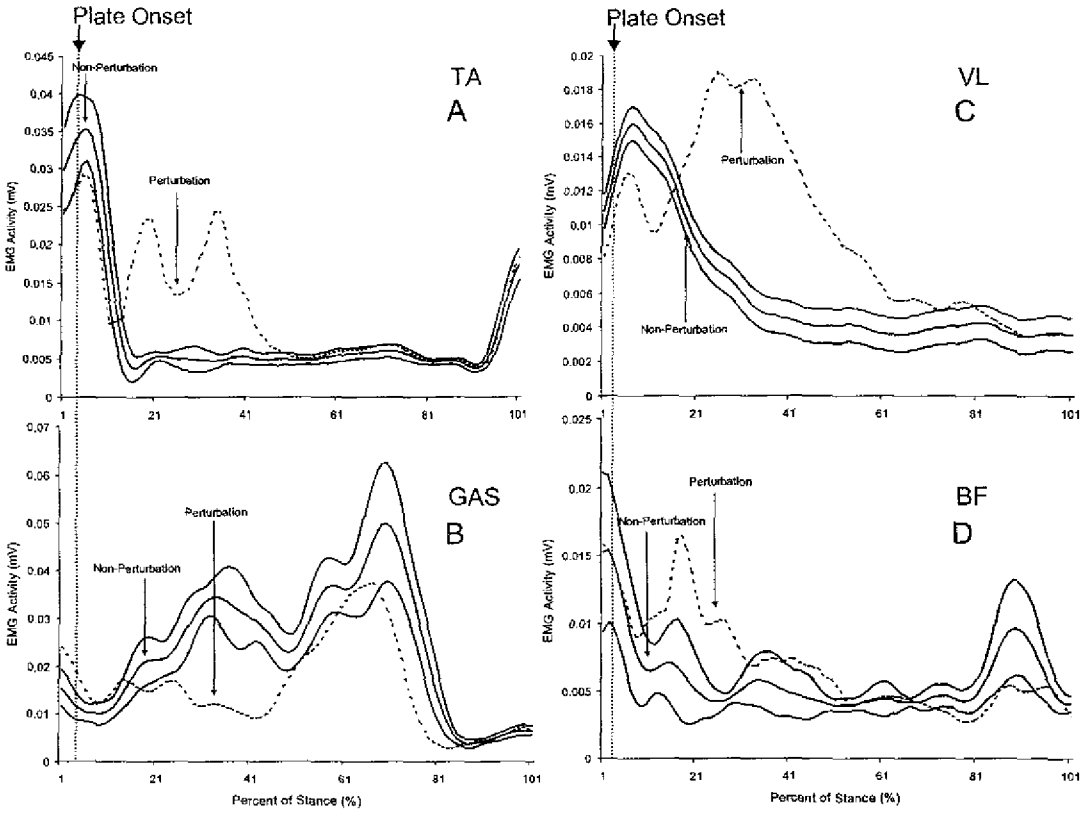

FIGURE 2.8: Representative example of muscle EMG activity during FP (dashed thick line) and NP (solid thick and thin lines are mean ± 1 SD) conditions for the tibialis anterior (TA: A), gastrocnemius (GAS: B), vastus lateralis (VL: C), and biceps femoris (BF: D).

Hypotheses Results

It was hypothesized that the forward perturbation would result in a greater knee and hip joint flexion and greater ankle plantarflexion. Observation of FIGURE 2.6A-C demonstrates that these hypotheses were confirmed although the subjects exhibited a static knee flexion angle as a reactive strategy to the FP. As a result of the alterations in lower extremity joint positions, it was also hypothesized that the perturbed limb would demonstrate a greater knee and hip extensor moment and a reduced ankle plantarflexion moment. Observation of FIGURE 2.5A-C demonstrates that these hypotheses were confirmed although the greater knee extensor moment occurred later in stance than hypothesized. In response to the FP, it was hypothesized that the subjects would demonstrate increases in ankle, knee, and hip joint extensor muscle EMG activity. Observation of FIGURE 2.8A-D supports these hypotheses as the TA, VL, and BF exhibited significantly greater EMG activity as a reactive strategy to maintain dynamic equilibrium during the FP.

Discussion

The purpose of this investigation was to determine the effect of unexpected forward gait perturbations on lower extremity joint moments and muscle EMG patterns in healthy subjects. This study was similar in methodology to two previous investigations (Nashner, 1980; Tang et al., 1998) in that an unexpected FP was applied to subjects upon heel strike using a hydraulically driven moveable force plate. However, this investigation is unique in that it is the first to calculate joint moments and powers associated with unexpected FP. It is hoped that these data will provide a more comprehensive understanding of how the lower extremity joints contribute to dynamic balance control when gait is disrupted.

Mechanical Aspects of Postural Control

Moment of Support

The results of this investigation suggest that control of dynamic equilibrium and maintenance of the Ms during an unexpected FP is a coordinated interaction between the three major lower extremity joints. An observation of the Ms during the FP condition revealed a pattern similar to NP but reduced in overall EAI by 14%. During early NP stance, the hip is the primary contributor to a positive Ms as the knee and ankle demonstrate a flexor and dorsiflexor moment, respectively (FIGURE 2.4A). During the first half of NP mid-stance, all three joints contribute to a positive Ms as extensor moments at the hip and knee were produced along with an ankle plantarflexor moment (FIGURE 2.4A). During the last half of NP stance the ankle was the only major contributor to a positive Ms as the hip and knee produced flexor moments until toe-off (FIGURE 2.4A). In contrast to NP, the knee made little, if any contribution to the maintenance of

a positive Ms when gait was perturbed during early stance (FIGURE 2.4B). This appears to be compensated for by a hip extensor moment and to a lesser extent by an ankle plantarflexor moment. However, near mid-stance of FP, the hip produced a flexor moment which, in turn, is compensated for by an extensor moment at the knee and an increase in the plantarflexor moment at the ankle. For the last half of FP stance, the hip, knee, and ankle demonstrated moments similar to NP gait (FIGURE 2.4B).

In summary, the results of this investigation demonstrated a positive Ms for the NP and FP conditions. However, dissimilar joint moment patterns were necessary to achieve the positive FP-Ms between NP and FP. Results from this investigation suggest that when one joint opposes or does not contribute to vertical support of the body, one or both of the other joints compensate to prevent collapse.

Ankle-Knee Complex

During early and mid- FP stance, the ankle was significantly more plantarflexed than NP (FIGURE 2.6A). During early FP stance, the TA produced significantly less EMG activation than NP (FIGURE 2.8A) and the GAS produced significantly more EMG activation than NP (FIGURE 2.8B). However, during FP mid-stance, the EMG activity patterns in the TA and GAS exhibited a reciprocal increase and decrease of EMG activity, respectively. These results are similar to those reported by Tang et al. (1998) and Nashner (1980) and suggest that humans exhibit characteristic leg EMG and ankle joint kinematic patterns in response to a gait perturbation. Tang et al. (1998) postulated that TA EMG activity served to restore the disrupted ankle joint trajectory and realign the leg segment of the perturbed limb. However, an explanation for suppressed GAS EMG activity from the FP was not addressed in that study. It is possible that suppressed GAS activation would attenuate the effect of FP-induced ankle plantarflexion and

help maintain balance. Another possible explanation for the increase in TA and suppression of GAS activity may come from examination of the moments and powers produced at the ankle joint during the FP.

A small eccentric ankle dorsiflexor moment (power absorption) followed by a large eccentric ankle plantarflexor moment was observed for early and mid- NP stance respectively (FIGURE 2.5A). A large concentric plantarflexor moment (power generation) was observed during the latter half of NP gait (FIGURE 2.5A). During FP, no ankle dorsiflexor moment was observed early in stance, in contrast to NP. Instead, the ankle produced a sustained, but significantly reduced, ankle plantarflexor moment throughout stance as compared to NP. Since the knee angle is under the control of moments of force at the hip and ankle, as well as the knee due to action of bi-articular muscles (van Ingen Schenau, 1990), a stronger than normal ankle plantarflexor moment can serve to slow down, or even reverse, forward rotation of the leg segment, resulting in a reduction in knee flexion (Winter 1980, 1990b). The sustained reduction in the ankle plantarflexor moment during early FP stance may allow for the static knee flexion position observed during the FP as a possible reactive balance strategy. It is also possible that the increase in TA EMG activity and reduction in GAS EMG activity reduced the ankle plantarflexor moment to allow for a static knee position necessary for balance recovery in FP.

During the late phase of FP stance, a reduction in the peak ankle plantarflexor moment and ankle power generation was observed as compared to NP (FIGURE 2.5A & 2.7A). Since the ankle power absorption is reduced during the first half of FP stance, the subsequent drop in the ankle plantarflexor moment and power in late FP stance could possibly be attributed to a reduced storage of elastic energy in the plantarflexor muscles during the first half of stance. Elastic energy storage, however, was not directly measured in this study.

Knee-Hip Complex

The motor patterns of the knee during early and mid-stance FP consisted of a large flexor moment during early stance which did not switch to an extensor moment until significantly later in mid-stance compared to NP (FIGURE 2.5B). Significant fluctuations in power production (FIGURE 2.7B) and a static knee flexion position (FIGURE 2.6B) were also observed during early and mid-stance of FP. An initial suppression of FP-VL EMG activity relative to NP was observed early in stance followed by a strong VL activation coincident with a large FP-BF EMG burst (FIGURE 2.8C & D). The EMG activity demonstrated by these two antagonistic muscles is indicative of a co-contraction, possibly to maintain knee joint stability during FP. Tang et al. (1998) reported similar thigh muscle EMG and knee joint kinematic results for the FP condition, suggesting that humans demonstrate characteristic thigh segment EMG and knee joint kinematic patterns in response to an unexpected FP.

It has been shown that there is a reciprocal trade-off between the hip and knee joints such that dynamic balance and control of the HAT segment occurs via a coordination between posterior muscles (hip extensors/knee flexors) and anterior muscles (hip flexors/knee extensors) acting at either joint (Winter, 1984, 1989). In the present study this reciprocal trade-off between the knee and hip was demonstrated during FP mid-stance when the knee exhibited an extensor moment in contrast to the knee flexor moment, observed during NP (FIGURE 2.5B). At this same time, the FP hip produced a large flexor moment in contrast to the extensor moment observed during NP (FIGURE 2.5C). The reciprocal trade-off between the knee and hip moments may be necessary to maintain a positive Ms and dynamic equilibrium in response to an unexpected FP.

It is important to note that within this forward perturbation paradigm, two perturbations actually take place: the first perturbation is the forward acceleration of the force plate upon heel

strike (~3% of stance, ~29ms after heel strike) and the second is the deceleration of the force plate (~59% of stance, ~543ms after force plate onset). The acceleration phase of the force plate was characterized by a displacement profile that ramped to a desired velocity. During the deceleration phase, force plate displacement followed a parabolic path. Previous investigations involving the same paradigm (Nashner, 1980; Tang et al., 1998) used in this investigation provided no information regarding the effects of deceleration of the force plate on lower extremity gait.

From general observation of the joint moment and position curves, there does not seem to be a significant alteration in pattern shortly after the deceleration of the force plate (~59% of stance). Although there are significant differences in patterns between the two conditions even late into stance, it is difficult to discern those postural adjustment responses due to the first and those due to the second perturbation. However, at the onset of the second perturbation, mid-stance has been achieved, full acceptance of body weight has occurred, and the body's COM is well over the BOS. This suggests that the second perturbation would have less influence on lower extremity gait mechanics as compared to the first perturbation. Bothner et al. (2001) investigated standing postural responses to backward perturbations and reported that platform deceleration had a quantifiable impact on lower extremity postural responses particularly at the knee and ankle joints. However, Bothner (2001) utilized a standing paradigm and significantly greater force plate displacements and movement velocities than employed in this investigation. These data suggest that the second perturbation might significantly influence lower extremity joint mechanics and that the joint moment data should be further decomposed to discern postural adjustments due to force plate movement and those due to muscular force generation.

From a mechanical standpoint, it is possible that an external perturbation applied at mid-stance, regardless of whether it results from deceleration or acceleration of the force plate, would

have little effect on gait patterns since full acceptance of body weight has occurred and the COM is completely under the BOS. Furthermore, a perturbation applied at mid-stance may require different balance requirements and may be less challenging than a perturbation applied at heel strike. However, it is clear that a heel strike perturbation is of sufficient magnitude to elicit alterations in lower extremity gait patterns, but the neurological locus of control of such postural responses remains unclear.

Neurological Aspects of Postural Control

Tang et al. (1998) and Nashner (1980) suggested that postural activity from leg and thigh muscles was the key to reactive balance control. Nashner (1980) also suggested that alteration of the ankle joint from its normal trajectory serves as the primary sensory input to the central nervous system (CNS) to trigger reactive balance mechanisms. However, results from this investigation demonstrate that an increase in the hip extensor moment as a result of the FP begins approximately 9.7ms (~1% after plate onset) after initiation of the force plate movement, whereas the reactive TA EMG burst is observed approximately 97ms (~10% after plate onset) after movement. Tang et al. (1998) reported similar TA EMG onset latencies of 91.2ms concomitant with suppression of GAS EMG activity and postulated that the control of these occurrences resulted from polysynaptic spinal reflexes or supraspinal loops. Other investigations have also demonstrated vestibulo-spinally mediated muscle onset latencies of 90-100ms during standing perturbations (Allum et al., 1995; Dietz et al., 1984; Gollhofer et al., 1986; Nashner, 1976) and have suggested that postural reactions to unexpected forward perturbations prior to 90ms are due to the mechanical viscoelastic stretching of muscle, tendon, and joint capsule (Herman et al., 1973; Nashner, 1977). Herman et al. (1973) studied the gait initiation torque patterns of humans

and reported that changes in muscle stiffness (torque development) immediately following gait initiation may be attributed to the inherent mechanical properties of muscle relative to lengthening of series-elastic tissue rather than changes in motor discharge patterns (EMG). Nashner (1977) described a significant stabilizing effect due primarily to ankle joint stiffness prior to leg muscle EMG activation approximately 90ms after plate onset. Furthermore, Nashner (1977) suggested that cortically-mediated responses would not begin until at least 250 ms following plate onset and that muscle EMG responses prior to 250ms are spinally-mediated.

In the present investigation, postural adjustment responses observed during the stance phase of FP can be divided into 3 sections that are representative of the type of postural adjustment responses thought to occur within each (FIGURE 2.9). Within this conceptual model, postural responses observed from the time immediately following the onset of plate movement (P1, Pt1, Pt2) are considered to be mechanical responses and can be solely attributed to viscoelastic changes in the tissues surrounding the lower extremity joints due to alteration of the body's COM relative to the BOS. Postural adjustment responses observed between 90 and 250ms (P2, Pt3) are considered to be a combination of mechanical and vestibulo-spinally mediated neuromuscular responses, whereas postural responses observed after 250ms (P3, Pt4, P4, Pt5, P5) are a combination of mechanical, vestibulo-spinally-mediated, and cortically-mediated neuromuscular responses.

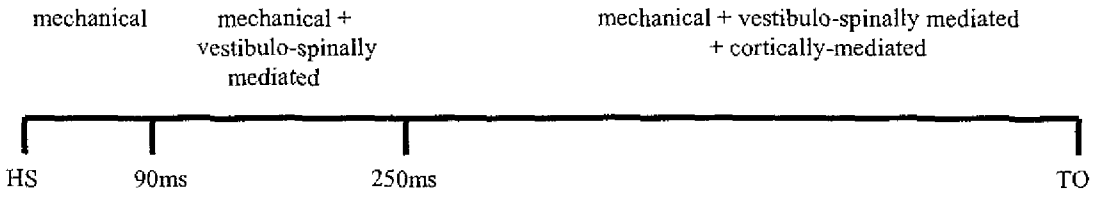

FIGURE 2.9: Division of stance phase (heel strike (HS) to toe off (TO)) into 3 divisions based on mechanical or neuromuscular postural adjustment responses.

Results of this investigation suggest that the initial responses to plate movement are mechanical in nature, rather than a neuromuscular response, and can be attributed to a viscoelastic stretching of pre-loaded joint muscles. For example, prior to heel strike, the muscles surrounding the hip are programmed to produce a certain amount of viscoelastic tension to provide vertical support to the body and prevent forward acceleration of the HAT segments upon heel strike and early in stance. In response to the FP, a large extensor hip moment is observed almost immediately after force plate movement (~9.7ms) which is possibly due to the mechanical stretching of pre-loaded hip muscles. Since spinally-mediated neuromuscular responses take 90-100ms to occur, and vertical support of the body is maintained until muscle EMG responses are observed 90ms after force plate movement, postural adjustment responses prior to 90ms can only be mechanical in nature.

The increase in TA EMG activity and reduction of GAS EMG activity exhibited approximately 97ms after onset of FP is consistent with previous literature (Nashner, 1977; Tang et al., 1998) and can be attributed to spinally-mediated neurological motor reflexes as a result of mechanical muscle stretch and alterations in lower extremity joint trajectories. Although the ankle is the first joint to undergo alterations in trajectory as a result of force plate movement, it is unlikely that the ankle is the only input to the CNS to initiate postural adjustments since the hip

and knee joints also undergo significant mechanical postural adjustments. EMG responses are observed in the BF and VL approximately 120-190ms after force plate movement and could correspond to the delayed alteration of knee and hip joint trajectories, relative to the ankle joint, and subsequent spinally-mediated neuromuscular responses.

Afferent input from mechanical postural responses most likely provide input to the CNS to initiate spinally-mediated postural adjustments first observed at approximately 90-100ms, and cortically-mediated neurologic responses observed 250ms after plate onset. Mechanical postural responses that occur later in FP stance may serve to provide continued afferent feedback to the CNS to promote continued spinal and higher level motor responses in an effort to maintain dynamic equilibrium, a positive Ms, and forward propulsion of the body during FP gait.

Summary

The findings of the present research suggest that reactive balance control is a coordinated and synchronized effort of the lower extremity joints in an effort to maintain dynamic equilibrium and the overall Ms during an unexpected FP applied at heel strike. The muscles surrounding the hip were found to be most important in maintaining control of the HAT segment and preventing collapse of the lower extremity as an initial response to the FP. Muscle EMG activity from the leg and thigh segments and joint kinematics demonstrated similar patterns compared to previous investigations. These results indicate that healthy subjects, in response to an unexpected FP, demonstrate joint moment and power patterns that are distinct from NP gait in order to maintain dynamic equilibrium during locomotion.

Bridge

The first study characterized the kinetic, kinematic, and muscle activation responses to an unexpected FP in non-injured young adults. Therefore, the purpose of the second study was to investigate how normal gait patterns may change as a result of chronic anterior cruciate ligament (ACL) injury and to determine the effect of unexpected FP on lower extremity joint moments, power, and kinematics as compared to healthy controls. With a better understanding of the neuromuscular and mechanical adaptations associated with ACL deficiency, improved rehabilitation protocols may be developed to prevent further injury. Chapter III summarizes the similarities and differences in the reactive balance responses evoked during unexpected FP between non-injured and ACL injured adults.

CHAPTER III

GAIT PERTURBATION RESPONSE IN CHRONIC ANTERIOR CRUCIATE LIGAMENT DEFICIENCY

Introduction

Injury to the anterior cruciate ligament (ACL) is among the most frequent sports-related injuries. Some studies have estimated that 1 per 3000 people suffer ACL tears annually in the United States with an estimated cost for these injuries of almost a billion dollars per year (Fu et al, 1999; Griffin et al, 2000). Recent epidemiological data suggest that injury to the ACL alone accounts for 59% of all knee ligament injuries, with 70% of ACL injuries occurring in non-contact situations (Bollen, 2000).

Although walking is the most common and repetitive non-contact movement, it is an extremely complex motor task. 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 gait. 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. Investigations involving acute (< 1 month) ACL injured subjects are limited. Devita et al. (1997) suggested

that, during the stance phase of gait, acutely ACL injured limbs exhibit a significantly more flexed knee position and a sustained knee extensor moment as compared to non-injured control subjects. Kinetic and kinematic results from investigations involving chronic (> 2 yr) ACL deficient (ACLD) subjects in the latter stages of rehabilitation and/or repair differ significantly from the results of Devita et al. (1997). Berchuck (1990) found alterations in the knee moments associated with gait in 16 ACLD patients compared to healthy controls. Specifically, Berchuck (1990) demonstrated that ACLD patients exhibit a sustained knee flexor moment during midstance as well as a more flexed knee position throughout stance. This type of gait pattern was interpreted as a tendency to avoid or reduce the demand placed on the quadriceps and was termed a "quadriceps avoidance gait" possibly serving to reduce anterior tibial shear during gait. Birac et al. (1991) supported this finding but suggested that development of a "quadriceps avoidance" pattern was directly related to time since injury. Data indicated that 88% of subjects who were at least 6 years post-injury demonstrated the avoidance pattern compared to only 44% of subjects injured less than 1.5 years. More recently, Wexler et al. (1998) investigated 30 ACLD patients for the presence of an avoidance gait pattern. Patients were divided into three groups according to time between injury and testing: 0 to 2.5 years (early), 2.5 to 7.5 years (intermediate), and greater than 7.5 years (chronic). The results revealed that all patients demonstrated a more flexed knee position and that 57% of the patients demonstrated a quadriceps avoidance gait pattern. Specifically, all but one of the eight patients in the chronic group and 50% of the subjects in the early and intermediate groups had this gait adaptation. This concept of an avoidance pattern was supported by Mikosz et al. (1992) who mathematically modeled the knee joint to quantify ligament loading during quadriceps avoidance and normal gait. Results revealed that the adaptive quadriceps avoidance gait pattern reduced strain to ligamentous structures stabilizing the knee

during level walking whereas normal gait patterns associated with ACLD generated higher strain values.

Other investigators have been unable to reproduce the quadriceps avoidance phenomenon in chronic ACLD patients (Bulgheroni et al., 1997; Roberts et al., 1999; Rudolph et al., 1998). Rudolph et al. (1998) investigated ACLD individuals having sustained ACL injury 1.5 years to 5.5 years prior to testing and classified patients as either a 'coper' or 'non-coper' depending on their ability to return to pre-injury activity levels. Results revealed that both ACLD groups demonstrated greater knee flexion position throughout stance for the involved limb. ACLD subjects also demonstrated a biphasic knee moment pattern similar to gait patterns in normal subjects and the subject's contralateral limb but reduced in magnitude at peak knee flexion angle. Bulgheroni et al. (1997) studied the gait patterns of 10 ACLD subjects 2 years post-injury and reported a decreased knee extensor moment during early stance but a typical biphasic knee moment pattern throughout stance as compared to healthy controls. Roberts et al. (1999) calculated the joint moments and recorded muscle electromyographic (EMG) values from 18 ACLD subjects with post-injury intervals ranging from 0.75 to 12 years for the specific purpose of corroborating the quadriceps avoidance pattern. Subjects demonstrated greater knee flexion position from mid- to late stance as compared to control subjects, a finding consistent with all previous investigations involving ACLD subjects. However, no ACLD patients demonstrated a sustained knee flexor moment, a decreased knee extensor moment, or a decreased duration of quadriceps EMG activity during stance regardless of time since injury. Roberts et al. (1999) concluded that quadriceps avoidance, as a gait adaptation in ACLD patients, might be less common than previously reported. Roberts et al. (1999) also hypothesized that the greater knee flexion angle may allow the hamstring muscles to aid in compensating for ACLD.

An antagonistic effect of the hamstring muscles to help reduce anterior tibial shear generated by quadriceps activity in ACL injured subjects has been demonstrated in previous investigations (Baratta et al., 1988; Devita et al., 1998; Kennedy et al., 1982). However, there is controversy surrounding the role of the hamstrings, and other knee muscles, in ACLD subjects with respect to both hamstring function as well as support of a quadriceps avoidance gait pattern. An EMG investigation by Limbird et al. (1988) supported an avoidance gait pattern and also suggested that knee flexor patterns may also change as a result of ACL injury. Limbird et al. (1988) reported decreased quadriceps EMG activity and increased hamstring EMG activity late in stance during normal gait in ACL injured patients as compared to healthy controls. Shiavi et al. (1992) also reported a decrease in quadriceps EMG activity in 20 ACLD patients 1 year after injury but also reported a decrease in hamstring EMG activity during normal gait. Beard et al. (1994) examined 18 patients with arthroscopically diagnosed unilateral ACLD approximately 2 years after initial injury and 9 uninjured volunteers for changes in lower extremity muscle EMG patterns during gait. The results indicated significantly increased duration of hamstring EMG activity but no change in duration of quadriceps EMG activity. Lass et al. (1991) investigated 14 patients with an arthroscopically verified complete rupture of the ACL 46 months after injury, and 16 uninjured controls, while subjects walked on a treadmill at different gradients. They reported an earlier onset of hamstring, quadriceps, and gastrocnemius EMG activity and a prolonged duration of vastus medialis and gastrocnemius EMG bursts as compared to control subjects.

It has been suggested that specific gait adaptations as a result of ACL injury do occur and that these adaptations may depend on the time since injury, although the existence of a quadriceps avoidance gait pattern has been questioned. Additional studies are needed to either support or refute the development of quadriceps avoidance in normal and perturbed gait.

There is a paucity of literature serving to explain reactive balance mechanisms to unexpected perturbations during gait (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 EMG activity in comparison to the unperturbed condition. However, these investigations only provided EMG and kinematic data. In Chapter II it was reported that the muscles surrounding the hip were found to be most important in maintaining control of the upper body and preventing collapse of the lower extremity as an initial response to the FP (Ferber, 2001). However, later in stance, the ankle, knee, and hip joints demonstrated significantly different joint moment patterns compared to normal gait in order to maintain dynamic equilibrium. During an unexpected gait perturbation, the ability of an ACL injured individual to react and maintain equilibrium is critical in prevention of reinjury, especially since individuals often encounter obstacles or perturbations during gait.

The purpose of this study was twofold: 1) to determine how normal gait patterns may change as a result of chronic ACLD and 2) to determine the effect of unexpected forward perturbations on chronic ACLD subjects as compared to healthy controls. With a better understanding of the neuromuscular and mechanical adaptations associated with ACLD during normal gait and in response to unexpected forward perturbations, improved rehabilitation protocols may be developed to prevent further injury.

It was hypothesized that the injured limb would exhibit a greater knee and hip extensor moment and that no subject would exhibit a quadriceps avoidance gait pattern during the NP and FP conditions. It was also hypothesized that the injured limb would exhibit a greater knee and

hip joint flexion position and increased co-activation of knee joint muscles in comparison to the uninjured controls during the NP and FP conditions.

Method

Subjects

Ten (5 males and 5 females) ACL deficient (ACLD) individuals aged 18-40 years were recruited as subjects for this study. The mean age, body mass, and body height of subjects were 27.7 yr (± 9.1 yr), 79.0 kg (± 13.8 kg), and 165.8 cm (± 20.2 cm) respectively. All 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 (range 2.2yr – 16.1yr; mean 5.7 yr). All ACLD subjects had a normal contralateral knee and did not exhibit dysfunction at any other lower extremity joint. The demographic information as well as the time since injury information is listed in TABLE 3.1.

Ten (5 males and 5 females) healthy uninjured young adults also participated in the study as control subjects (CON). Demographic information regarding these subjects has been described in Chapter II. Prior to participation, each subject signed a consent form (APPENDIX A) approved by the Human Subjects Compliance Committee at the University of Oregon.

Table 3.1. ACLD Subject Demographic and Time Post-Injury Information

Patient no.	Age (yr)	Body Mass (kg)	Height (cm)	Time Post-Injury (yr)
1	20	59.5	163.2	6.1
2	24	84.7	180.3	2.6
3	32	101.4	188.5	2.0
4	20	87.0	159.6	2.1
5	41	87.5	180.9	13.2
6	45	74.2	115.0	7.7
7	21	69.5	165.7	2.5
8	20	74.4	175.3	2.3
9	24	92.5	168.5	2.2
10	30	60.3	163.5	16.1

Experimental Apparatus and Protocol

The experimental apparatus and protocol was the same as that described in Chapter II. The ACLD subjects completed the same 48 walking trial tests as the CON subjects.

Instrumentation

EMG Data

As described in Chapter II, the EMG data for the FP and catch NP conditions were normalized to EMG activity during true control NP condition for the corresponding phases or discrete points of the same gait cycle. EMG data for the true control NP condition were normalized to the peak EMG muscle activity within a given trial. Ensemble averages were then calculated from the normalized EMG data.

Data Analysis

Data analysis was the same as that described in Chapter II. Individual joint moment, power, and position and muscle EMG curves were divided into 5 phases of the stance phase according to discrete kinetic events along with selection of 5 discrete points for analysis.

Statistical Analysis

Three-way repeated measures ANOVAs (10 x 3 x 2) were used to determine differences of, if any, between the two groups within the three conditions. The independent variables were 1) the 5 phases and 5 discrete points of stance and 2) condition (true control NP, catch NP, and FP),

and 3) group (CON and ACLD). The dependent variables were joint (ankle, knee, hip) 1) moment, 2) power, 3) position, and muscle (TA, GAS, BF, VL) EMG activation. A priori post-hoc tests were then performed to detect differences, if any, between groups within condition and between catch NP and true control NP trials within group. A maximum α level of 0.05 was used to indicate statistical significance.

Results

This study was conducted to determine how normal gait patterns may change as a result of chronic ACLD and to investigate the effect of unexpected forward perturbations during gait on lower extremity joint kinematics, moments, powers, and muscle EMG patterns in chronic ACLD and healthy subjects. In this section, descriptive measures of stance are presented first followed by lower extremity joint moments, joint kinematics, joint powers, and muscle EMG responses for 3 general aspects of stance: 1) early stance from heel strike to Pt3 including P1, Pt1, Pt2, and P2, 2) mid-stance from Pt3 to Pt5 including Pt3, P3, Pt4, and P5, and 3) late stance from Pt5 to toe-off including Pt5 and P5.

Descriptive Measures of Stance

Time

Total time of stance for the NP (CON: 863.06 ± 77.27ms; ACLD: 865.08 ± 52.22ms) and FP conditions (CON: 977.14 ± 58.33ms; ACLD: 962.33 ± 77.00ms) were similar ($p>0.05$) for the CON and ACLD groups.

Trials

No significant ($p>0.05$) differences were found between the true control blocked NP trials and the randomized catch NP trials for any lower extremity variable for either the CON or ACLD group.

Moment of Support

The results revealed an overall positive moment of support (Ms) for the NP and FP conditions (FIGURE 3.1). There were no significant differences ($p>0.05$) in total extensor angular impulse (EAI) between the CON (NP: 75.5 ± 14.23; FP: 65.91 ± 18.99) and ACLD (NP: 80.50 ± 25.85; FP: 67.55 ± 28.55) groups for the NP or FP conditions (FIGURE 3.1).

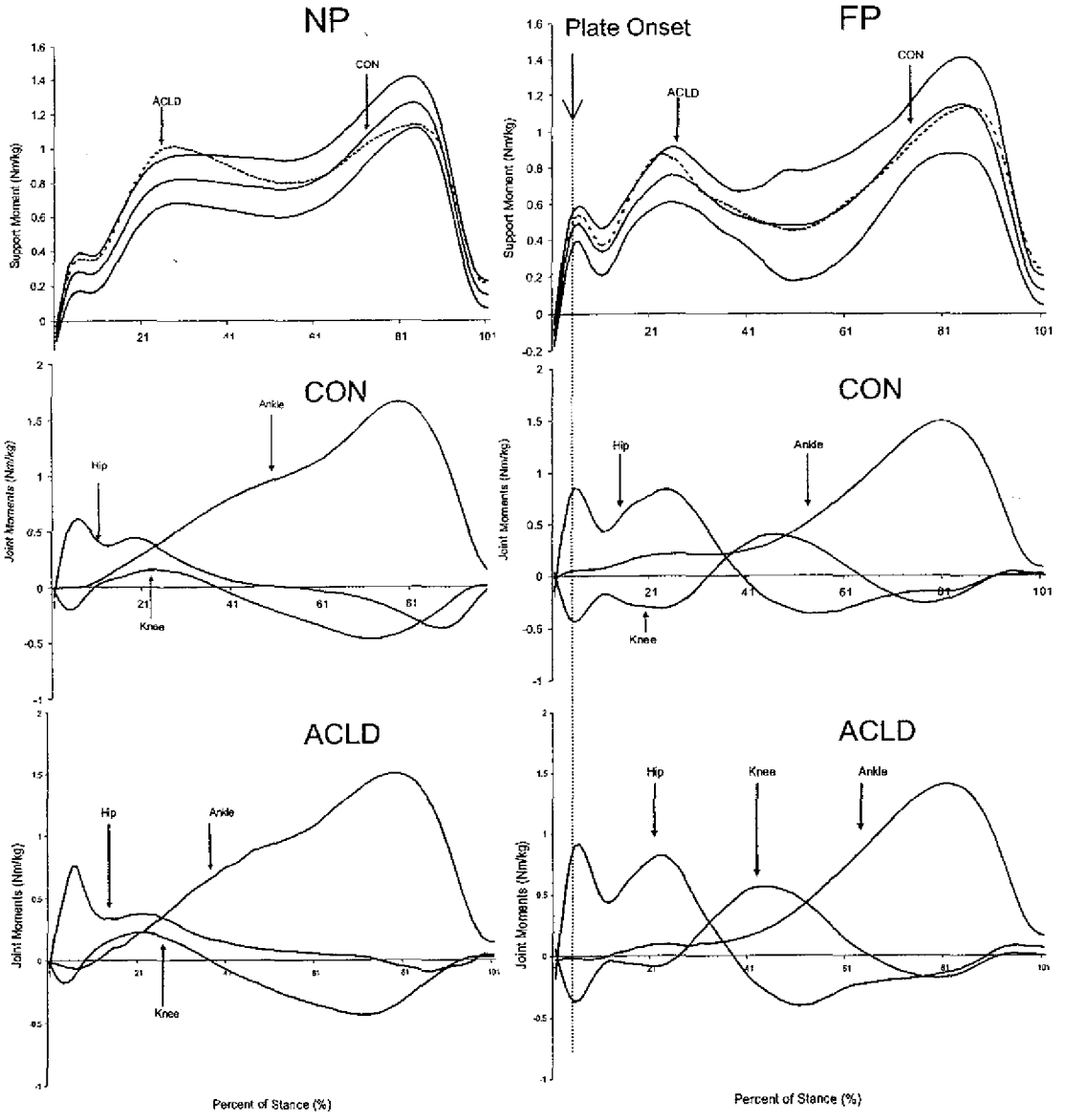

Figure 3.1. Moment of support (top graphs) and overlay of individual joint moments for CON and ACLD groups during NP (left graphs) and FP (right graphs) conditions. Solid thick line and thin dotted lines are mean ± 1SD for control subjects (CON). Positive values are net extensor and plantarflexor moments and negative values are net flexor and dorsiflexor moments.

Joint Moments

Non-perturbed Gait

TABLE 3.2 presents a comparison of ACLD and CON lower extremity joint moments during the NP condition for each of the 5 phases (P) and 5 discrete points (Pt) of total stance as well as the total joint extensor angular impulse (EAI) for stance. No significant ($p>0.05$) differences in ankle or knee EAI were observed between CON and ACLD; however, ACLD subjects exhibited significantly ($p<0.05$) greater hip NP-EAI than did CON subjects (TABLE 3.2).

The CON ankle NP plantarflexor moment rose steadily from heel strike through mid-stance to Pt5 before declining rapidly during the latter half of P5 (TABLE 3.2; FIGURE 3.2A). In contrast, the ACLD ankle NP demonstrated a dorsiflexor moment ($p<0.05$) during early stance (P1-Pt2), paralleled the CON-NP ankle plantarflexor moment during midstance, and generated a significantly ($p<0.05$) smaller plantarflexion moment than CON during late stance (P4, Pt5; TABLE 3.2; FIGURE 3.2A).

The CON and ACLD knee NP 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 3.2; FIGURE 3.2B).

The CON hip NP extensor moment rose sharply in early stance and then decreased steadily until mid-stance after which a flexor moment was observed (TABLE 3.2; FIGURE 3.2C). The ACLD hip NP extensor moment was significantly ($p<0.05$) greater during early stance (Pt1, Pt2, P2) and mid-stance (Pt3 – Pt4) periods and then switched to a significantly ($p<0.05$) reduced flexor moment, compared to CON, for the remainder of stance (P4, Pt5, P5: TABLE 3.2; FIGURE 3.2C).

Forward Perturbation Gait

TABLE 3.3 presents a comparison of ACLD and CON lower extremity joint moments during the FP condition 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 ankle, knee, or hip FP-EAI were observed between CON and ACLD groups (TABLE 3.3).

The CON ankle FP plantarflexor moment rose slowly from heel strike to midstance then increased rapidly during the latter half of stance (TABLE 3.3; FIGURE 3.3A). The ACLD ankle FP moment paralleled the CON ankle but was significantly ($p<0.05$) reduced in magnitude during early stance (P1 – P2; TABLE 3.3; FIGURE 3.3A).

The CON knee FP moment exhibited a flexor-extensor-flexor pattern through early, mid-, and late stance respectively (TABLE 3.3; FIGURE 3.3B). The ACLD knee FP moment paralleled the CON knee FP moment 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 3.3; FIGURE 3.3B).

The CON and ACLD hip FP moments paralleled one another throughout stance ($p>0.05$). The hip extensor moments rose sharply after heel strike and then rapidly decreased to a flexor moment throughout the remainder of stance (TABLE 3.3; FIGURE 3.3C).

TABLE 3.2. Mean (±SD) of Ankle, Knee, and Hip Joint Moments † for Anterior Cruciate Deficient (ACLD) and Control (CON) Subjects during NP Condition (n=10)

Stance Partition	Ankle		Knee		Hip	
Phase (P)/ Point(Pt)	CON	ACLD	CON	ACLD	CON	ACLD
P1	0.02±0.02	-0.04±0.02*	-0.13±0.06	-0.12±0.04	0.30±0.11	0.33±0.08
Pt1	0.01±0.04	-0.06±0.03*	-0.12±0.09	-0.14±0.05	0.39±0.17	0.55±0.09*
Pt2	0.02±0.05	-0.07±0.04*	-0.09±0.07	-0.07±0.09	0.38±0.18	0.58±0.12*
P2	0.22±0.01	0.15±0.08*	0.09±0.06	0.15±0.05	0.24±0.13	0.65±0.05*
Pt3	0.50±.016	0.47±0.18	0.17±0.11	0.24±0.08	0.16±0.14	0.28±0.07*
P3	0.83±0.09	0.79±.014	-0.08±0.12	-0.02±0.05	0.04±0.13	0.21±0.07*
Pt4	0.97±0.11	0.94±0.11	-0.23±0.08	-0.16±0.03	-0.01±0.12	0.04±0.05*
P4	1.36±0.09	1.26±0.06*	-0.41±0.09	-0.33±0.08	-0.31±0.10	-0.03±0.02*
Pt5	1.65±.014	1.49±0.12*	-0.44±0.07	-0.33±0.05	-0.34±0.11	-0.08±0.16*
P5	0.98±0.05	0.94±0.12	-0.18±0.04	-0.10±0.05	-0.07±0.05	0.11±0.01*
EAI	78.8±4.54	75.2±6.62	3.44±2.34	6.97±3.57	12.93±7.19	16.14±2.05*

† Positive values indicate extensor and plantarflexor moments, negative values indicate flexor and dorsiflexor moments (Nm/kg)
* Significantly different than CON ($p<0.05$)

TABLE 3.3. Mean (±SD) of Ankle, Knee, and Hip Joint Moments † for Anterior Cruciate Deficient (ACLD) and Control (CON) Subjects during FP Condition (n=10)

Stance Partition	Ankle		Knee		Hip	
Phase (P)/ Point(Pt)	CON	ACLD	CON	ACLD	CON	ACLD
P1	0.03±0.02	-0.02±0.01*	-0.26±0.06	-0.19±0.06	0.49±0.11	0.46±0.12
Pt1	0.04±0.01	-0.02±0.01*	-0.38±0.09	-0.34±0.08	0.80±0.15	0.89±0.16
Pt2	0.03±0.01	-0.03±0.02*	-0.27±0.07	-0.17±0.05*	0.59±0.14	0.62±0.12
P2	0.17±0.06	0.09±0.06*	-0.14±0.10	0.05±0.07*	0.55±0.17	0.53±0.14
Pt3	0.26±0.13	0.16±0.09	0.22±0.15	0.50±0.25*	0.14±0.14	-0.07±0.30
P3	0.57±0.21	0.46±0.13	0.02±0.14	0.36±0.17*	-0.21±0.17	-0.28±0.15
Pt4	0.77±0.21	0.71±0.22	0.09±0.02	0.13±0.05	-0.28±0.19	-0.25±0.18
P4	1.28±0.16	1.19±0.05	-0.22±0.11	-0.10±0.05	-0.14±0.16	-0.19±0.20
Pt5	1.51±0.12	1.41±0.14	-0.27±0.13	-0.16±0.15	-0.12±0.21	-0.15±0.03
P5	0.91±0.08	0.91±0.11	-0.09±0.06	-0.06±0.05	-0.02±0.07	0.01±0.15
EAI	58.51±9.64	51.11±5.94	8.39±2.66	14.79±7.66	22.66±7.25	23.61±10.38

† Positive values indicate extensor and plantarflexor moments, negative values indicate flexor and dorsiflexor moments (Nm/kg)
* Significantly different than CON ($p<0.05$)

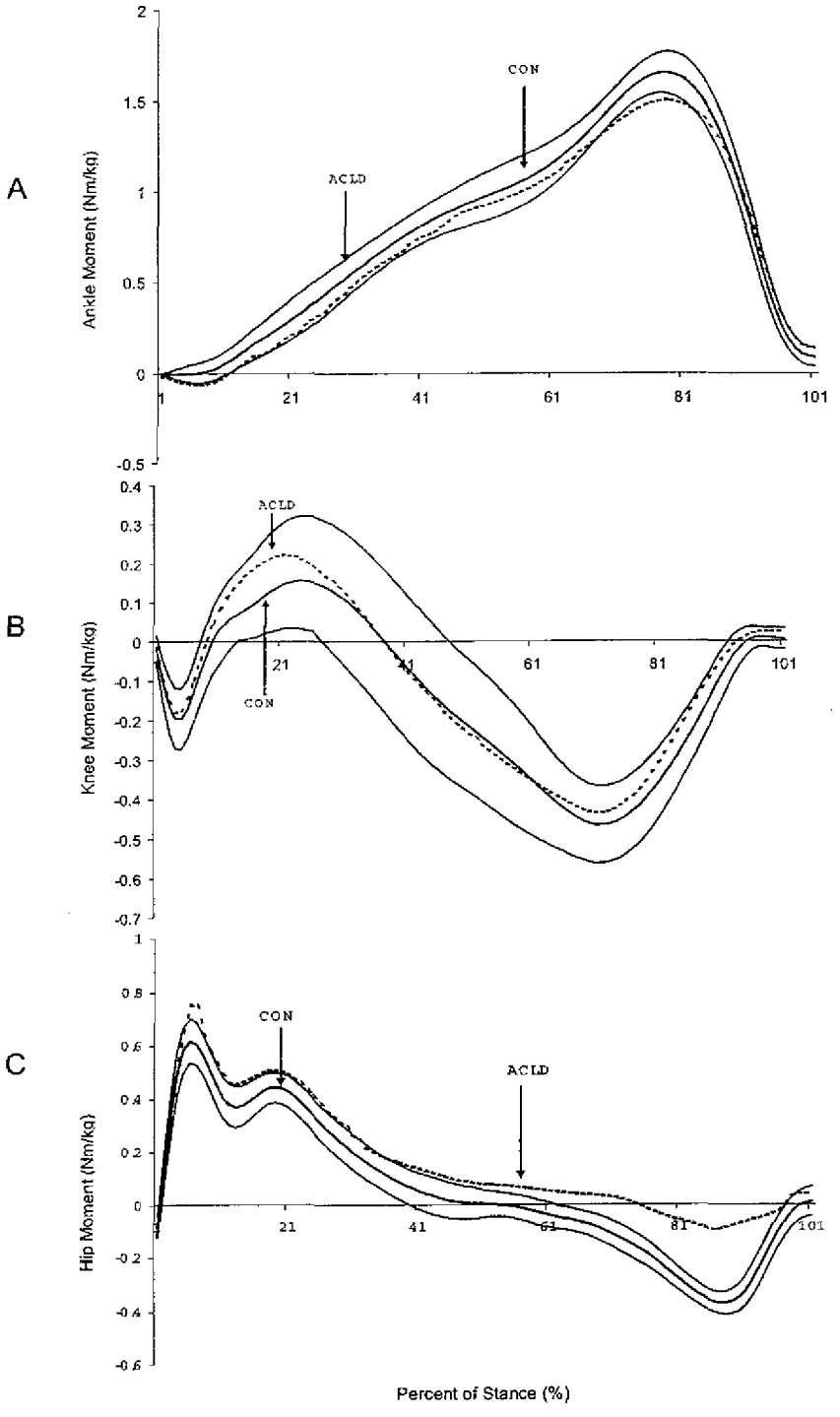

Figure 3.2. Ankle (A), knee (B), and hip (C) joint moments for anterior cruciate ligament deficient (ACLD) and control (CON) subjects during NP condition. Positive values indicate extensor and plantarflexor moments, negative values indicate flexor and dorsiflexor moments. Solid thick line and thin dotted lines are mean ± 1 SD for CON, dashed thick line is mean of ACLD group.

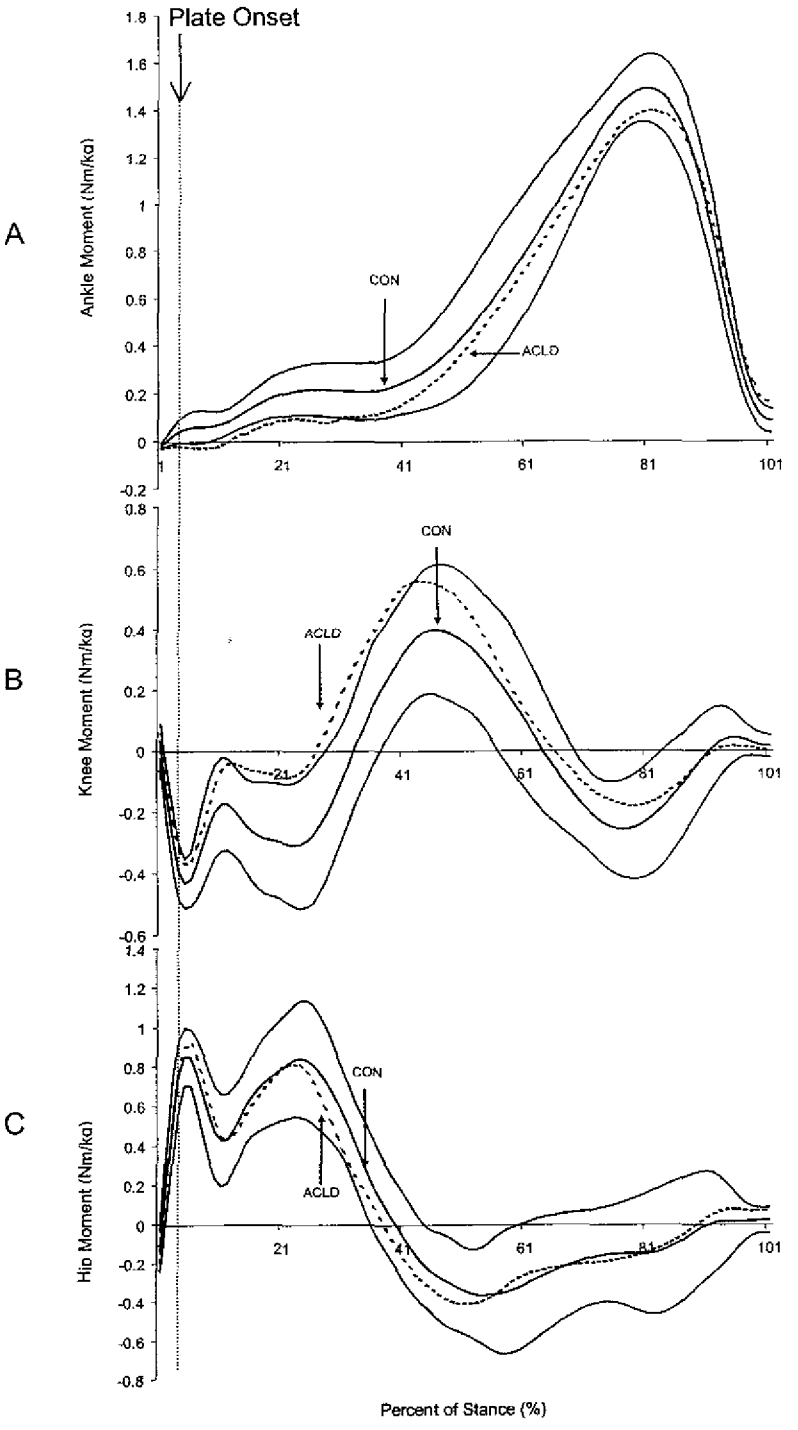

Figure 3.3. Ankle (A), knee (B), and hip (C) joint moments for anterior cruciate ligament deficient (ACLD) and control (CON) subjects during NP condition. Positive values indicate extensor and plantarflexor moments, negative values indicate flexor and dorsiflexor moments. Solid thick line and thin dotted lines are mean ± 1 SD for CON, dashed thick line is mean of ACLD group.

Joint Kinematics

Non-perturbed Gait

TABLE 3.4 compares ACLD and CON lower extremity joint position values during the NP condition for P1-5 and Pt1-5 of total stance. The CON ankle NP position curve followed a plantarflexion-dorsiflexion-plantarflexion pattern over early, mid-, and late stance periods, respectively

(TABLE 3.4; FIGURE 3.4A). The ACLD ankle NP position curve generally paralleled the CON NP curve but was significantly ($p<0.05$) more plantarflexed throughout most of stance (P2 – P5; TABLE 3.4; FIGURE 3.4A).

The ACLD and CON knee NP position curves paralleled one another throughout stance ($p>0.05$) and followed a flexion-extension-flexion pattern over early, mid-, and late stance periods respectively (TABLE 3.4; FIGURE 3.4B).

The CON hip NP position curve declined steadily from a flexed position to extension from early to mid-stance respectively after which it followed a flexion-extension pattern from the latter half of mid- to late stance (TABLE 3.4; FIGURE 3.4C). The ACLD hip NP curve generally paralleled the CON NP curve but was significantly ($p<0.05$) more flexed during early stance (P1, Pt1, Pt2, P2) and the first half of mid-stance (Pt3-Pt4; TABLE 3.4; FIGURE 3.4C).

Forward Perturbation Gait

TABLE 3.5 compares ACLD and CON lower extremity joint position values during the FP condition for P1-5 and Pt1-5 of total stance. During FP, the CON ankle steadily dorsiflexed until midstance when it began to plantarflex for the remainder of stance (TABLE 3.4; FIGURE 3.4A). The ACLD ankle FP curve generally paralleled the CON ankle curve but was

significantly ($p<0.05$) more plantarflexed throughout most of stance (P1 – P5; TABLE 3.5; FIGURE 3.5A).

The ACLD and CON knee FP curves paralleled one another throughout stance ($p>0.05$) and remained in a relatively static position until late stance when a sharp increase in knee flexion was observed (TABLE 3.5; FIGURE 3.5B).

The CON hip FP curve declined steadily from a flexed position to extension from early to mid-stance after which it followed a flexion-extension pattern until toe-off (TABLE 3.5; FIGURE 3.5C). The ACLD hip FP curve followed a similar pattern but was significantly ($p<0.05$) more flexed during early stance (P1 – P2) and most of mid-stance (Pt3 – Pt4; TABLE 3.5; FIGURE 3.5C).

TABLE 3.4. Mean (±SD) of Ankle, Knee, and Hip Joint Positions † for Anterior Cruciate Deficient (ACLD) and Control (CON) Subjects during NP Condition (n=10)

Stance Partition	Ankle		Knee		Hip	
Phase (P)/ Point(Pt)	CON	ACLD	CON	ACLD	CON	ACLD
P1	-4.66±1.33	-5.3±1.91	8.55±2.23	9.2±2.82	18.6±1.87	23.95±3.34*
Pt1	-5.47±1.78	-7.2±2.34	10.31±2.34	10.56±3.17	17.94±1.88	23.64±4.31*
Pt2	-5.45±1.62	-7.86±2.42	10.86±2.58	11.68±4.08	17.77±1.93	23.47±4.28*
P2	-1.08±1.74	-6.26±3.37*	14.54±2.24	16.23±4.62	14.68±1.05	21.13±4.12*
Pt3	5.99±1.35	-1.67±2.31*	16.13±2.14	18.76±3.34	10.06±0.82	17.76±3.87*
P3	9.88±2.61	2.06±3.00*	12.41±2.47	14.52±3.18	7.45±1.36	12.9±3.02*
Pt4	8.71±2.74	2.97±3.41*	9.52±2.00	11.4±2.51	6.36±1.34	10.35±3.36*
P4	10.48±3.18	3.6±3.25*	8.21±1.87	9.04±2.55	8.46±1.79	11.01±3.04
Pt5	9.18±0.19	1.95±3.35*	9.51±2.10	10.01±3.73	10.64±1.92	12.47±3.00
P5	-1.18±3.72	-7.82±2.24*	21.26±1.92	19.82±4.47	10.95±3.84	12.56±3.69

† Positive values indicate flexion and dorsiflexion, negative values indicate extension and plantarflexion (°)
* Significantly different than CON ($p<0.05$)

TABLE 3.5. Mean (±SD) of Ankle, Knee, and Hip Joint Positions † for Anterior Cruciate Deficient (ACLD) and Control (CON) Subjects during NP Condition (n=10)

Stance Partition	Ankle		Knee		Hip	
Phase (P)/ Point(Pt)	CON	ACLD	CON	ACLD	CON	ACLD
P1	-3.79±3.61	-7.13±3.22*	8.53±1.86	10.23±2.46	18.74±2.06	24.15±4.27*
Pt1	-3.95±3.34	-8.77±3.65*	9.46±1.56	11.27±2.87	18.15±2.34	23.78±4.28*
Pt2	-3.46±1.00	-9.92±4.78*	10.47±2.22	12.96±3.84	17.55±2.18	23.12±4.04*
P2	-2.03±1.26	-6.41±3.32*	11.76±2.00	14.89±3.67	14.02±2.04	19.65±3.00*
Pt3	2.17±1.64	-0.30±1.87*	13.67±2.44	16.52±2.29	12.84±2.00	15.66±4.22*
P3	6.04±1.05	4.13±1.34*	12.92±2.16	15.01±2.34	9.31±1.35	13.36±3.18*
Pt4	10.91±1.42	6.5±2.00*	11.9±2.32	13.49±2.04	7.96±1.44	11.71±2.44*
P4	11.35±2.73	7.81±1.34*	10.74±2.33	12.56±4.56	10.09±1.29	12.93±2.13
Pt5	10.28±2.69	6.81±2.06*	11.61±2.47	13.09±2.74	11.21±1.48	13.71±3.77
P5	1.05±1.86	-3.33±2.33*	22.23±2.69	21.78±2.45	11.31±5.44	13.85±3.44

† Positive values indicate flexion and dorsiflexion, negative values indicate extension and plantarflexion (°)
* Significantly different than CON ($p<0.05$)

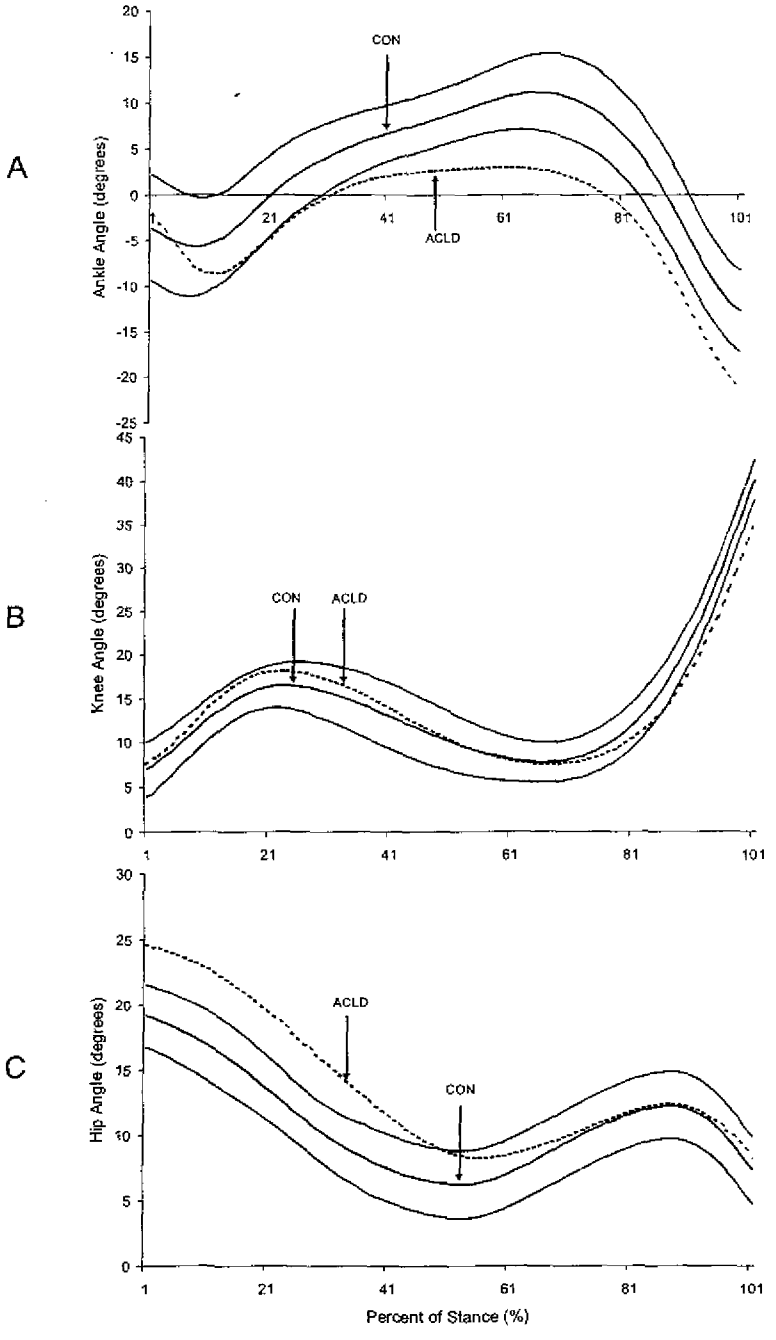

Figure 3.4. Ankle (A), knee (B), and hip (C) joint positions for anterior cruciate ligament deficient (ACLD) and control (CON) subjects during NP condition. Positive values indicate extensor and plantarflexor moments, negative values indicate flexor and dorsiflexor moments. Solid thick line and thin dotted lines are mean ± 1 SD for CON, dashed thick line is mean of ACLD group.

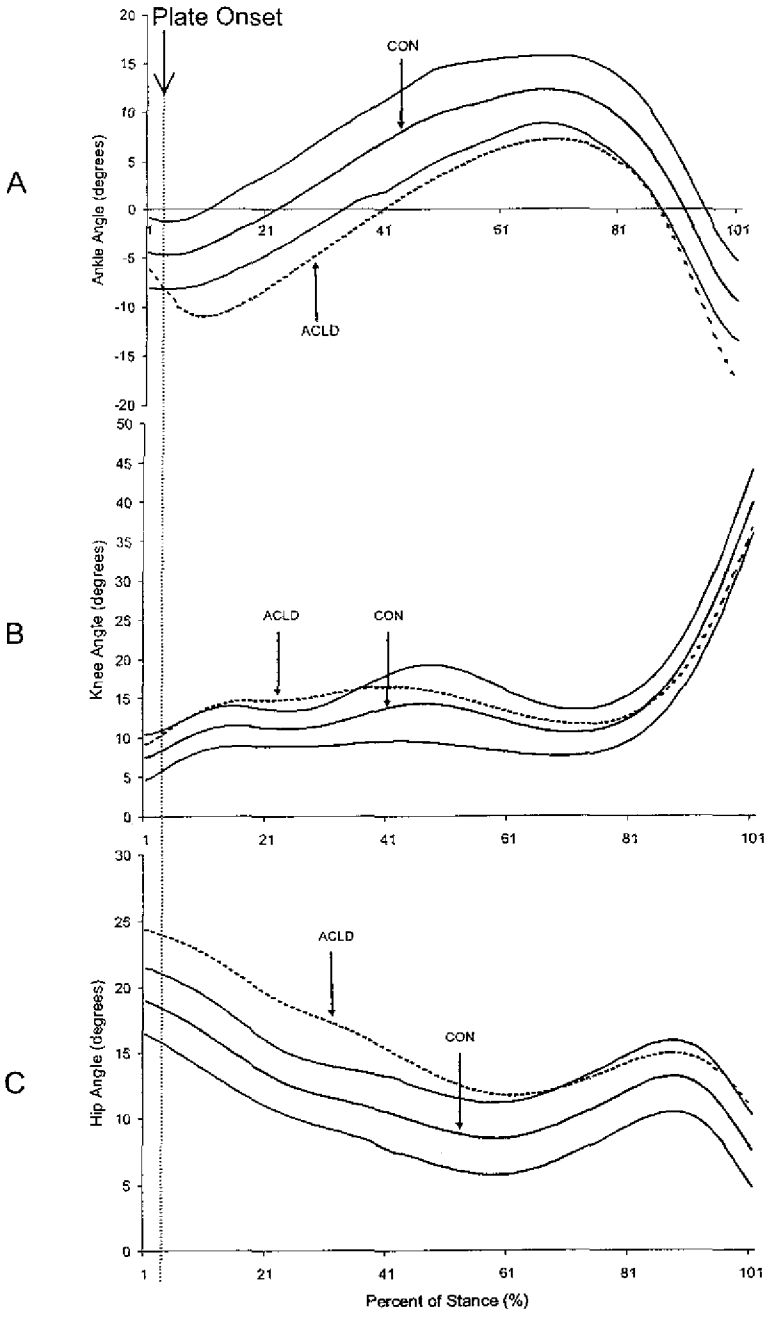

Figure 3.5. Ankle (A), knee (B), and hip (C) joint positions for anterior cruciate ligament deficient (ACLD) and control (CON) subjects during FP condition. Positive values indicate extensor and plantarflexor moments, negative values indicate flexor and dorsiflexor moments. Solid thick line and thin dotted lines are mean ± 1 SD for CON, dashed thick line is mean of ACLD group.

Joint Powers

Non-perturbed Gait

TABLE 3.6 compares ACLD and CON lower extremity joint powers during the NP condition for P1-5 and Pt1-5 of total stance. The CON ankle NP power curve demonstrated that the ankle absorbed small amounts of power during early stance and the first half of mid-stance after which the ankle produced substantial power until late stance (TABLE 3.6; FIGURE 3.6A). During NP, the ACLD ankle absorbed significantly ($p<0.05$) more power during early stance (P1 – Pt2) then paralleled the CON NP for the remainder of stance (TABLE 3.6; FIGURE 3.6A).

The CON knee NP power was undulating in nature until later in mid-stance when the knee absorbed relatively large amounts of power until toe-off (TABLE 3.6; FIGURE 3.6B). The ACLD knee NP power curve generally paralleled the CON NP curve until late stance when the ACLD knee absorbed significantly ($p<0.05$) less power (P5; TABLE 3.6; FIGURE 3.6B).

The CON hip NP produced power during early stance after which power was absorbed for the first part of mid-stance. Small amounts of power were generated for the remainder of stance (TABLE 3.6; FIGURE 3.6C). The ACLD hip NP curve was similar to CON during early stance but differed significantly ($p<0.05$) during mid-stance when large amounts of power were generated (Pt3, P3; TABLE 3.6; FIGURE 3.6C). The ACLD hip NP also generated significantly ($p<0.05$) more power than CON near the end of mid-stance and significantly ($p<0.05$) less power during late stance (Pt5, P5; TABLE 3.6; FIGURE 3.6C).

Forward Perturbation Gait

TABLE 3.7 compares ACLD and CON lower extremity joint powers during the FP condition for P1-5 and Pt1-5 of total stance. The ACLD and CON ankle FP power curves

paralleled one another ($p>0.05$) as small amounts of power were absorbed during early stance and the first half of mid-stance. The ACLD and CON ankle then rapidly produced power until late stance (TABLE 3.7; FIGURE 3.7A).

The ACLD and CON knee FP power curves were similar to one another ($p>0.05$) and demonstrated undulating patterns of power absorption and production throughout stance (TABLE 3.7; FIGURE 3.7B).

The ACLD and CON hip FP power curves also paralleled one another ($p>0.05$) with large amounts of power produced during early stance, small amounts of power absorbed during mid-stance, and small amounts of power produced during late stance (TABLE 3.7; FIGURE 3.7C).

TABLE 3.6. Mean (±SD) of Ankle, Knee, and Hip Joint Powers † for Anterior Cruciate Deficient (ACLD) and Control (CON) Subjects during NP Condition (n=10)

Stance Partition	Ankle		Knee		Hip	
Phase (P)/ Point(Pt)	CON	ACLD	CON	ACLD	CON	ACLD
P1	-0.01±0.08	-0.13±0.09*	-0.40±0.19	-0.35±0.19	0.36±0.18	0.26±0.17
Pt1	-0.02±0.15	-0.19±0.05*	-0.37±0.18	-0.46±0.19	0.51±0.32	0.47±0.28
Pt2	-0.01±0.02	-0.13±0.05*	-0.26±0.06	-0.19±0.17	0.49±0.32	0.47±0.25
P2	-0.53±0.19	-0.74±0.18	0.06±0.06	0.16±0.10	0.46±0.25	0.56±.027
Pt3	-0.93±0.19	-0.97±0.32	-0.03±0.10	-0.13±0.10	-0.33±0.22	0.61±0.26*
P3	-0.84±0.12	-0.48±0.35	0.06±0.04	0.03±0.06	-0.09±0.04	0.35±0.15*
Pt4	0.94±0.51	-0.28±0.26	0.22±0.05	0.23±0.06	0.12±0.07	0.11±0.05
P4	0.85±0.55	0.84±0.51	-0.23±0.11	-0.08±0.10	0.04±0.09	0.17±0.06*
Pt5	3.96±1.64	3.24±1.45	-0.92±0.37	-0.56±0.40	0.01±0.03	0.22±0.16*
P5	4.14±0.81	4.06±0.73	-0.69±0.14	-0.25±0.35*	0.39±0.08	0.16±0.08*

† Positive values indicate power generation, negative values indicate power absorption (W/kg)
* Significantly different than CON ($p<0.05$)

TABLE 3.7. Mean (±SD) of Ankle, Knee, and Hip Joint Powers † for Anterior Cruciate Deficient (ACLD) and Control (CON) Subjects during FP Condition (n=10)

Stance Partition	Ankle		Knee		Hip	
Phase (P)/ Point(Pt)	CON	ACLD	CON	ACLD	CON	ACLD
P1	-0.01±0.03	-0.09±0.08	-0.42±0.24	-0.41±0.15	0.56±0.22	0.39±0.16
Pt1	-0.02±0.12	-0.09±0.05	-0.66±0.45	-0.76±0.25	0.90±0.27	0.78±0.45
Pt2	-0.03±0.14	-0.08±0.06	-0.30±0.17	-0.28±0.11	0.79±0.26	0.67±0.35
P2	-0.31±0.16	-0.17±0.10	-0.01±0.07	-0.03±0.02	0.61±0.21	0.57±0.26
Pt3	-0.45±0.16	-0.27±0.10	0.15±0.17	-0.06±0.77	0.05±0.15	-0.21±0.24
P3	-0.38±0.19	-0.39±0.25	-0.07±0.10	-0.27±0.16	0.08±0.12	-0.12±0.15
Pt4	-0.39±0.22	-0.27±0.06	-0.13±0.15	-0.05±0.05	0.33±0.22	0.16±0.13
P4	1.16±0.81	0.76±0.45	-0.19±0.12	-0.16±0.14	0.25±0.17	0.37±0.15
Pt5	2.96±0.69	2.79±0.58	-0.49±0.26	-0.16±0.15	0.11±0.17	0.37±0.24
P5	3.42±0.43	4.05±0.66	-0.26±0.15	-0.10±0.08	0.05±0.09	0.10±0.05

† Positive values indicate power generation, negative values indicate power absorption (W/kg)
* Significantly different than CON ($p<0.05$)

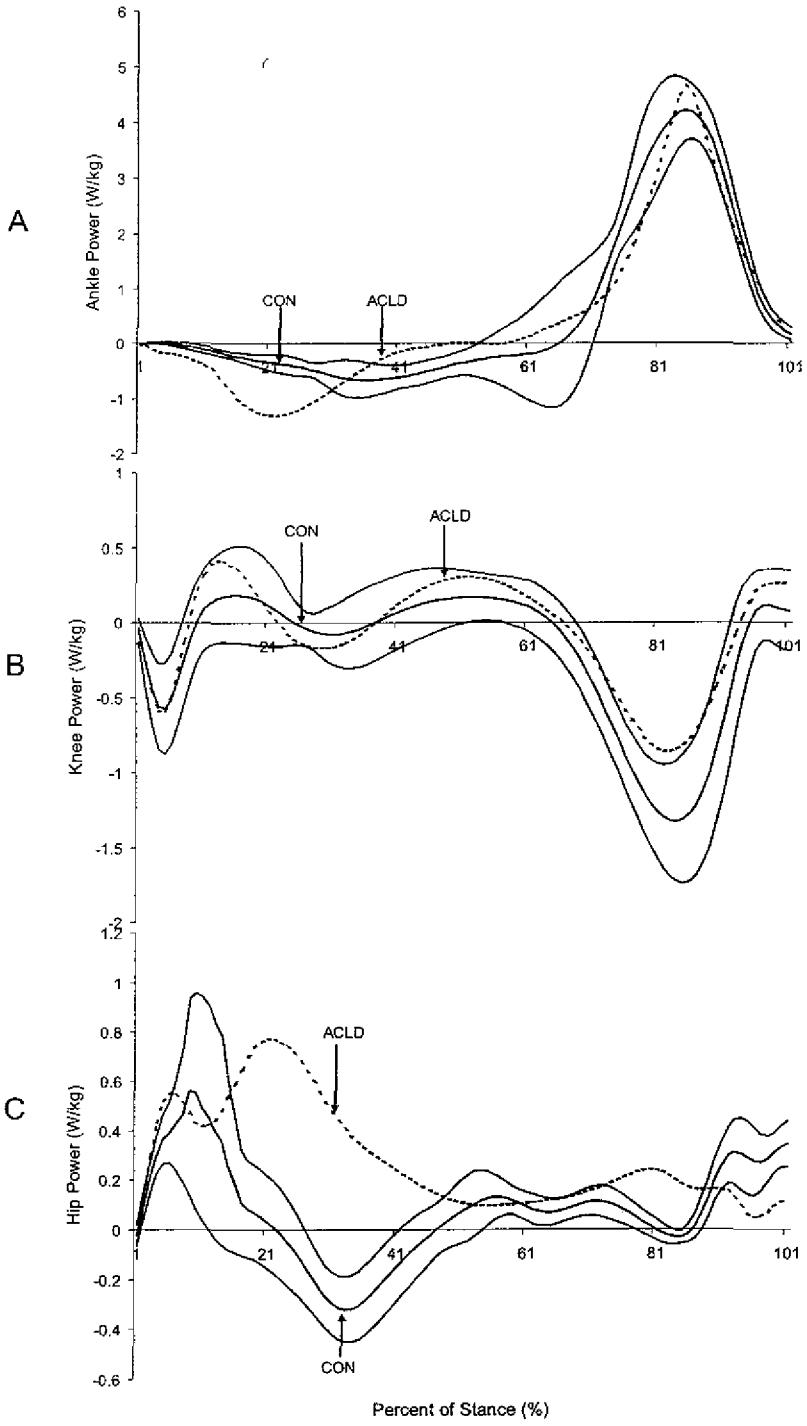

Figure 3.6. Ankle (A), knee (B), and hip (C) joint powers for anterior cruciate ligament deficient (ACLD) and control (CON) subjects during NP condition. Positive values indicate extensor and plantarflexor moments, negative values indicate flexor and dorsiflexor moments. Solid thick line and thin dotted lines are mean ± 1 SD for CON, dashed thick line is mean of ACLD group.

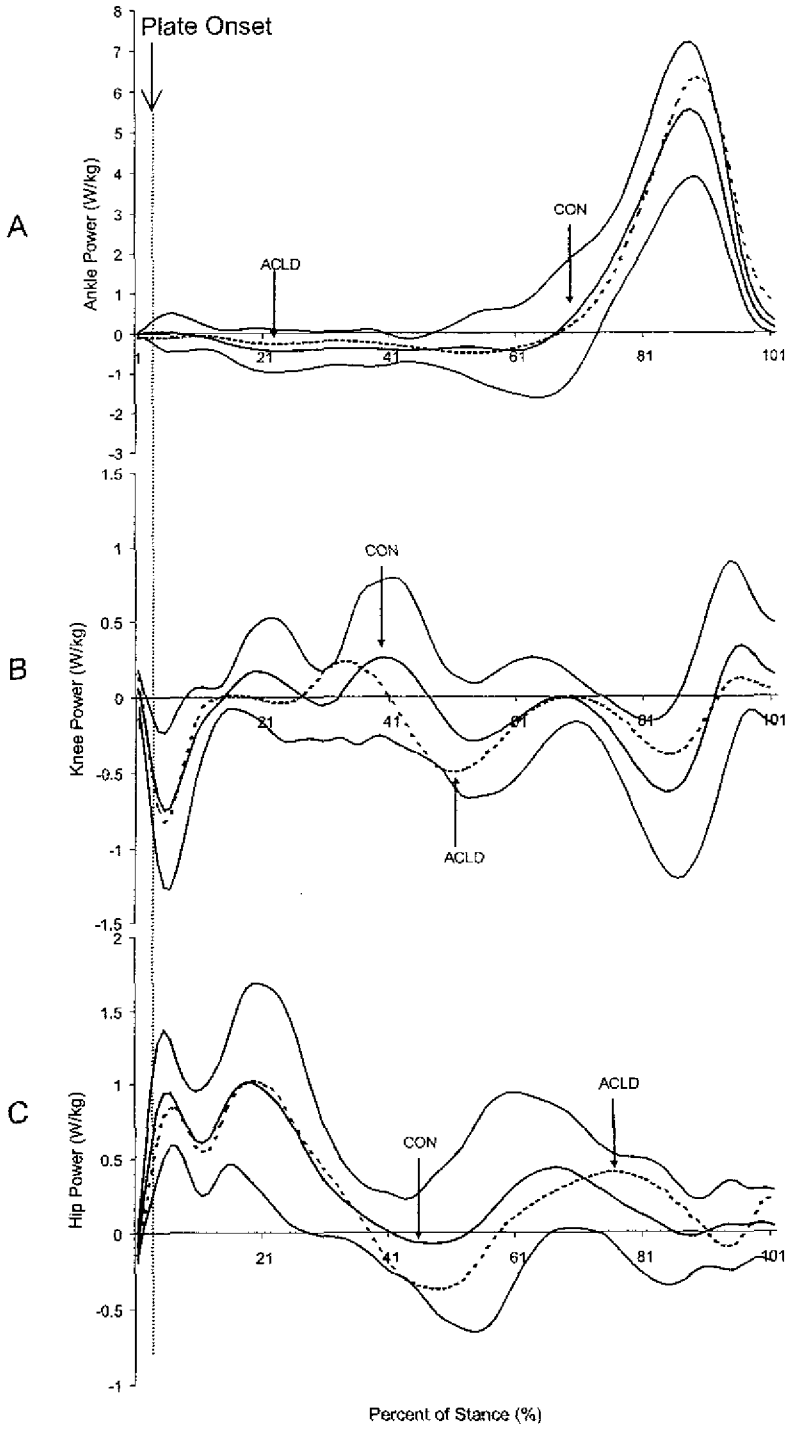

Figure 3.7. Ankle (A), knee (B), and hip (C) joint powers for anterior cruciate ligament deficient (ACLD) and control (CON) subjects during FP condition. Positive values indicate extensor and plantarflexor moments, negative values indicate flexor and dorsiflexor moments. Solid thick line and thin dotted lines are mean ± 1 SD for CON, dashed thick line is mean of ACLD group.

Muscle EMG

Non-perturbed Gait

TABLE 3.8 compares ACLD and CON lower extremity muscle EMG values during the NP condition 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 CON NP-TA muscle response was characterized by strong activation during early stance followed by a rapid decrease to a low levels for all of mid-stance and the first part of late stance with another surge of activity occurring prior to toe-off (TABLE 3.8; FIGURE 3.8A). The ACLD NP-TA EMG activity was significantly ($p<0.05$) greater than CON during early stance (Pt1, P2) and the latter part of mid-stance (Pt4, P4, Pt5; TABLE 3.8; FIGURE 3.8A).

The CON NP-GAS muscle response was characterized by a steady rise from heel strike through mid-stance and then a rapid decrease during late stance (TABLE 3.8; FIGURE 3.8B). The ACLD NP-GAS EMG activity showed a similar pattern to CON but was significantly ($p<0.05$) greater during mid-stance (Pt3, P3, Pt4; TABLE 3.8; FIGURE 3.8B).

The CON NP-VL muscle response produced a large burst of EMG activity during early stance that steadily dropped and remained relatively low throughout the remainder of stance (TABLE 3.8; FIGURE 3.8C). The ACLD NP-VL produced significantly ($p<0.05$) less EMG activity during early stance compared to CON (P1 – Pt2; TABLE 3.8; FIGURE 3.8C).

The CON NP-BF muscle response produced strong EMG activation during early stance and declined steadily for all of mid-stance and the first part of late stance after which a surge of activity was generated prior to toe-off (TABLE 3.8; FIGURE 3.8D). The ACLD NP-BF

produced significantly ($p<0.05$) greater EMG activity than CON during early stance (P1 – P2; TABLE 3.8; FIGURE 3.8D).

Forward Perturbation Gait

TABLE 3.9 compares ACLD and CON lower extremity muscle EMG values during the FP condition for P1-5 and Pt1-5 of total stance. Values expressed are the FP:NP ratio for the corresponding phase or discrete point of the NP condition for each group.

The CON FP-TA muscle response was characterized by a small burst early in stance followed by a relatively large burst that rapidly subsided until toe-off when another small burst of activity was observed (FIGURE 3.9A). The ACLD FP-TA muscle pattern was similar to CON but demonstrated significantly ($p<0.05$) less EMG activity during the last half of early stance (P2, Pt3; TABLE 3.9; FIGURE 3.9A).

The CON FP-GAS muscle response was characterized by a steady rise from heel strike through mid-stance after which it rapidly decreased during late stance (FIGURE 3.9B). The ACLD FP-GAS muscle was similar to CON but exhibited significantly ($p<0.05$) greater EMG activity during late stance (P4 – P5; TABLE 3.9; FIGURE 3.9B).

The CON FP-VL muscle response was characterized by a steady rise from heel strike through mid-stance after which it slowly decreased through to late stance (FIGURE 3.9C). The ACLD FP-VL muscle produced a similar pattern but demonstrated significantly ($p<0.05$) less EMG activity during mid-stance (Pt3 – Pt5; TABLE 3.9; FIGURE 3.9C).

The CON FP-BF muscle response was characterized by strong activation during early stance followed by a slow decline and consistently low EMG activity throughout the rest of stance (FIGURE 3.9D). The ACLD FP-BF muscle produced significantly ($p<0.05$) more EMG

activity than CON during the last part of mid-stance (P4) and all of late stance (Pt5, P5; TABLE 3.9; FIGURE 3.9D).

TABLE 3.8. Mean (±SD) of Muscle EMG Activity † for Anterior Cruciate Ligament Deficient (ACLD) and Control (CON) Subjects during NP Condition (n=10)

Stance Partition	TA		GAS		BF		VL	
Phase (P)/ Point(Pt)	CON	ACLD	CON	ACLD	CON	ACLD	CON	ACLD
P1	0.87±0.05	0.89±0.05	0.29±0.04	0.32±0.09	0.81±0.10	0.94±0.03*	0.80±0.07	0.68±0.07*
Pt1	0.87±0.04	0.96±0.03*	0.26±0.06	0.34±0.11	0.68±0.12	0.92±0.08*	0.95±0.03	0.83±0.18*
Pt2	0.90±0.12	0.95±0.06	0.26±0.07	0.33±0.11	0.63±0.12	0.87±0.14*	0.97±0.02	0.88±0.07*
P2	0.41±0.06	0.53±0.06*	0.28±0.06	0.41±0.13	0.39±0.05	0.50±0.06*	0.73±0.10	0.78±0.06
Pt3	0.31±0.16	0.40±0.14	0.34±0.07	0.56±0.05*	0.32±0.08	0.32±0.05	0.47±0.07	0.48±0.10
P3	0.31±0.11	0.39±0.13	0.41±0.12	0.68±0.13*	0.26±0.10	0.31±0.13	0.35±0.15	0.36±0.09
Pt4	0.19±0.07	0.32±0.10*	0.49±0.05	0.74±0.18*	0.36±0.04	0.35±0.06	0.30±0.12	0.32±0.12
P4	0.17±0.95	0.25±0.04*	0.79±0.06	0.85±0.07	0.37±0.18	0.35±0.15	0.30±0.04	0.36±0.04
Pt5	0.13±0.02	0.23±0.08*	0.55±0.08	0.58±0.13	0.21±0.06	0.30±0.11	0.33±0.11	0.42±0.08
P5	0.18±0.04	0.25±0.07	0.24±0.09	0.25±0.10	0.33±0.07	0.27±0.10	0.30±0.13	0.34±0.04

† Muscle EMG activity normalized to maximum amplitude within NP condition.
* Significantly different than CON ($p<0.05$)

TABLE 3.9. Mean (±SD) of Muscle EMG Activity † for Anterior Cruciate Ligament Deficient (ACLD) and Control (CON) Subjects during FP Condition (n=10)

Stance Partition	TA		GAS		BF		VL	
Phase (P)/ Point(Pt)	CON	ACLD	CON	ACLD	CON	ACLD	CON	ACLD
P1	0.87±0.09	1.04±0.08	1.15±0.09	1.18±0.05	1.24±0.12	1.11±0.10	0.75±0.18	0.90±0.11
Pt1	0.96±0.10	1.06±0.08	1.14±0.08	1.16±0.06	1.26±0.08	1.14±0.07	0.78±0.17	0.92±0.07
Pt2	0.70±0.05	0.66±0.06	1.01±0.04	1.05±0.04	1.17±0.06	0.98±0.10	0.76±0.12	0.95±0.05
P2	1.71±0.03	1.54±0.06*	0.92±0.03	0.95±0.06	1.51±0.21	1.44±0.16	1.23±0.07	1.12±0.05
Pt3	1.63±0.15	1.48±0.13*	0.12±0.22	0.44±0.10	1.39±0.05	1.24±0.19	1.55±0.14	1.27±0.04*
P3	1.17±0.03	1.13±0.06	0.80±0.03	0.86±0.05	1.41±0.13	1.30±0.21	1.51±0.14	1.26±0.04*
Pt4	0.97±0.03	0.96±0.04	1.10±0.12	1.12±0.03	1.28±0.07	1.19±0.10	1.34±0.14	1.20±0.09*
P4	1.06±0.10	0.96±0.08	0.87±0.03	1.01±0.02*	0.66±0.05	1.08±0.03*	1.23±0.07	0.99±0.12*
Pt5	0.99±0.09	0.92±0.04	0.68±0.08	0.97±0.07*	0.74±0.08	0.94±0.08*	1.12±0.13	0.80±0.09*
P5	0.98±0.03	0.99±0.02	0.85±0.06	1.13±0.04*	0.22±0.09	1.15±0.01*	1.04±0.01	1.01±0.09

† Values greater than 1.0 indicate FP EMG activity greater than NP condition, values less than 1.0 indicate FP EMG activity less than NP condition.

* Significantly different than CON ($p<0.05$)

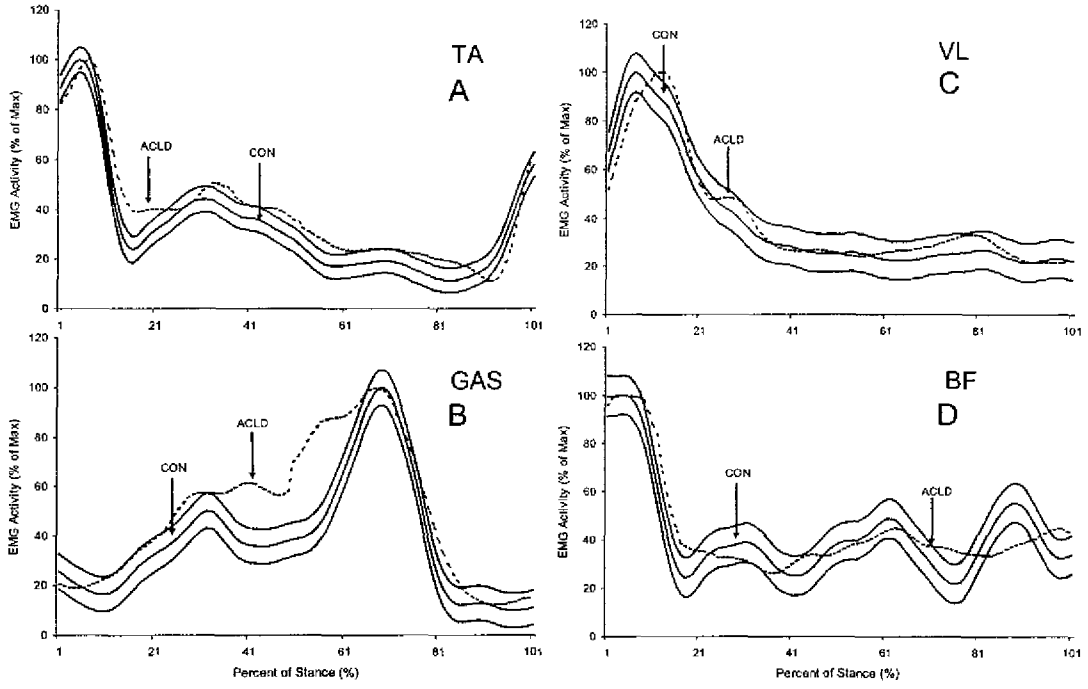

Figure 3.8. Muscle EMG activity of anterior cruciate ligament deficient (ACLD: dashed thick line) and control (CON: solid thick line and thin dotted lines are mean ± 1 SD) subjects during NP condition tibialis anterior (TA: A), gastrocnemius (GAS:B), vastus lateralis (VL: C),and biceps femoris (BF: D).

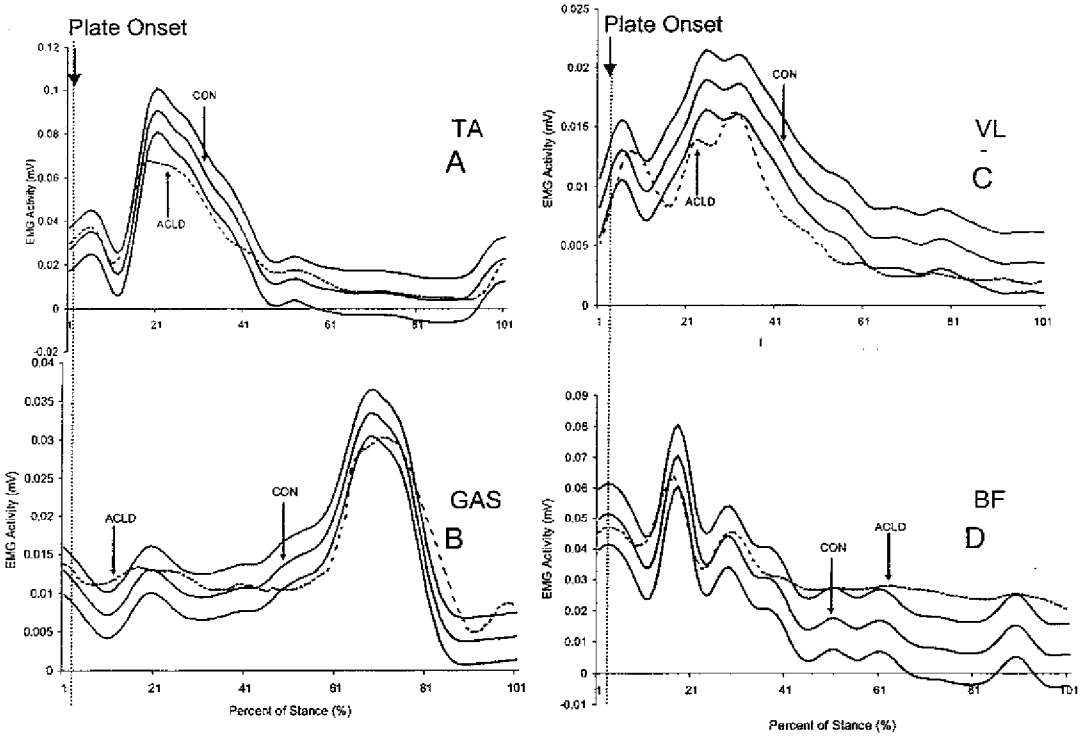

Figure 3.9. Muscle EMG activity of anterior cruciate ligament deficient (ACLD: dashed thick line) and control (CON: solid thick line and thin dotted lines are mean ± 1 SD) subjects during FP condition tibialis anterior (TA: A), gastrocnemius (GAS:B), vastus lateralis (VL: C),and biceps femoris (BF: D).

Hypotheses Results

It was hypothesized that the injured limb would exhibit a greater knee and hip extensor moment and that no subject would exhibit a quadriceps avoidance gait pattern during the NP or FP conditions. The results indicated that these hypotheses were partially confirmed as the ACLD group did not exhibit a quadriceps avoidance gait pattern but that there were no differences in knee moment (FIGURE 3.2B) patterns between the ACLD and CON groups during NP stance. However, during NP gait, the ACLD group exhibited a significantly greater hip extensor moment (FIGURE 3.2C) as compared to the CON group. During FP, the ACLD subjects exhibited a significantly greater knee extensor moment (FIGURE 3.3B) but no change in hip extensor moment (FIGURE 3.3C) patterns as compared to CON. It was also hypothesized that the injured limb would exhibit a greater knee and hip joint flexion position and increased co-activation of knee joint muscles in comparison to the uninjured controls during the NP and FP conditions. These hypotheses were partially confirmed as the ACLD demonstrated a significantly greater hip flexion (FIGURE 3.4C) but no differences in knee position (FIGURE 3.4B) as compared to the CON group during NP stance. Muscle EMG activity during NP stance revealed that the ACLD group exhibited prolonged near-maximal BF-EMG activity (FIGURE 3.8D) and a reduced rise in VL-EMG activity (FIGURE 3.8C) compared to CON. The ACLD group were significantly more flexed at the hip (FIGURE 3.5C) but no differences in knee position (FIGURE 3.5B) were observed during FP compared to CON. In response to the FP, the ACLD group exhibited less VL-EMG activity (FIGURE 3.9C) and greater BF-EMG activity compared to CON during mid and late FP-stance, respectively.

Discussion

The purpose of this study was to determine how gait patterns during non-perturbed (NP) conditions may change as a result of chronic ACLD and to determine the effect of unexpected forward perturbations (FP) on lower extremity joint moments compared to healthy controls. To date, several investigations have espoused that a "quadriceps avoidance" adaptation develops in chronic ACLD subjects due to neuromuscular re-programming possibly to reduce anterior tibial shear during the stance phase of gait (Andriacchi et al., 1993, Berchuck et al., 1990, Birac et al., 1991, Hurwitz et al., 1997; Wexler et al., 1998). However, other investigators have found no evidence of quadriceps avoidance in chronic ACLD subjects and suggest that incidence of an avoidance pattern may be less common than previously reported (Roberts et al., 1999; Rudolph et al., 1998). No studies have been conducted to study the effects of an unexpected gait perturbation on ACLD individuals. During an unexpected gait perturbation, the ability of an ACL injured individual to react and maintain equilibrium is critical in prevention of reinjury especially since individuals often encounter obstacles or perturbations while walking. With better understanding of the influence of ACLD on human locomotion, better rehabilitation protocols may be developed to prevent joint re-injury.

Non-Perturbed Gait

Quadriceps Avoidance Gait

It has been suggested that time since injury may play an important role in the type of gait adaptation observed in ACL injured patients. Several investigations (Andriacchi et al., 1993, Berchuck et al., 1990, Birac et al., 1991, Hurwitz et al., 1997; Wexler et al., 1998) have reported

that ACLD patients injured for 2 or more years tend to develop a quadriceps avoidance gait pattern indicated by a sustained knee flexor moment throughout stance. Since the quadriceps muscles can produce anterior tibial shear, development of this avoidance gait pattern was thought to reduce strain to ligamentous structures stabilizing the knee in the absence of the ACL. Other investigators have not demonstrated the quadriceps avoidance phenomenon in chronic ACLD patients (Roberts et al., 1999; Rudolph et al., 1998). In the present investigation, evidence of a quadriceps avoidance gait pattern was not observed, as chronic ACLD subjects exhibited no differences in knee moment characteristics compared to the CON group (FIGURE 3.3B).

Wexler et al. (1998) reported that 57% of all ACLD patients exhibited an avoidance pattern. Birac et al. (1991) reported an 80% incidence rate in ACLD greater than 6 years post-injury. Since all ACLD subjects in the present investigation were similar in time since injury to subjects reported by Wexler et al. (1998) and some (n=4) fell within the >6 years group reported by Birac et al. (1991), it is surprising to not observe at least one ACLD individual involved in the present study to exhibit a quadriceps avoidance gait pattern.

Roberts et al. (1999) suggested that inherent differences in the methodology used to calculate joint moments might serve to explain the quadriceps avoidance pattern. Roberts et al. (1999) used a method adopted by the Gait and Clinical Movement Analysis Society (GCMAS) that uses Euler angles with imbedded three-dimensional coordinate systems within each lower extremity segment. The model used by the group from Chicago (Andriacchi et al., 1993; Berchuck et al., 1990; Birac et al., 1991; Hurwitz et al., 1997; Wexler et al., 1998) used a simple linked segment model which assumes that flexion and extension occur on a purely sagittal plane. Furthermore, none of these investigations measured muscle EMG activity to help corroborate the existence of a sustained knee flexor moment. Roberts et al. (1999) contended that the different models themselves could be attributed to the discrepancies in reported results. However, the

present investigation also used a simple linked segment model and recorded quadriceps EMG data yet no evidence of a sustained knee flexor moment or reduced knee extensor moment during the stance phase of NP gait was observed. The results of this investigation suggest that development of a quadriceps avoidance gait pattern may be less common than previously reported.

During early NP stance, the ACLD group exhibited significantly reduced VL-EMG activity compared to CON (FIGURE 4.8C). However, examination of FIGURE 3.8C reveals that the ACLD VL muscle activity was increasing from heel strike to early mid-stance. Since NP-EMG data were normalized to the maximum within-trial amplitude, EMG amplitude relative to the CON group is not available. Therefore, the reduced VL activity can be interpreted as a reduced rise (slope) in VL-EMG activity toward maximum activation. The reduced slope could result from maximum VL activation occurring slightly later in NP stance in the ACLD group compared to CON (FIGURE 4.8C). It is possible that the later maximum activation and subsequent reduced rise in activation was a strategy to help reduce anterior tibial strain in the ACLD group.

Although alterations in knee joint moments may provide important information regarding development of neuromuscular adaptations as a result of ACLD, identifying alterations in moments at the ankle and hip joints during NP gait may be equally important. Winter (1980) suggested that when one joint opposes or does not contribute to vertical support of the body, one or both of the other joints must compensate to prevent collapse of the lower extremity. To support this premise, Winter calculated an overall moment of support from the algebraic summation of ankle, knee, and hip moments. An overall positive moment of support indicates the tendency of the lower limb to resist collapse of the lower extremity as the result of downward gravitational forces. Andriacchi et al. (1993) and Berchuck et al. (1990) suggested that a

sustained knee flexor moment is indicative of a quadriceps avoidance gait pattern. In this circumstance, the ACLD knee would contribute little to the vertical support of the body throughout stance and the hip and ankle joint must compensate. Berchuck et al. (1990) reported an increased hip extensor moment during NP gait that could somewhat compensate for a sustained knee flexor moment but reported no alterations in ankle moments. Wexler et al. (1999), Andriacchi et al. (1990, 1993) and Birac et al. (1991) provided no information regarding ankle or hip moments. Based on these data, it is unknown whether ACLD subjects who demonstrated a quadriceps avoidance gait pattern in previous investigations exhibited an overall positive moment of support, a finding that would help to shed light on the development of a quadriceps avoidance pattern.

Gait Adaptations to ACLD

Previous investigations (Berchuck et al., 1990; Roberts et al., 1999; Wexler et al., 1998) have reported that ACLD subjects were more flexed at the hip and knee joints and exhibited a greater hip extensor moment during stance. In the present study, ACLD subjects demonstrated no significant differences from the CON group subjects in knee joint position, throughout stance, but did demonstrate significantly greater ankle plantarflexion and hip flexion throughout most of NP stance (FIGURE 3.1A & 3.1C). During NP in the present study, the ACLD subjects also demonstrated a significantly greater and prolonged hip extensor moment compared to the CON group (FIGURE 3.3C). A more flexed hip position would possibly demand a greater hip extensor moment early in NP stance to maintain control and reduce the forward acceleration of the upper body. However, Devita et al. (1997) suggested that a fundamental change in the length-tension relationship in hip extensor muscles of ACLD subjects during the stance phase of gait may occur as a result of an increased hip flexion position possibly to help reduce anterior tibial shear. If

knee position of the ACLD 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.

Pandy and Shelburne (1997) and Li et al. (1999) reported that anterior tibial shear increased from full extension to 15° flexion, then decreased as knee flexion position increased. The knee is near full extension at two points during NP stance: following heel strike and following midstance (FIGURE 3.10). 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 shear (Pandy & Shelburne, 1997; Osternig et al., 2000). In the present investigation ACLD subjects demonstrated a significantly greater hip extensor moment (FIGURE 3.3C), reduced rise in VL EMG activity (FIGURE 3.7C), and prolonged near-maximal BF EMG activity (FIGURE 3.7D) compared to CON following heel strike (FIGURE 3.3C). It is possible that the increase in the hip extensor moment and reciprocal activity of the thigh muscles exhibited by ACLD subjects was necessary to reduce anterior tibial shear when strain to the knee joint would be greatest. Following midstance, CON subjects demonstrated a hip flexor moment while ACLD subjects exhibited a prolonged hip extensor moment (FIGURE 3.3C). It is possible that CON subjects were able to produce a hip flexor moment following midstance since the intact ACL was able to restrain the tibia posteriorly and it was therefore not necessary to exhibit hamstring muscle activation in the form of a hip extensor moment. In the absence of an ACL, the hip extensor moment following midstance exhibited by ACLD subjects may have been necessary in order to reduce anterior tibial shear at the second point of NP stance when anterior tibial shear is greatest.

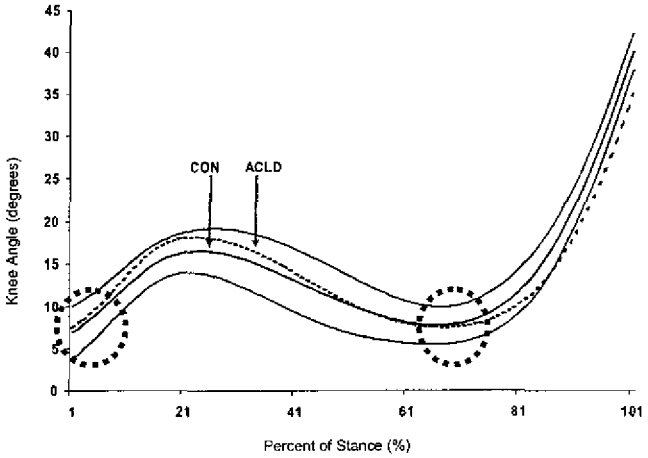

FIGURE 3.10. Knee joint position curves for ACLD (dashed thick lines) and CON (solid thick and thin lines = mean ± 1SD) subjects during NP gait. Positive values indicate flexion, negative values indicate extension. Dashed circles indicate two times during of stance when the knee joint is near full extension and anterior tibial shear is greatest.

Coinciding with the prolonged hip extensor moment following mid-stance, ACLD subjects demonstrated significantly greater hip extensor power generation (FIGURE 3.5C), a knee flexor moment (FIGURE 3.3B), and significantly reduced knee flexor muscle power absorption (FIGURE 3.5B). It is possible that the reduction in knee power absorption in ACLD subjects was due to the bi-articular action of the hamstring muscles that were simultaneously generating extensor power possibly in order to stabilize the tibia when the knee was near full extension during NP stance.

The ACLD subjects exhibited alterations in ankle joint gait patterns that may also serve to reduce anterior tibial shear. The gastrocnemius muscle has a bi-articular role during gait functioning at the ankle joint as a plantarflexor and the knee joint as a flexor and may also serve to stabilize the knee during stance (O'Connor, 1993). During the first half of NP stance, ACLD subjects exhibited significantly greater ankle power absorption and greater GAS EMG activity than the CON group (FIGURE 3.1A, 3.5A &3.7B). With greater ankle plantarflexor power

absorption should come greater power generation and an increase in the plantarflexor moment as the ankle begins to plantarflex for the last half of NP stance. However, late in NP stance, a reduction in ankle plantarflexor moment (FIGURE 3.3A), and no significant differences in GAS EMG activity or ankle power generation, were observed in ACLD subjects. It is possible that the energy absorbed in the ankle plantarflexor muscles during the first half of stance while the ankle was dorsiflexing was necessary to help stabilize the knee following midstance as the knee approached an extended position.

Winter (1987) hypothesized that the central nervous system (CNS) is programmed to control the amount of knee flexion during stance. It is possible that ACLD subjects developed a kinematic pattern necessary to reduce anterior tibial shear by increasing the ankle plantarflexion and hip flexion positions with no change in knee flexion position. Compared to CON, the leg segment of the ACLD subjects must rotate posteriorly allowing for a more plantarflexed ankle position. To allow for no change in knee angle, the thigh segment must be more horizontal while the HAT segment orientation remains unchanged compared to CON (FIGURE 3.11). It is possible that the ACLD subjects developed this gait strategy in response to pain and/or excessive and continual anterior tibial shear upon heel strike following ACL injury. In light of observing no significant differences in the knee moment patterns between ACLD and CON subjects it is not surprising to observe between group differences in hip and ankle moments. Data from this investigation suggest that ACLD subjects appear to accommodate to chronic ACL deficiency through alterations of hip and ankle joint gait patterns, possibly to reduce anterior tibial shear during the stance phase of gait. Future investigations involving ACLD subjects should include data from all three major lower extremity joints to better understand joint accommodations as a result of ACLD.

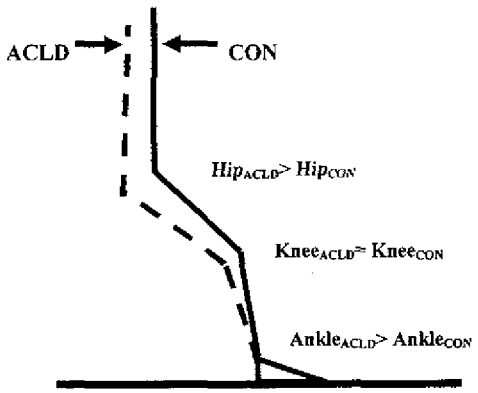

FIGURE 3.11. Representative example of ankle, knee, and hip joint positions of ACLD (dashed line) and CON (solid line) subjects during the NP condition. ACLD demonstrated significantly more ankle plantarflexion and hip flexion with no change in knee position.

Response to Unexpected Forward Perturbations During Gait

As previously discussed, an overall positive moment of support indicates the tendency of the lower limb to prevent collapse of the body due to downward gravitational forces. In response to an unexpected FP, a strong knee extensor moment is necessary during FP midstance to support the body against vertical collapse, because the initial hip extensor moment that quickly reverses direction and the ankle plantarflexor moment are small (FIGURE 3.1). Hypothetically, a quadriceps avoidance pattern would manifest itself under the FP condition but this was not the case in the present study; rather than a reduction in the knee extensor moment, a significantly greater knee extensor moment was produced near midstance as compared to the CON group (FIGURE 3.4B). It should be noted that other investigations (Andriacchi et al., 1993; Berchuck et al., 1990) have demonstrated that ACLD subjects demonstrated quadriceps avoidance only

during NP gait and not while ascending stairs, an activity that also places a relatively large demand on knee extensor muscles compared to normal walking. In response to an unexpected FP, the knee is held in a more static position compared to NP gait (FIGURE 3.2B & 3.3B). In a more static position, the knee is more stable, possibly allowing for a greater net knee extensor moment to prevent vertical collapse. Prevention of vertical collapse in the present study may be of greater necessity than during stair ascent due to the nature and intensity of the unexpected FP. This possibility is supported by the finding that the ACLD subjects demonstrated a significantly greater ankle plantarflexion position compared to CON during FP. In the ankle plantarflexion position, the thigh and HAT segments are now located further away from the knee joint center thereby demanding a greater knee extensor moment to prevent vertical collapse (FIGURE 3.11).

The ACLD subjects demonstrated significantly less VL EMG activity in response to the FP compared to CON. It is important to note that the FP EMG values were normalized to NP EMG activity. Therefore, it can be interpreted that ACLD subjects demonstrated FP-VL EMG activity that was more similar to NP-VL EMG activity compared to the CON group. However, since ACLD subjects demonstrated a significantly greater knee extensor moment during mid-stance compared to the CON group, it is likely that the magnitude of the FP-VL EMG activity was also greater than the CON group.

In Chapter II it was reported that CON demonstrated a significantly reduced ankle plantarflexor moment in an effort to maintain a static knee flexion position during the FP. During the FP, the ACLD subjects demonstrated a significantly reduced ankle plantarflexion moment but no change in knee position compared to the CON group. Perhaps the similar knee position, exhibited by the ACLD and CON subjects, was ideal to maintain dynamic equilibrium, maintain knee joint stability, and minimize anterior tibial shear during the FP. A stronger than normal ankle plantarflexor moment can serve to slow down, or even reverse forward rotation of the leg

segment, resulting in knee extension (Winter, 1990a). The reduced ankle plantarflexor moment exhibited by the ACLD subjects early in FP stance could serve to maintain knee position and thus reduce tibial shear.

It is interesting that the ACLD subjects demonstrated similar kinematic gait patterns in both the NP and FP conditions, exhibiting significantly greater ankle plantarflexion and hip flexion with no change in knee position compared to the CON group. As previously discussed, perhaps ACLD subjects developed this gait strategy to place the knee in a more stable position at heel strike in response to pain and/or to prevent excessive anterior tibial shear. If the ACLD subjects have altered their lower extremity joint trajectories in preparation for heel strike, this adaptation would still be observed during the FP condition as the unexpected FP is applied approximately 27ms following heel strike (~3% of stance).

Summary

The data did not reveal a quadriceps avoidance gait pattern in the ACLD subjects during the NP or FP conditions. The ACLD subjects appeared to accommodate to chronic ACL deficiency through alterations of hip and ankle joint kinematic and kinetic and muscle power patterns, possibly in an effort to reduce anterior tibial shear during the stance phase of NP gait. In response to an unexpected FP, ACLD subjects demonstrated greater knee extensor muscle activity that is necessary to prevent collapse.

Bridge

The first two studies characterized the kinetic, kinematic, and muscle activation responses to an unexpected FP in ACLD and non-injured young adults. Studies involving gait adaptations to ACL injury and subsequent surgical repair are limited and no studies have been conducted to investigate how ACL surgically repaired individuals respond to an unexpected FP during gait. Therefore, the purpose of the third study was to investigate how normal gait patterns of chronic ACLD subjects may change as a result of ACL surgical repair and to determine the effect of unexpected FP on lower extremity joint moments, power, and kinematics on ACL repaired subjects. Chapter IV summarizes the similarities and differences in normal walking patterns and the reactive balance responses evoked during unexpected FP between non-injured and pre- and post-surgical ACLD individuals.

CHAPTER IV

GAIT PERTURBATION RESPONSE IN ANTERIOR CRUCIATE LIGAMENT DEFICIENCY AND SURGERY

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. Consensus of opinion is that these gait patterns develop as a result of muscle adaptations and neuromuscular reprogramming, possibly in response to pain and/or instability, to stabilize the knee and to prevent re-injury during gait (Berchuck et al., 1990; Devita et al., 1997; Wexler et al., 1998). It has also been demonstrated that acute (<1 month) ACL injured patients exhibit significantly different knee moment patterns compared to chronic (>2 years) ACL deficient (ACLD) subjects during gait (Berchuck et al., 1990; Devita et al., 1997). Individuals who have recently suffered ACL injury exhibit 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 hypothesized that, over time, ACLD individuals develop a sustained knee flexor

moment during mid-stance termed a "quadriceps avoidance gait", possibly serving to reduce anterior tibial shear during gait (Andriacchi et al., 1993; Berchuck et al., 1990; Birac et al., 1991; Hurwitz et al., 1997; Wexler et al., 1998). However, other investigations have found no evidence of a quadriceps avoidance gait pattern and suggest that knee moment patterns of chronic ACLD subjects resume pre-injury knee moment characteristics (Ferber, 2001; Roberts et al., 1999; Rudolph et al., 1998).

Clinical studies have reported that approximately one-third of ACLD 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 (Noyes et al., 1983). Reconstructive surgery is sometimes used to reestablish functional and mechanical stability of the knee in those ACLD 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 (ACLR) subjects are limited and suggest that time since surgery may play an important role in the return of normal gait patterns (Bulgheroni et al., 1997; Bush-Joseph et al., 2001; Cicotti et al., 1994; Devita et al., 1998; Ernst et al., 2000; Timoney et al., 1993). Devita et al. (1998) examined ACLR 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 ACLR subjects 3 weeks post-surgically. However, at 6 months following surgery, ACLR subjects demonstrated knee and hip moment patterns more similar to control group values suggesting that ACLR subjects can regain pre-injury gait characteristics. Bush-Joseph (2001) studied a group of ACLR subjects 8 months after surgery and reported only slight reductions in the peak knee extensor moment during gait. However, it was also reported

that 2 ACLR subjects exhibited a quadriceps avoidance gait pattern. Timoney (1993) reported that at 10 months post-surgery, ACLR 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. (1997) studied the gait patterns of ACLR subjects 2 years post-operatively and reported no significant differences in sagittal plane knee or hip moments suggesting that, given time, ACLR subjects can regain normal knee moment gait patterns.

The time between injury and surgery may also influence the type of gait pattern observed in ACLR subjects. Few comprehensive gait studies have investigated ACL injured subjects prior to and following surgical repair (Devita, 1997). Devita (1997) examined the gait patterns of ACL injured subjects 2 weeks after ACL injury but before surgery and 3 and 5 weeks post-surgically. It was demonstrated that 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, ACL injured subjects involved in this investigation were acutely injured ACL patients who also exhibited a sustained knee extensor moment pre-surgically. It is therefore not known whether the post-surgical gait pattern exhibited by the ACLR subjects resulted from ACL injury, reconstructive surgery, or both factors. It is possible that subjects who had sustained ACL injury 2 or more years prior to surgery would develop different gait patterns following surgery as a result of the mechanical stress of ACL reconstructive surgery. Additional studies are needed to better understand the neurological and mechanical influences that chronic ACLD and subsequent surgical repair have on the development of gait patterns. Furthermore, few studies have examined the effects of unexpected gait perturbations on ACLD and ACLR individuals.

Walking involves the integration of muscular contractions across different joints in an effort to initially lose, then regain dynamic equilibrium as the body is propelled through space. During an unexpected gait perturbation, the ability of an ACL injured individual to react and maintain equilibrium is critical for the prevention of reinjury, especially since individuals often encounter obstacles or perturbations during gait. Few studies have been conducted that quantify reactive gait alterations due to unexpected gait perturbations (Nashner, 1980, Tang, 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 EMG activity in comparison to the unperturbed condition. However, these investigations only provided EMG and kinematic data. In Chapter II it was reported that the muscles surrounding the hip were found to be most important in maintaining control of the upper body and preventing collapse of the lower extremity as an initial response to the FP (Ferber, 2001). However, later in stance, the ankle, knee, and hip joints demonstrated significantly different joint moment patterns compared to normal gait in order to maintain dynamic equilibrium. In Chapter III it was reported that ACLD individuals demonstrate a significantly greater knee extensor moment in response to an unexpected forward perturbation compared to healthy individuals (Ferber, 2001). However, how these same individuals ambulate during normal walking and in response to an unexpected perturbation 3 month following reconstructive surgery is unknown. Therefore, the purpose of this study was twofold: 1) to determine how normal gait patterns may change as a result of ACL reconstructive surgery and 2) to determine the effect of unexpected forward perturbations on ACLR subjects compared to pre-surgical values and healthy controls.

It was hypothesized that 3 months following reconstructive surgery the ACL repaired limb would demonstrate a sustained knee extensor moment, greater hip extensor moment, greater hip and knee flexion, and increased knee extensor muscle EMG activity during perturbed and unperturbed walking.

Method

Subjects

Ten (5 males and 5 females) ACL deficient (ACLD) individuals aged 18-40 years were recruited as subjects for this study. All ACLD 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 (range 2.2yr – 16.1yr; mean 5.7 yr). All ACLD 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. Demographic information regarding these subjects was described in Chapter III.

Ten (5 males and 5 females) healthy uninjured young adults also participated in the study as control subjects (CON). Demographic information regarding these subjects was described in Chapter II. Prior to participation, each subject signed a consent form (APPENDIX A) approved by the Human Subjects Compliance Committee at the University of Oregon.

Experimental Apparatus and Protocol

The experimental apparatus and protocol were the same as described in Chapter II. The ACLD subjects completed the same 48 walking trial test 3 months following ACL surgical repair. Following surgery, the ACLD subjects were identified as ACL reconstructed (ACLR) subjects.

Data Analysis

Data analysis was the same as that described in Chapter II. Individual joint moment, power, and position and muscle EMG curves were divided into 5 phases of the stance phase according to discrete kinetic events along with selection of 5 discrete points for analysis.

Statistical Analysis

Three-way repeat measures ANOVAs (10 x 3 x 2) were used to determine differences, if any, between the ACLR vs. ACLD and ACLR vs. CON groups within the true control NP and FP conditions. The independent variables were 1) the 5 phases and 5 discrete points of stance and 2) the three groups (ACLD, ACLR, and CON), and 3) the two conditions (true control NP and FP). The dependent variables were joint (ankle, knee, hip) 1) moment, 2) power, 3) position, and muscle (TA, GAS, BF, VL) EMG magnitude. Since the results of ACLD vs. CON have been presented in CHAPTER III, a priori post-hoc tests were performed to detect differences, if any, between ACLR and ACLD and between ACLR and CON within the NP and FP conditions. Additional a priori post-hoc tests were performed to detect differences, if any, between catch NP and true control NP trials within the ACLR group. A maximum α level of 0.05 was used to indicate statistical significance.

Results

This study was conducted to determine how normal gait patterns may change as a result of ACL reconstructive surgical repair and to investigate the effect of unexpected forward perturbations during gait on lower extremity joint kinematics, moments, powers, and muscle EMG patterns. Chronic ACLD subjects were tested prior to, and following, surgical repair. Since the results of the ACLD versus CON were presented in Chapter III, results presented here are limited to ACLR versus CON and ACLR versus ACLD. In this section, descriptive measures of stance are presented first followed by lower extremity joint moments, joint kinematics, joint powers, and muscle EMG responses for 3 general aspects of stance: 1) early stance from heel strike to Pt3 including P1, Pt1, Pt2, and P2, 2) mid-stance from Pt3 to Pt5 including Pt3, P3, Pt4, and P5, and 3) late stance from Pt5 to toe-off including Pt5 and P5.

Descriptive Measures of Stance

Time

There were no differences in total time of stance between the CON (NP: 863.06 ± 77.27ms; FP: 977.14 ± 58.33ms), ACLD (NP: 865.08 ± 52.22ms; FP: 962.33 ± 77.00ms), and ACLR (NP: 853.22 ± 72.33ms; FP: 907.20 ± 72.24ms) groups for the NP or FP conditions.

Trials

No significant ($p>0.05$) differences were found between the true control blocked NP trials and the randomized catch NP trials for any lower extremity variable for the ACLR group.

Moment of Support

The results revealed an overall positive moment of support (Ms) for the NP and FP conditions (FIGURE 4.1). There were no significant differences ($p>0.05$) in total extensor angular impulse (EAI) between the CON (NP: 75.5 ± 14.23 Nm/kg; FP: 65.91 ± 18.99 Nm/kg), ACLD (NP: 80.50 ± 25.85 Nm/kg; FP: 67.55 ± 28.55 Nm/kg), and ACLR (NP: 81.00 ± 25.56 Nm/kg; FP: 72.79 ± 27.95 Nm/kg) groups for the NP or FP conditions (FIGURE 4.1).

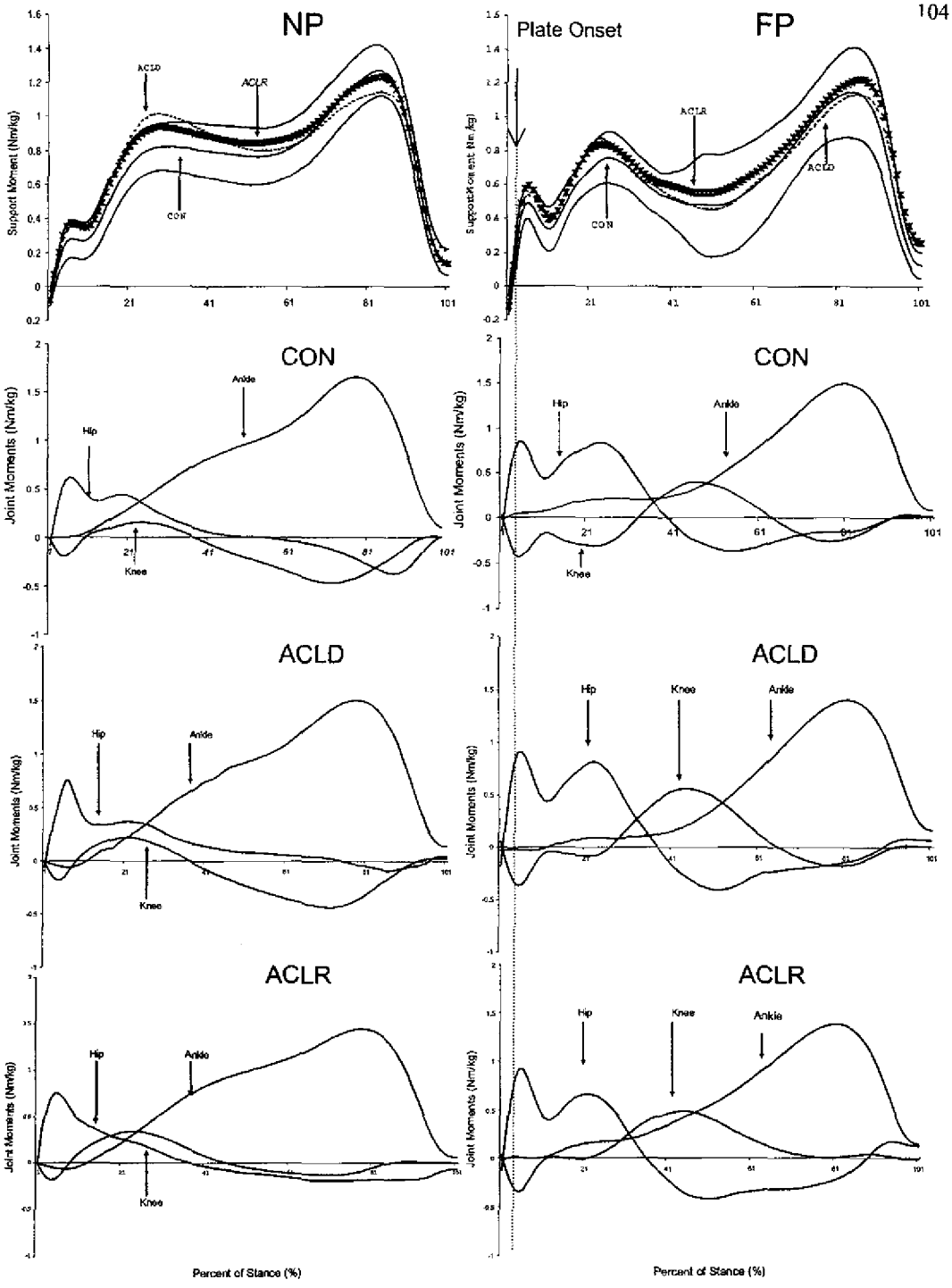

Figure 4.1. *Moment of support (top graphs) and overlay of individual joint moments for CON, ACLD, and ACLR group during NP (left graphs) and FP (right graphs) conditions.* Solid thick line and thin dotted lines are mean ± 1SD for control subjects. Positive values are net extensor and plantarflexor moments and negative values are net flexor and dorsiflexor moments.

Joint Moments

Non-perturbed Gait

TABLE 4.1 presents a comparison of ACLD, ACLR, and CON lower extremity joint moments during the NP condition for each of the 5 phases (P) and 5 discrete points (Pt) of total stance as well as the total joint EAI for stance. No significant ($p>0.05$) differences in ankle or hip EAI were observed between ACLR and CON or between ACLR and ACLD; however, the ACLR subjects exhibited significantly ($p<0.05$) greater knee NP-EAI than CON subjects (TABLE 4.1).

The CON ankle NP plantarflexor moment rose steadily from heel strike through mid-stance to Pt5 before declining rapidly during the latter half of P5 (TABLE 4.1; FIGURE 4.2A). In contrast, the ACLR ankle NP demonstrated a dorsiflexor moment ($p<0.05$) during early stance (P1-Pt2) but then paralleled the CON NP ankle plantarflexor moment during midstance but generated a significantly ($p<0.05$) smaller ankle plantarflexor moment during late stance (P4, Pt5; TABLE 4.1; FIGURE 4.2A).

The CON knee NP moments demonstrated an initial flexor moment in early stance followed by a biphasic extensor-flexor-extensor moment pattern for early, mid-, and late stance periods, respectively (TABLE 4.1; FIGURE 4.2B). The ACLR knee NP moment curve demonstrated a similar biphasic pattern but significantly ($p<0.05$) greater extensor moment during the first half of mid-stance (P2-P3) compared to CON (TABLE 4.1; FIGURE 4.2B). During the latter half of stance (Pt4-P5), ACLR exhibited a significantly ($p<0.05$) smaller flexor moment compared to CON and ACLD (TABLE 4.1; FIGURE 4.2B).

The CON hip NP extensor moment rose sharply in early stance and then decreased steadily until mid-stance after which a flexor moment was observed (TABLE 4.1; FIGURE 4.2C). The ACLR hip NP extensor moment was significantly ($p<0.05$) greater during early stance (Pt1, Pt2, P2) and during the latter half of mid-stance (P4) compared to CON (TABLE 4.1;

FIGURE 4.2C). The ACLR subjects exhibited a significantly ($p<0.05$) greater NP flexor moment during the first half of mid-stance (P3, Pt4) compared to ACLD (TABLE 4.1; FIGURE 4.2C).

Forward Perturbation Gait

TABLE 4.2 presents a comparison of ACLD, ACLR, and CON lower extremity joint moments during the FP condition 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 ankle or hip FP-EAI were observed between CON, ACLR, and ACLD groups but ACLR did demonstrate significantly ($p<0.05$) greater knee EAI compared to CON (TABLE 4.2).

The CON and ACLR ankle FP plantarflexor moments paralleled each other ($p>0.05$) throughout stance rising slowly from heel strike to midstance then increasing rapidly during the latter half of stance (TABLE 4.1; FIGURE 4.2). No significant ($p>0.05$) differences were observed between the ACLR and ACLD ankle FP moment throughout stance (TABLE 4.2; FIGURE 4.3A).

The CON knee FP moment exhibited a flexor-extensor-flexor pattern through early, mid-, and late stance, respectively (TABLE 4.2; FIGURE 4.3B). The ACLR knee FP 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 CON (TABLE 4.2; FIGURE 4.3B). During the latter half of stance (P4, Pt5), the ACLR knee produced a sustained knee extensor moment ($p>0.05$) compared to the CON and ACLD knee FP flexor moments (TABLE 4.2; FIGURE 4.3B).

The CON, ACLR, and ACLD hip FP 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 4.2; FIGURE 4.3C).

TABLE 4.1. Mean (±SD) of Ankle, Knee, and Hip Joint Moments † for Control (CON), Anterior Cruciate Ligament Deficient (ACLD), and Anterior Cruciate Ligament Reconstructed (ACLR) Subjects During NP Condition (n=10)

Stance Partition	Ankle			Knee			Hip		
Phase (P)/ Point(Pt)	CON	ACLD	ACLR	CON	ACLD	ACLR	CON	ACLD	ACLR
P1	0.02±0.02	-0.04±0.02*	-0.03±0.03*	-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	0.01±0.04	-0.06±0.03*	-0.06±0.03*	-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	0.02±0.05	-0.07±0.04*	-0.06±0.06*	-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	0.22±0.01	0.15±0.08*	0.11±0.09*	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	0.50±.016	0.47±0.18	0.39±0.20	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	0.83±0.09	0.79±.014	0.80±0.18	-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	0.97±0.11	0.94±0.11	0.96±0.16	-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	1.36±0.09	1.26±0.06*	1.26±0.05*	-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	1.65±.014	1.49±0.12*	1.47±0.12*	-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	0.98±0.05	0.94±0.12	0.88±0.12*	-0.18±0.04	-0.10±0.05	-0.01±0.05*#	-0.07±0.05	0.11±0.01*	-0.01±0.10
EAI	78.8±4.54	75.2±6.62	74.28±12.01	3.44±2.34	6.97±3.57	9.92±3.34*	12.93±7.19	16.14±2.05*	11.36±5.8

† Positive values indicate extensor and plantarflexor moments, negative values indicate flexor and dorsiflexor moments (Nm/kg)
* Significantly different than CON ($p<0.05$)
# Significantly different than ACLD ($p<0.05$)

TABLE 4.2. Mean (±SD) of Ankle, Knee, and Hip Joint Moments † for Control (CON), Anterior Cruciate Ligament Deficient (ACLD), and Anterior Cruciate Ligament Reconstructed (ACLR) Subjects During FP Condition (n=10)

Stance Partition	Ankle			Knee			Hip		
Phase (P)/ *Point*(Pt)	CON	ACLD	ACLR	CON	ACLD	ACLR	CON	ACLD	ACLR
P1	0.03±0.02	-0.02±0.01*	-0.01±0.02	-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	0.04±0.01	-0.02±0.01*	-0.01±0.01	-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	0.03±0.01	-0.03±0.02*	-0.01±0.01	-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	0.17±0.06	0.09±0.06*	0.11±0.05	-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	0.26±0.13	0.16±0.09	0.26±0.14	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	0.57±0.21	0.46±0.13	0.57±.019	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	0.77±0.21	0.71±0.22	0.81±0.21	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	1.28±0.16	1.19±0.05	1.21±0.06	-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	1.51±0.12	1.41±0.14	1.41±0.19	-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	0.91±0.08	0.91±0.11	0.90±0.11	-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	58.51±9.64	51.11±5.94	56.92±10.98	8.39±2.66	14.79±7.66	15.28±8.7*	22.66±7.25	23.61±10.38	19.40±5.46

† Positive values indicate extensor and plantarflexor moments, negative values indicate flexor and dorsiflexor moments (Nm/kg)
* Significantly different than CON ($p<0.05$)

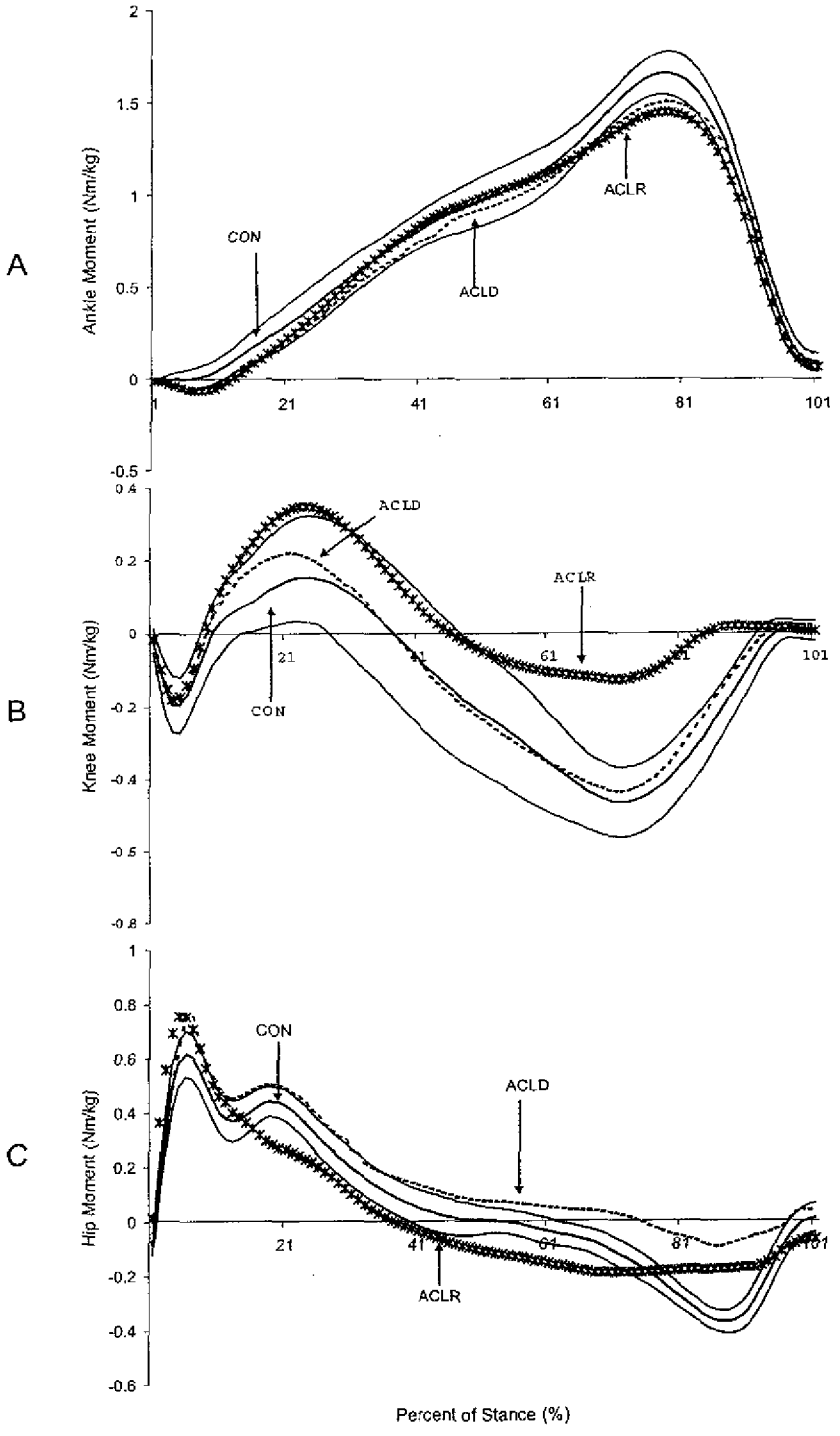

Figure 4.2. Ankle (A), knee (B), and hip (C) joint moments for anterior cruciate ligament deficient (ACLD), anterior cruciate ligament reconstructed (ACLR), and control (CON) subjects during NP condition. Positive values indicate extensor and plantarflexor moments, negative values indicate flexor and dorsiflexor moments. Solid thick line and thin dotted lines are mean ± 1 SD for CON, dashed thick line is mean of ACLD, and dashed hatches are mean of ACLR group.

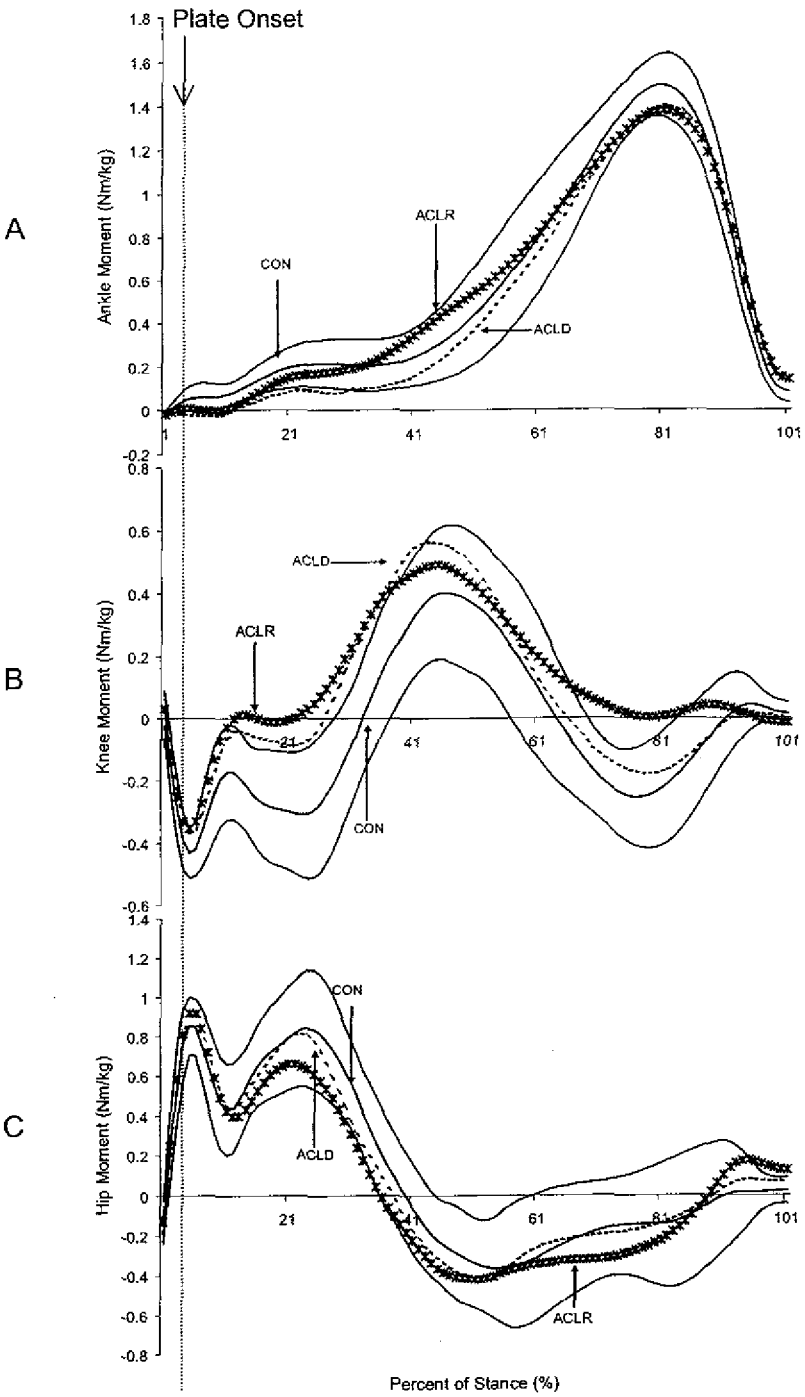

Figure 4.3. Ankle (A), knee (B), and hip (C) joint moments for anterior cruciate ligament deficient (ACLD), anterior cruciate ligament reconstructed (ACLR), and control (CON) subjects during FP condition. Positive values indicate extensor and plantarflexor moments, negative values indicate flexor and dorsiflexor moments. Solid thick line and thin dotted lines are mean ± 1 SD for CON, dashed thick line is mean of ACLD, and dashed hatches are mean of ACLR group.

Joint Kinematics

Non-perturbed Gait

TABLE 4.3 presents a comparison of ACLD, ACLR, and CON lower extremity joint position values during the NP condition for P1-5 and Pt1-5 of total stance. The CON ankle NP position curve followed a plantarflexion-dorsiflexion-plantarflexion pattern over early, mid-, and late stance periods, respectively (TABLE 4.3; FIGURE 4.4A). The ACLR ankle NP position curve generally paralleled the CON NP curve but was significantly ($p<0.05$) more plantarflexed throughout early stance (Pt2-Pt3) and late stance (P5; TABLE 4.3; FIGURE 4.4A). No significant ($p>0.05$) differences were observed between the ACLR and ACLD ankle NP position throughout stance (TABLE 4.3; FIGURE 4.4A).

The CON knee NP position curve demonstrated a flexion-extension-flexion pattern over early, mid-, and late stance respectively (TABLE 4.3; FIGURE 4.4B). The ACLR knee NP paralleled CON throughout stance but was significantly ($p<0.05$) more flexed during the latter half of stance (P3-Pt5; TABLE 4.3; FIGURE 4.4B). No significant ($p>0.05$) differences were observed between the ACLR and ACLD knee NP position throughout stance (TABLE 4.3; FIGURE 4.4B).

The CON hip NP position curve declined steadily from a flexed position to extension from early to mid-stance respectively after which it followed a flexion-extension pattern from the latter half of mid- to late stance (TABLE 4.3; FIGURE 4.4C). The ACLR hip NP curve generally paralleled the CON NP curve but was significantly ($p<0.05$) more flexed during early stance (P1, Pt1, Pt2, P2) and the first half of mid-stance (Pt3-Pt4; TABLE 4.3; FIGURE 4.4C). No significant ($p>0.05$) differences were observed between the ACLR and ACLD hip NP position throughout stance (TABLE 4.3; FIGURE 4.4C).

Forward Perturbation Gait

TABLE 4.3 presents a comparison of ACLD, ACLR, and CON lower extremity joint position values during the FP condition for P1-5 and Pt1-5 of total stance. During FP, the CON and ACLR ankle FP position curves paralleled one another ($p>0.05$) demonstrating steady dorsiflexion until midstance when the ankle began to plantarflex for the remainder of stance (TABLE 4.4; FIGURE 4.5A). No significant ($p>0.05$) differences were observed between the ACLR and ACLD ankle FP position throughout stance (TABLE 4.4; FIGURE 4.5A).

The ACLD, ACLR, and CON knee FP 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 4.4; FIGURE 4.5B).

The CON hip FP curve declined steadily from a flexed position to extension from early to mid-stance after which it followed a flexion-extension pattern until toe-off (TABLE 4.4; FIGURE 4.5C). The ACLR hip FP curve followed a similar pattern but was significantly ($p<0.05$) more flexed during early stance (P1 – P2) and the first half of mid-stance (Pt3 - Pt4; TABLE 4.4; FIGURE 4.5C). No significant ($p>0.05$) differences were observed between the ACLR and ACLD hip FP position throughout stance (TABLE 4.4; FIGURE 4.5C).

TABLE 4.3. Mean (±SD) of Ankle, Knee, and Hip Joint Positions † for Control (CON), Anterior Cruciate Ligament Deficient (ACLD), and Anterior Cruciate Ligament Reconstructed (ACLR) Subjects During NP Condition (n=10)

Stance Partition	Ankle			Knee			Hip		
Phase (P)/ Point(Pt)	CON	ACLD	ACLR	CON	ACLD	ACLR	CON	ACLD	ACLR
P1	-4.66±1.33	-5.3±1.91	-6.60±2.66	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	-5.47±1.78	-7.2±2.34	-8.03±4.72	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	-5.45±1.62	-7.86±2.42	-8.43±2.99*	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	-1.08±1.74	-6.26±3.37*	-6.21±2.24*	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	5.99±1.35	-1.67±2.31*	-1.25±2.47*	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	9.88±2.61	2.06±3.00*	4.61±3.89	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	8.71±2.74	2.97±3.41*	5.03±3.24	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	10.48±3.18	3.6±3.25*	6.12±2.91	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	9.18±0.19	1.95±3.35*	4.66±3.76	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	-1.18±3.72	-7.82±2.24*	-7.16±4.44*	21.26±1.92	19.82±4.47	23.95±4.60	10.95±3.84	12.56±3.69	13.68±3.79

† Positive values indicate flexion and dorsiflexion, negative values indicate extension and plantarflexion (°)
* Significantly different than CON ($p<0.05$)

TABLE 4.4. Mean (±SD) of Ankle, Knee, and Hip Joint Positions † for Control (CON), Anterior Cruciate Ligament Deficient (ACLD), and Anterior Cruciate Ligament Reconstructed (ACLR) Subjects During FP Condition (n=10)

Stance Partition	Ankle			Knee			Hip		
Phase (P)/ Point(Pt)	CON	ACLD	ACLR	CON	ACLD	ACLR	CON	ACLD	ACLR
P1	-3.79±3.61	-7.13±3.22*	-6.64±4.68	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	-3.95±3.34	-8.77±3.65*	-7.73±4.81	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	-3.46±1.00	-9.92±4.78*	-8.56±5.12	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	-2.03±1.26	-6.41±3.32*	-5.00±3.34	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	2.17±1.64	-0.30±1.87*	0.20±2.83	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	6.04±1.05	4.13±1.34*	5.24±2.00	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	10.91±1.42	6.5±2.00*	8.33±5.42	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	11.35±2.73	7.81±1.34*	10.29±5.10	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	10.28±2.69	6.81±2.06*	9.69±5.28	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	1.05±1.86	-3.33±2.33*	-1.39±3.39	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 and dorsiflexion, negative values indicate extension and plantarflexion (°)
* Significantly different than CON ($p<0.05$)

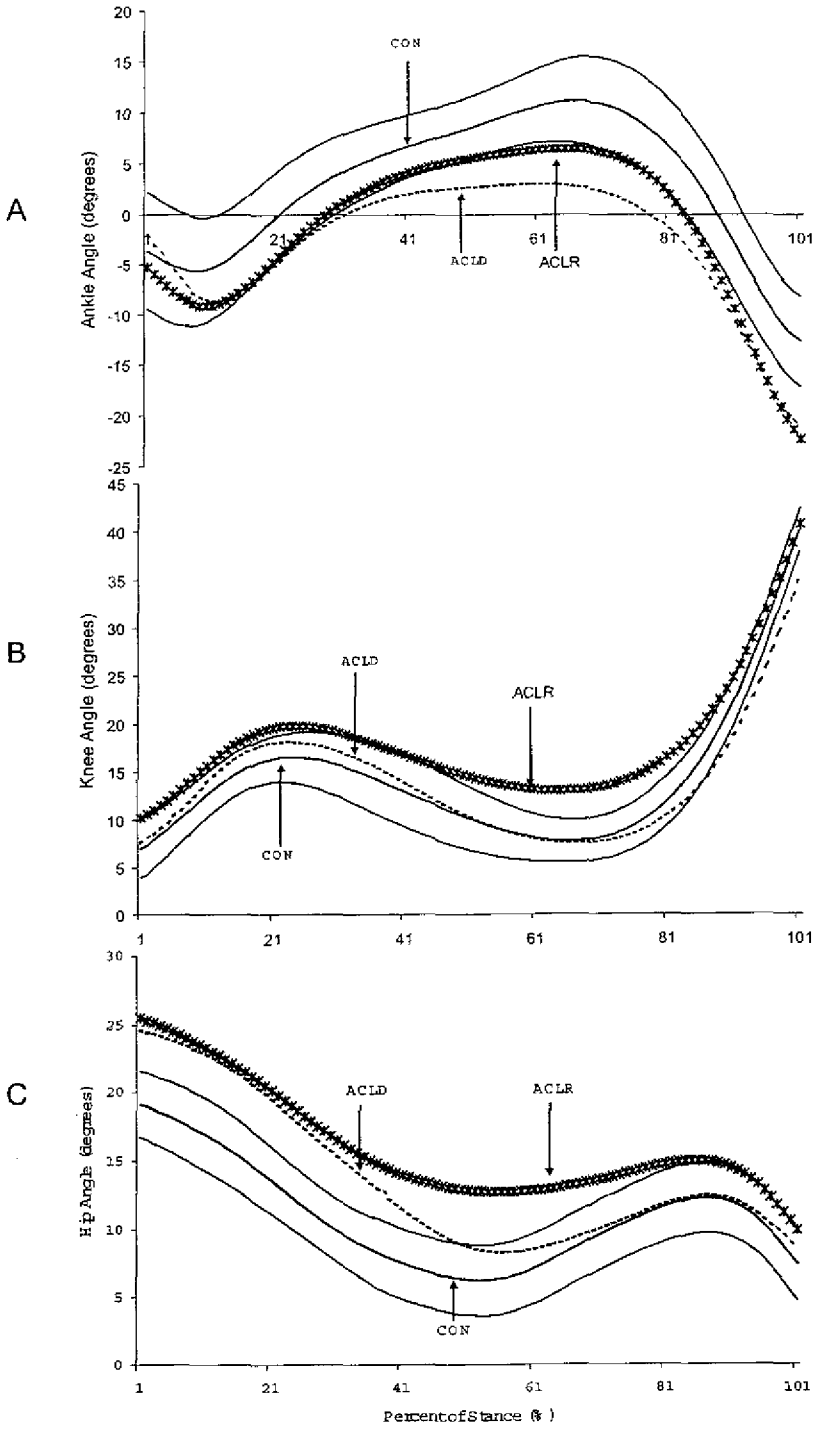

Figure 4.4. Ankle (A), knee (B), and hip (C) joint positions for anterior cruciate ligament deficient (ACLD), anterior cruciate ligament reconstructed (ACLR), and control (CON) subjects during NP condition. Positive and negative values are flexed and extended positions. Solid thick line and thin dotted lines are mean ± 1 SD for CON, dashed thick line is mean of ACLD, and dashed hatches are mean of ACLR group.

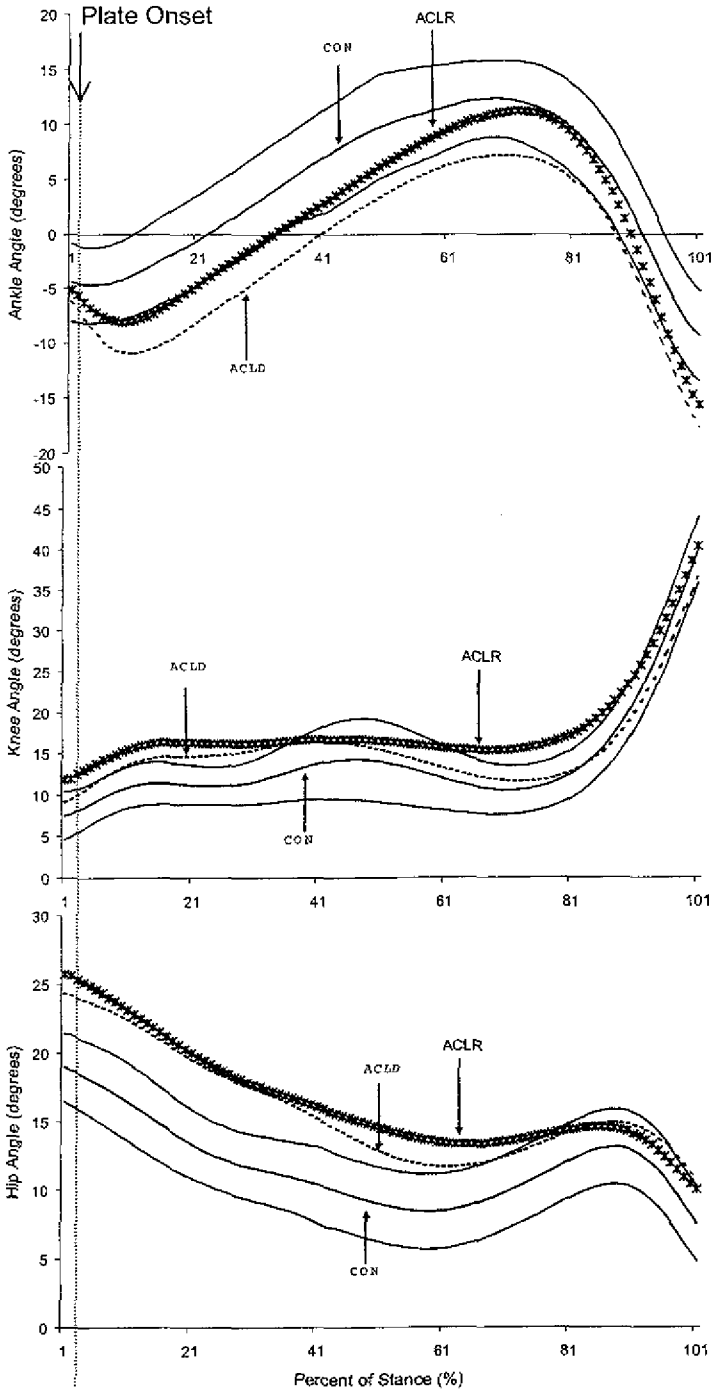

Figure 4.5. Ankle (A), knee (B), and hip (C) joint positions for anterior cruciate ligament deficient (ACLD), anterior cruciate ligament reconstructed (ACLR), and control (CON) subjects during FP condition. Positive and negative values are flexed and extended positions. Solid thick line and thin dotted lines are mean ± 1 SD for CON, dashed thick line is mean of ACLD, and dashed hatches are mean of ACLR group.

Joint Powers

Non-perturbed Gait

TABLE 4.5 presents a comparison of ACLD, ACLR, and CON lower extremity joint powers during the NP condition for P1-5 and Pt1-5 of total stance. The CON ankle NP power curve demonstrated that the ankle absorbed small amounts of power during early stance and the first half of mid-stance after which the ankle produced substantial power until late stance (TABLE 4.5; FIGURE 4.6A). During NP, the ACLR ankle absorbed significantly ($p<0.05$) more power during early stance (P1 – Pt2) then paralleled the CON NP for the remainder of stance (TABLE 4.5; FIGURE 4.6A). No significant ($p>0.05$) differences were observed between the ACLR and ACLD ankle NP power throughout stance (TABLE 4.5; FIGURE 4.6A).

The CON knee NP power was undulating in nature until later in mid-stance when the knee absorbed relatively large amounts of power until toe-off (TABLE 4.5; FIGURE 4.6B). The ACLR knee NP power curve generally paralleled the CON and ACLD NP curve until late stance when the ACLR knee absorbed significantly ($p<0.05$) less power compared to CON (Pt4-P5) and ACLD (Pt5, P5; TABLE 4.5; FIGURE 4.6B).

The CON NP hip produced power during early stance after which power was absorbed for the first part of mid-stance. Small amounts of power generation for the remainder of stance (TABLE 4.5; FIGURE 4.6C). The ACLR hip NP curve was similar to CON during early stance but differed significantly ($p<0.05$) during mid-stance when large amounts of power were generated (Pt3, P3; TABLE 4.5; FIGURE 4.6C). No significant ($p>0.05$) differences were observed between the ACLR and ACLD hip NP power throughout stance (TABLE 4.5; FIGURE 4.6C).

Forward Perturbation Gait

TABLE 4.6 presents a comparison of ACLD, ACLR, and CON lower extremity joint powers during the FP condition for P1-5 and Pt1-5 of total stance. The ACLD, ACLR, and CON ankle FP power curves paralleled one another ($p>0.05$) as small amounts of power were absorbed during early stance and the first half of mid-stance. The ACLR subjects then produced power until late stance (TABLE 4.6; FIGURE 4.7A).

The ACLD, ACLR, and CON knee FP power curves were similar to one another ($p>0.05$) and demonstrated undulating patterns of power production and absorption throughout stance (TABLE 4.6; FIGURE 4.7B).

The CON and ACLD FP hip produced power during early stance, and absorbed power during mid-stance. Small amounts of power were generated during late stance (TABLE 4.6; FIGURE 4.7C). The ACLR hip FP power curve generally paralleled the CON and ACLD NP curve but the ACLR hip FP produced significantly ($p<0.05$) more power during early stance (P1-Pt2; TABLE 4.6; FIGURE 4.7C).

TABLE 4.5. Mean (±SD) of Ankle, Knee, and Hip Joint Powers † for Control (CON), Anterior Cruciate Ligament Deficient (ACLD), and Anterior Cruciate Ligament Reconstructed (ACLR) Subjects During NP Condition (n=10)

Stance Partition	Ankle			Knee			Hip		
Phase (P)/ Point(Pt)	CON	ACLD	ACLR	CON	ACLD	ACLR	CON	ACLD	ACLR
P1	-0.01±0.08	-0.13±0.09*	-0.10±0.04*	-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	-0.02±0.15	-0.19±0.05*	-0.12±0.02*	-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	-0.01±0.02	-0.13±0.05*	-0.08±0.02*	-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	-0.53±0.19	-0.74±0.18	-0.58±0.19	0.06±0.06	0.16±0.10	0.15±0.06	0.46±0.25	0.56±.027	0.48±0.29
Pt3	-0.93±0.19	-0.97±0.32	-0.92±0.36	-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	-0.84±0.12	-0.48±0.35	-0.64±0.32	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	0.94±0.51	-0.28±0.26	-0.47±0.44	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	0.85±0.55	0.84±0.51	0.76±0.28	-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	3.96±1.64	3.24±1.45	3.37±1.39	-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	4.14±0.81	4.06±0.73	4.38±1.03	-0.69±0.14	-0.25±0.35*	-0.03±0.03*#	0.39±0.08	0.16±0.08*	0.32±0.08#

† Positive values indicate power generation, negative values indicate power absorption (W/kg)
* Significantly different than CON ($p<0.05$)
# Significantly different than ACLD ($p<0.05$)

TABLE 4.6. Mean (±SD) of Ankle, Knee, and Hip Joint Powers † for Control (CON), Anterior Cruciate Ligament Deficient (ACLD), and Anterior Cruciate Ligament Reconstructed (ACLR) Subjects During FP Condition (n=10)

Stance Partition	Ankle			Knee			Hip		
Phase (P)/ Point(Pt)	CON	ACLD	ACLR	CON	ACLD	ACLR	CON	ACLD	ACLR
P1	-0.01±0.03	-0.09±0.08	-0.03±0.03	-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	-0.02±0.12	-0.09±0.05	-0.04±0.04	-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	-0.03±0.14	-0.08±0.06	-0.04±0.02	-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	-0.31±0.16	-0.17±0.10	-0.21±0.14	-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	-0.45±0.16	-0.27±0.10	-0.39±0.24	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	-0.38±0.19	-0.39±0.25	-0.34±0.17	-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	-0.39±0.22	-0.27±0.06	-0.27±0.27	-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	1.16±0.81	0.76±0.45	0.69±0.57	-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	2.96±0.69	2.79±0.58	2.89±0.78	-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	3.42±0.43	4.05±0.66	4.32±0.72	-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 ($p<0.05$)

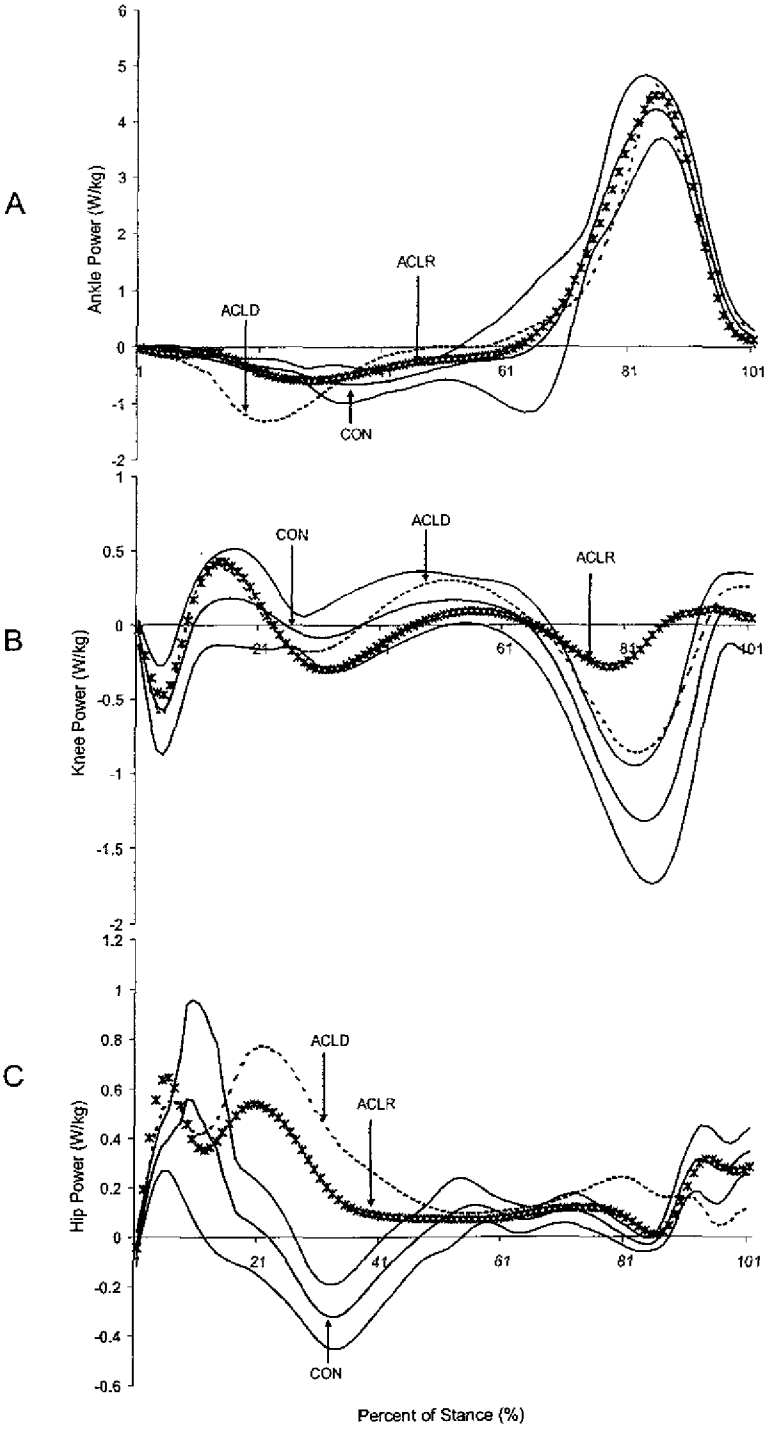

Figure 4.6. Ankle (A), knee (B), and hip (C) joint powers for anterior cruciate ligament deficient (ACLD), anterior cruciate ligament reconstructed (ACLR), and control (CON) subjects during NP condition. Positive and negative values are energy generation and absorption by the muscles. Solid thick line and thin dotted lines are mean ± 1 SD for CON, dashed thick line is mean of ACLD, and dashed hatches are mean of ACLR group.

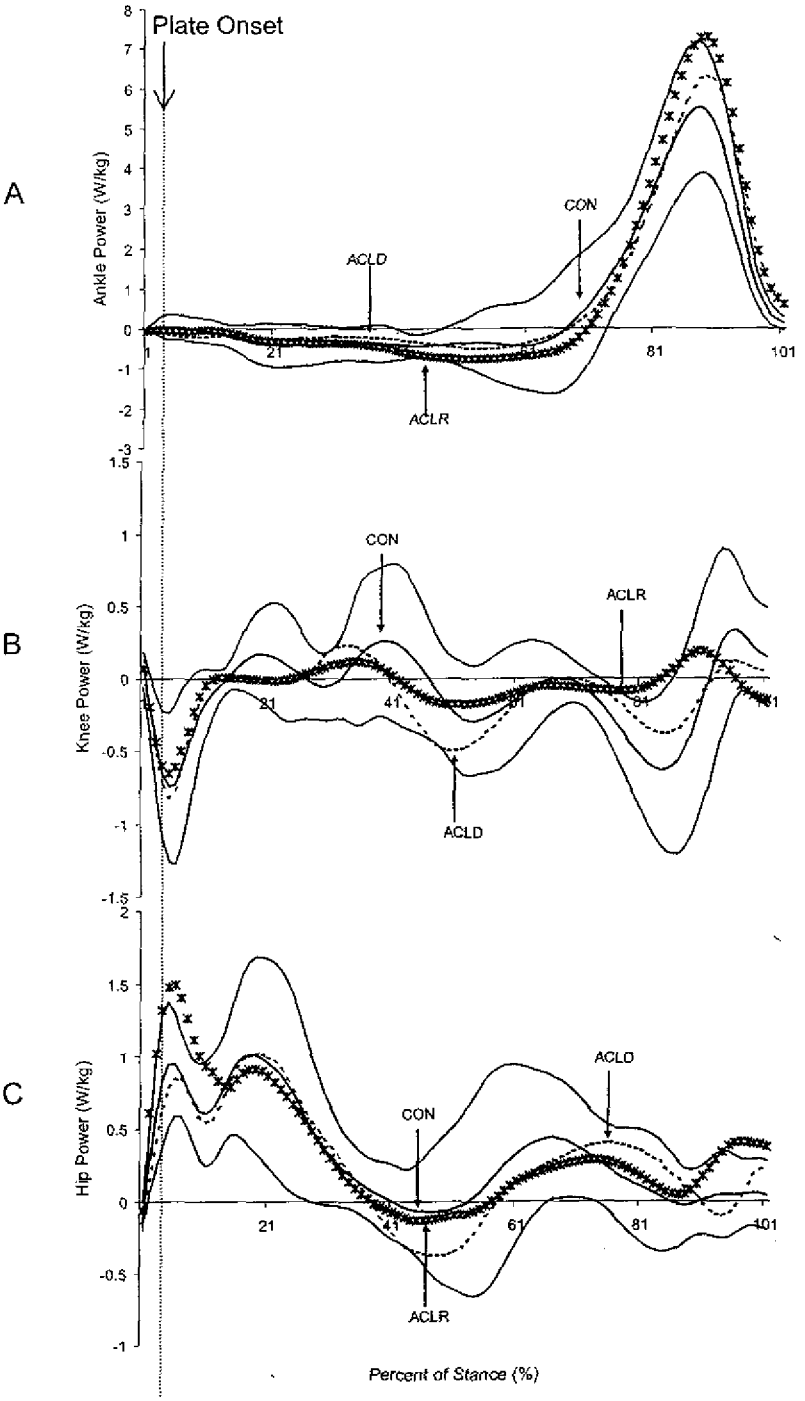

Figure 4.7. Ankle (A), knee (B), and hip (C) joint powers for anterior cruciate ligament deficient (ACLD), anterior cruciate ligament reconstructed (ACLR), and control (CON) subjects during FP condition. Positive and negative values are energy generation and absorption by the muscles. Solid thick line and thin dotted lines are mean ± 1 SD for CON, dashed thick line is mean of ACLD, and dashed hatches are mean of ACLR group.

Muscle EMG

Non-perturbed Gait

TABLE 4.7 presents a comparison of ACLD, ACLR, and CON lower extremity muscle EMG values during the NP condition 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 CON NP-TA muscle response was characterized by strong activation during early stance followed by a rapid decrease to a low levels for all of mid-stance and the first part of late stance with another surge of activity occurring prior to toe-off (TABLE 4.7; FIGURE 4.8A). The ACLR NP-TA EMG activity was similar ($p>0.05$) throughout stance compared to CON and ACLD (TABLE 4.7; FIGURE 4.8A).

The CON NP-GAS muscle response was characterized by a steady rise from heel strike through mid-stance and then a rapid decrease during late stance (TABLE 4.7; FIGURE 4.8B). The ACLR NP-GAS EMG activity was similar ($p>0.05$) throughout stance compared to CON and ACLD (TABLE 4.7; FIGURE 4.8B).

The CON NP-VL muscle produced a large burst of EMG activity during early stance that steadily dropped and remained relatively low throughout the remainder of stance (TABLE 4.7; FIGURE 4.8C). The ACLR NP-VL EMG activity produced significantly ($p<0.05$) less EMG activity during early stance (Pt1, Pt2) compared to CON (TABLE 4.7; FIGURE 4.8C). No significant ($p>0.05$) differences were observed between ACLR and ACLD NP-VL EMG activity throughout stance (TABLE 4.7; FIGURE 4.8C).

The CON NP-BF muscle response produced strong EMG activation during early stance declined steadily for all of mid-stance and the first part of late stance after which another surge of

activity was generated prior to toe-off (TABLE 4.7; FIGURE 4.8D). The ACLR NP-BF EMG activity produced significantly ($p<0.05$) greater EMG activity during early stance (P1 – P2) compared to CON (TABLE 4.7; FIGURE 4.8D). No significant ($p>0.05$) differences were observed between ACLR and ACLD NP-BF EMG activity throughout stance (TABLE 4.7; FIGURE 4.8D).

Forward Perturbation Gait

TABLE 4.8 presents a comparison of ACLD, ACLR, and CON lower extremity muscle EMG values during the FP condition for P1-5 and Pt1-5 of total stance. Values expressed are the FP:NP ratio for the corresponding phase or discrete point of the NP condition for each group.

The CON FP-TA muscle response was characterized by a small burst early in stance followed by a relatively large burst that rapidly subsided until toe-off when another small burst of activity was observed (FIGURE 4.9A). The ACLR FP-TA EMG activity demonstrated significantly ($p<0.05$) less EMG activity during the last half of early stance (P2) compared to CON (TABLE 4.8; FIGURE 4.9A). No significant ($p>0.05$) differences were observed between ACLR and ACLD FP-TA EMG activity throughout stance (TABLE 4.8; FIGURE 4.9A).

The CON FP-GAS muscle response was characterized by a steady rise from heel strike through mid-stance and then a rapid decrease during late stance (FIGURE 3.9B). No significant ($p>0.05$) differences were observed between ACLR and CON FP-GAS EMG activity throughout stance (TABLE 4.8; FIGURE 4.9B). The ACLR FP-GAS activity produced significantly ($p<0.05$) more EMG activity during late stance (Pt5, P5) compared to ACLD (TABLE 4.8; FIGURE 4.9B).

The CON FP-VL muscle response produced a steady rise in EMG activity from heel strike through mid-stance and steadily declined until late stance (FIGURE 4.9C). The ACLR FP-

VL EMG activity demonstrated significantly ($p<0.05$) less EMG activity during mid-stance (P2 – Pt4) compared to CON (TABLE 4.8; FIGURE 4.9C). No significant ($p>0.05$) differences were observed between ACLR and ACLD FP-VL EMG activity throughout stance (TABLE 4.8; FIGURE 4.9C).

The CON FP-BF muscle produced strong activation during early stance and declined steadily throughout the rest of stance (FIGURE 4.9D). The ACLR FP-BF muscle produced significantly ($p<0.05$) greater EMG activity during the last part of mid-stance (P4) and all of late stance (Pt5, P5) compared to CON (TABLE 4.8; FIGURE 4.9D). No significant ($p>0.05$) differences were observed between ACLR and ACLD FP-BF EMG activity throughout stance (TABLE 4.8; FIGURE 4.9D).

TABLE 4.7. Mean (±SD) Muscle EMG Activity † for Control (CON), Anterior Cruciate Ligament Deficient (ACLD), and Anterior Cruciate Ligament Reconstructed (ACLR) Subjects during NP Condition (n=10)

Stance Partition	TA			GAS			BF			VL		
Phase (P)/ Point(Pt)	CON	ACLD	ACLR	CON	ACLD	ACLR	CON	ACLD	ACLR	CON	ACLD	ACLR
P1	0.87±0.05	0.89±0.05	0.81±0.19	0.29±0.04	0.32±0.09	0.26±0.13	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	0.87±0.04	0.96±0.03*	0.85±0.22	0.26±0.06	0.34±0.11	0.27±0.14	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	0.90±0.12	0.95±0.06	0.85±0.23	0.26±0.07	0.33±0.11	0.26±0.15	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	0.41±0.06	0.53±0.06*	0.42±0.13	0.28±0.06	0.41±0.13	0.34±0.18	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	0.31±0.16	0.40±0.14	0.23±0.06	0.34±0.07	0.56±0.05*	0.50±0.17	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	0.31±0.11	0.39±0.13	0.29±0.11	0.41±0.12	0.68±0.13*	0.52±0.15	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	0.19±0.07	0.32±0.10*	0.34±0.25	0.49±0.05	0.74±0.18*	0.52±0.21	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	0.17±0.95	0.25±0.04*	0.22±0.06	0.79±0.06	0.85±0.07	0.73±0.16	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	0.13±0.02	0.23±0.08*	0.22±0.10	0.55±0.08	0.58±0.13	0.49±0.22	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	0.18±0.04	0.25±0.07	0.19±0.10	0.24±0.09	0.25±0.10	0.20±0.08	0.33±0.07	0.27±0.10	0.32±0.10	0.30±0.13	0.34±0.04	0.31±0.15

† Muscle EMG activity normalized to maximum amplitude within NP condition.

* Significantly different than CON ($p<0.05$)

TABLE 4.8. Mean (±SD) Muscle EMG Activity † for Control (CON), Anterior Cruciate Ligament Deficient (ACLD), and Anterior Cruciate Ligament Reconstructed (ACLR) Subjects during FP Condition (n=10)

Stance Partition	TA			GAS			BF			VL		
Phase (P)/ Point(Pt)	CON	ACLD	ACLR	CON	ACLD	ACLR	CON	ACLD	ACLR	CON	ACLD	ACLR
P1	0.87±0.09	1.04±0.08	0.94±0.06	1.15±0.09	1.18±0.05	1.14±0.14	1.24±0.12	1.11±0.10	1.15±0.14	0.75±0.18	0.90±0.11	0.87±0.16
Pt1	0.96±0.10	1.06±0.08	0.92±0.05	1.14±0.08	1.16±0.06	1.11±0.18	1.26±0.08	1.14±0.07	1.16±0.10	0.78±0.17	0.92±0.07	0.91±0.07
Pt2	0.70±0.05	0.66±0.06	0.74±0.07	1.01±0.04	1.05±0.04	1.09±0.07	1.17±0.06	0.98±0.10	1.04±0.13	0.76±0.12	0.95±0.05	0.88±0.12
P2	1.71±0.03	1.54±0.06*	1.43±0.27*	0.92±0.03	0.95±0.06	0.89±0.14	1.51±0.21	1.44±0.16	1.31±0.12	1.23±0.07	1.12±0.05	0.97±0.07*
Pt3	1.63±0.15	1.48±0.13*	1.58±0.09	0.12±0.22	0.44±0.10	0.32±0.22	1.39±0.05	1.24±0.19	1.21±0.18	1.55±0.14	1.27±0.04*	1.14±0.16*
P3	1.17±0.03	1.13±0.06	1.25±0.17	0.80±0.03	0.86±0.05	0.86±0.07	1.41±0.13	1.30±0.21	1.32±0.17	1.51±0.14	1.26±0.04*	1.16±0.13*
Pt4	0.97±0.03	0.96±0.04	1.08±0.09	1.10±0.12	1.12±0.03	1.11±0.14	1.28±0.07	1.19±0.10	1.21±0.29	1.34±0.14	1.20±0.09*	0.97±0.16*
P4	1.06±0.10	0.96±0.08	1.05±0.12	0.87±0.03	1.01±0.02*	0.87±0.13	0.66±0.05	1.08±0.03*	1.12±0.29*	1.23±0.07	0.99±0.12*	1.03±0.17
Pt5	0.99±0.09	0.92±0.04	1.08±0.12	0.68±0.08	0.97±0.07*	0.71±0.24#	0.74±0.08	0.94±0.08*	1.04±0.08*	1.12±0.13	0.80±0.09*	0.89±0.15
P5	0.98±0.03	0.99±0.02	1.17±0.18	0.85±0.06	1.13±0.04*	0.87±0.12#	0.22±0.09	1.15±0.01*	1.07±0.12*	1.04±0.01	1.01±0.09	0.91±0.18

† Values greater than 1.0 indicate FP EMG activity greater than NP condition, values less than 1.0 indicate FP EMG activity less than NP condition.

* Significantly different than CON ($p<0.05$)

Significantly different than ACLD ($p<0.05$)

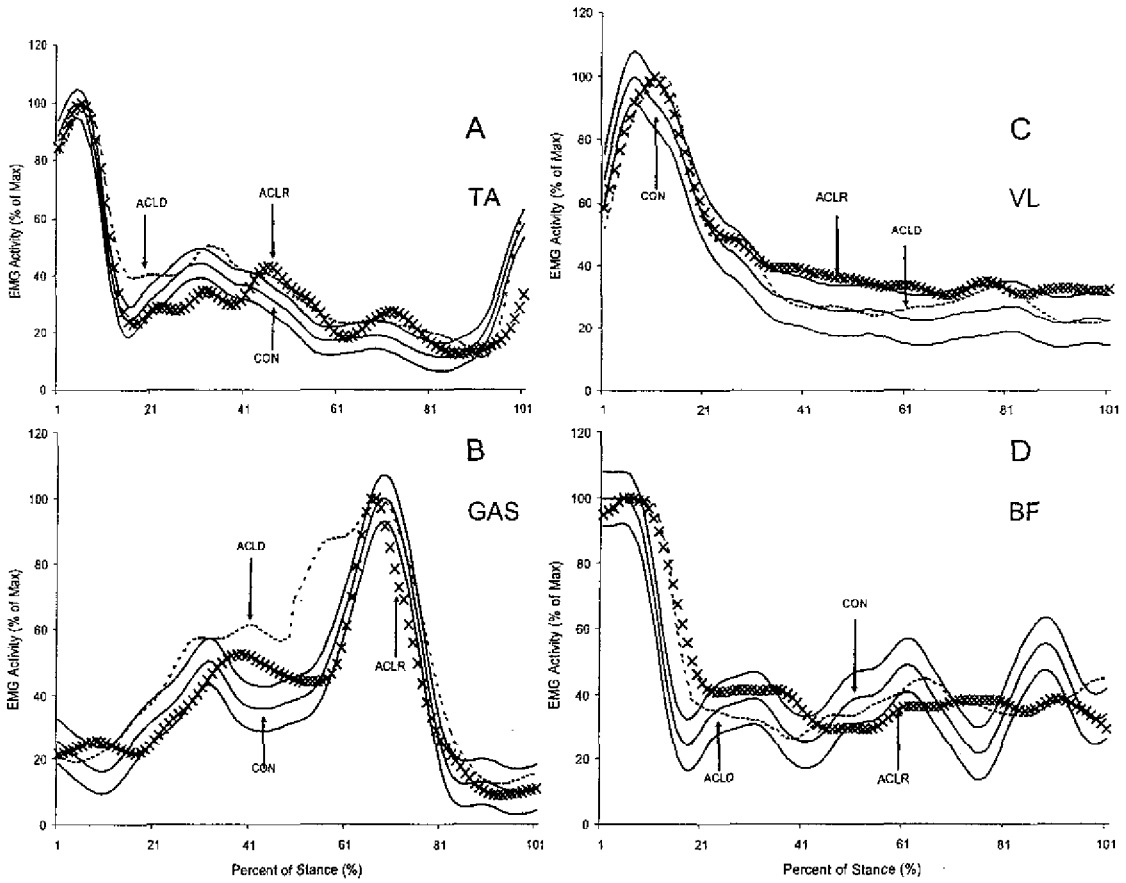

Figure 4.8. Muscle EMG activity of anterior cruciate ligament deficient (ACLD: dashed thick line), anterior cruciate ligament reconstructed (ACLR: dashed hatches), and control (CON: solid thick line and thin dotted lines are mean ± 1 SD) subjects during NP condition for the tibialis anterior (TA: A), gastrocnemius (GAS: B), vastus lateralis (VL: C), and biceps femoris (BF: D).

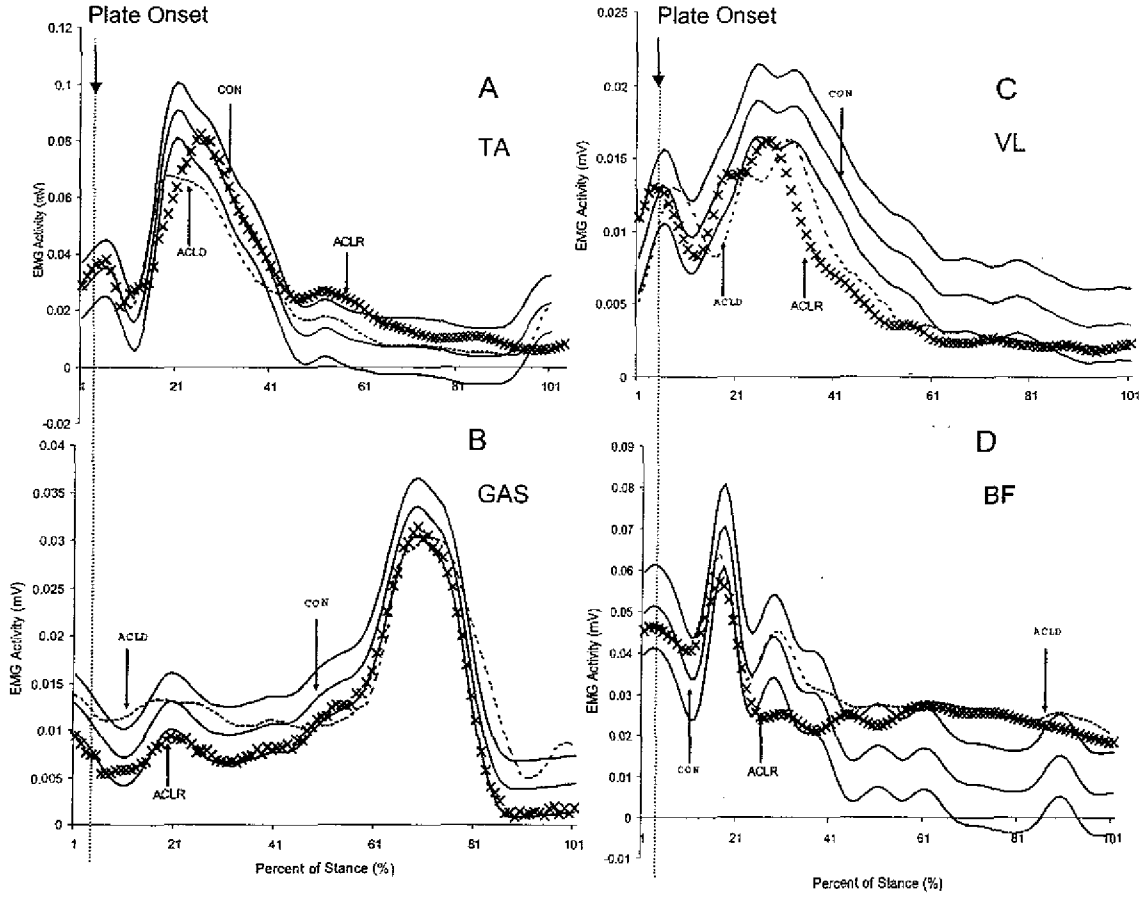

Figure 4.9. Representative example of muscle EMG activity of anterior cruciate ligament deficient (ACLD: dashed thick line), anterior cruciate ligament reconstructed (ACLR: dashed hatches), and control (CON: solid thick line and thin dotted lines are mean ± 1 SD) subjects during FP condition for the tibialis anterior (TA: A), gastrocnemius (GAS: B), vastus lateralis (VL: C), and biceps femoris (BF: D).

Hypotheses Results

It was hypothesized that 3 months following reconstructive surgery the ACL repaired limb would demonstrate a sustained knee extensor moment and greater hip extensor moment during perturbed and unperturbed walking. These hypotheses were partially confirmed as the ACLR group exhibited a significantly greater knee (FIGURE 4.2B) and hip extensor moment (FIGURE 4.2C) during the first half of NP stance compared to CON. During the latter half of NP stance the ACLR group exhibited a significantly reduced knee flexor moment (FIGURE 4.2B) compared to CON and pre-surgical ACLD values. In response to the FP, the ACLR group exhibited a sustained knee extensor moment (FIGURE 4.3B) as compared to the CON group. It was also hypothesized that the ACLR group would exhibit greater hip and knee flexion positions and increased knee extensor muscle EMG activity compared to CON during NP and FP conditions. These hypotheses were confirmed as the ACLR were significantly more flexed at the knee (FIGURE 4.4B &4.5B) and hip (FIGURE 4.4C & 4.4C) and exhibited a prolonged near-maximal BF-EMG (FIGURE 4.8D & 4.9D) during the NP and FP conditions 3 months following surgery as compared to the CON group.

Discussion

The purpose of this study was to determine how gait patterns during non-perturbed (NP) conditions may change as a result of ACL surgical repair and to determine the effect of unexpected forward perturbations (FP) on lower extremity joint moments in ACLR subjects compared to their pre-surgical ACLD values and healthy controls. To date, investigations involving ACLR subjects are limited and suggest that time since surgery may play an important role in the re-establishment of pre-injury gait patterns (Bulgheroni et al., 1997; Bush-Joseph et al., 2001; Cicotti et al., 1994; Devita et al., 1998; Ernst et al., 2000; Timoney et al., 1993). However, few comprehensive gait studies have investigated ACL injured subjects prior to and following surgical repair (Devita et al., 1997) and no studies have been conducted to examine the effects of unexpected gait perturbations on ACLD and ACLR individuals. During an unexpected gait perturbation, the ability of an ACLR individual to react and maintain equilibrium is critical in the prevention of re-injury. It is hoped that information from this investigation will allow for a better understanding of the neurological and mechanical gait accommodations resulting from ACL surgical repair.

Non-Perturbed Gait

Although the moments of support (Ms) were similar among the ACLR, pre-surgical ACLD, and CON groups during NP (FIGURE 4.1), the individual joint moments that produced the Ms differed significantly following surgery (FIGURE 4.2). The ACLR group produced a significantly greater knee extensor moment during early NP stance compared to the CON group and a significantly reduced knee flexor moment for the remainder of stance compared to the CON

and ACLD groups (FIGURE 4.2B). This pattern has not been previously reported in any ACLR gait investigations. No study to date involving ACLR subjects has provided data between 5 weeks and 6 months following surgery. Furthermore, only one gait investigation has reported pre and post-surgical ACL results on the same subjects and only data up to 5-week post-surgery were provided (Devita et al., 1997). Devita et al. (1997) reported that ACLR subjects exhibited a sustained knee extensor moment throughout stance both pre- and post-surgically, although the 5 week ACLR knee moment 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 this influenced post-operative gait patterns. In another study, Devita et al. (1998) investigated ACLR subjects who were undergoing an accelerated rehabilitation protocol 3 weeks and 6 months post-operatively, but provided no data regarding time between injury and surgery nor was there a comparison to pre-surgical data from the same subjects. The results indicated that the ACLR group demonstrated a sustained knee extensor moment throughout stance at 3 weeks post-operatively, a result similar to 3 week ACLR data previously reported (Devita et al., 1997). At 6 months post-surgery, the ACLR subjects exhibited a biphasic knee extensor-flexor-extensor moment pattern similar to healthy subjects but significantly reduced in positive extensor angular impulse. This suggests that after approximately 6 months, ACLR subjects can regain close to normal joint moment patterns but that more time may be needed to re-establish normal gait characteristics. In the present investigation, ACLR subjects demonstrated a biphasic knee moment pattern although the pattern differed significantly in magnitude compared to CON and pre-surgical ACLD values (FIGURE 4.2B). These data are supported by those of Devita et al. (1997, 1998) 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. (1993) reported data on ACLR subjects between 9 and 12 months after surgery with a range of 1 to 66 months between injury and surgery. They found that ACLR subjects walked with a 64% reduction in knee extensor moment at midstance compared with healthy controls. The knee extensor results reported by Timoney et al. (1993) were even lower than those reported in this investigation at 3 months and those reported by Devita (1997, 1998) at 3 weeks post-surgery. It is possible that factors such as patient compliance and different rehabilitation protocols could account for the discrepancies between the results from this investigation, Timoney (1993), and Devita (1998) as could the significantly different times between injury and surgery. Since subjects involved in this investigation were all more than 2 years post-injury, this was the first study to examine how chronic ACLD subjects respond to ACL reconstructive surgery.

In Chapter III it was postulated that ACLD subjects had adapted to ACL injury prior to undergoing reconstructive surgery since knee moment values were similar to the CON group (Ferber, 2001). However, 3 months following surgery, knee moment patterns observed in the present study were significantly different compared to pre-surgical ACLD values, suggesting that surgery had a significant influence on the knee moment characteristics.

During early NP stance, the ACLR group exhibited significantly reduced VL-EMG activity compared to CON (FIGURE 4.8C). However, examination of FIGURE 4.8C reveals that the ACLR VL muscle activity was increasing from heel strike to early mid-stance. Since NP-EMG data were normalized to the maximum within-trial amplitude, EMG amplitude relative to the CON and ACLD group is not available. Therefore, the reduced VL activity can be interpreted as a reduced rise (slope) in VL-EMG activity toward maximum activation. The reduced slope could result from maximum VL activation occurring slightly later in NP stance in the ACLR group as compared to CON (FIGURE 4.8C). It is possible that the later maximum activation and

subsequent reduced rise in activation was a strategy to help reduce anterior tibial strain and stress on the new ACL graft in the ACLR group.

The ACLR group exhibited significantly greater knee and hip flexion positions during NP stance compared to CON and pre-surgical ACLD values (FIGURE 4.4B & C). These data are similar to previous investigations involving ACLR subjects who were less than 1 year post-surgery (Bush-Joseph et al., 2001; Devita et al., 1998; Timoney et al., 1993). Devita et al. (1997) hypothesized that a more flexed (crouched) position demands a greater knee extensor moment to prevent collapse of the body during the stance phase of gait. The significantly greater knee extensor moment observed in the ACLR group during early NP stance, and the significantly reduced knee flexor moment for the remainder of stance, could be the result of greater knee extensor moment throughout stance to prevent collapse. However, a greater knee extensor moment may produce excessive anterior tibial shear during stance and place undue stress on the reconstructed ligament. The ACLR group may have counteracted this effect by producing a significantly greater concentric hip extensor moment (FIGURE 4.2C & 4.6C) and prolonged near-maximal BF-EMG activity during early NP stance as compared to CON (FIGURE 4.8C). The hamstring muscles, as a component of the hip extensor moment, are effective synergists to the ACL in reducing anterior tibial shear (Pandy & Shelburne, 1997; Osternig et al., 2000). It is possible that the increase in hip extensor moment and prolonged near-maximal BF-EMG activity was necessary to stabilize the knee, in response to the increased knee extensor moment, and help prevent excessive anterior tibial translation during early stance.

In Chapter III, it was reported that the CON subjects demonstrated a hip flexor moment while pre-surgical ACLD subjects exhibited a prolonged hip extensor moment during mid-stance of NP (Ferber, 2001). It was hypothesized that CON subjects were able to produce a hip flexor moment following midstance since the intact ACL was able to restrain the tibia posteriorly and it

was therefore not necessary to generate hamstring muscle activation in the form of a hip extensor moment. In the absence of an ACL, the prolonged hip extensor moment produced by ACLD subjects may have been necessary to reduce anterior tibial shear (Ferber, 2001). In the present investigation, it is possible that the new ACL graft was now able to restrain the tibia posteriorly allowing a hip flexor moment at mid-stance similar to that in the CON group (FIGURE 4.2C). The ACLR group did, however, exhibit a significantly reduced hip flexor moment during the latter half of mid-stance as compared to the CON group (FIGURE 4.2C), suggesting that the hip had not yet re-established normal joint moment characteristics at 3 months post-surgery.

During NP stance, the ACLR subjects were approximately 3° more flexed at the knee and hip as compared to pre-surgical values, and approximately 5° more flexed than CON (FIGURE 4.4). Devita et al. (1998) reported that ACLR subjects walked with approximately 10° more flexion at the three major lower extremity joints 3 weeks after surgery but no differences were observed 6 months later. In another study, Bush-Joseph et al. (2001) reported no significant differences in knee flexion angle at mid-stance 8 months after surgery. The results from this investigation are supported by these findings and suggest that ACLR subjects have not yet fully recovered 3 months after surgery. However, based on the results of this investigation and those of Devita et al. (1998) and Bush-Joseph et al. (2001), it is reasonable to postulate that, over time, ACLR subjects gradually regain a more erect posture during gait.

Response to Unexpected Forward Perturbations During Gait

In Chapter II it was reported that, during early FP stance, the knee produces a flexor moment and contributes little, if any, to the maintenance of a positive Ms during early stance in healthy uninjured subjects (Ferber, 2001). This appears to be compensated for by a hip extensor

moment and to a lesser extent by an ankle plantarflexor moment. However, near mid-stance of FP, the hip produced a flexor moment which, in turn, is compensated for by an extensor moment at the knee and an increase in the plantarflexor moment at the ankle (Ferber, 2001). In the present investigation, no significant differences in ankle moment patterns were observed during the FP condition between the ACLR, CON and ACLD groups. However, the ACLR group demonstrated a net zero knee moment during early stance and a significantly greater and sustained knee extensor moment for the remainder of FP stance as compared to the CON and pre-surgical ACLD groups (FIGURE 4.3B). Coincident with the net zero knee moment, the ACLR subjects exhibited significantly less VL-EMG activity and no differences in BF-EMG activity as compared to CON (FIGURE 4.9). It is important to note that FP-EMG data were normalized to NP-EMG activity and expressed as the FP:NP ratio. Therefore, in response to the unexpected FP, ACLR subjects exhibited similar VL-EMG activity and an increase in BF-EMG activity as compared to NP gait. It is possible that the FP had little effect on the VL-EMG activity since the ACLR subjects rely more on the quadriceps muscles to maintain support during NP stance and therefore demonstrated little change in VL-EMG activity in response to an unexpected FP. The net zero knee moment observed during early FP stance was most likely the result of the co-activating hamstring muscle in response to the unexpected FP. Thus, the quadriceps and hamstrings produced near equal torque at the knee joint and possibly increased knee joint stability during the FP. During early FP stance, the ACLR subjects also demonstrated no power production (FIGURE 4.7B) and a negligible change in knee angle (FIGURE 4.5B). These data also support the premise that opposing knee muscles were co-activated possibly to maintain knee stability and protect the new ACL graft during early FP stance.

During FP mid-stance, the ACLR group demonstrated a significantly greater and sustained knee extensor moment as compared to CON (FIGURE 4.3B). The ACLR group also

exhibited significantly less VL-EMG activity as compared to CON in that the unexpected FP had less effect on VL-EMG activity (FIGURE 4.9C). In the present investigation, it was reported that during NP stance, ACLR subjects demonstrated greater quadriceps reliance during stance to prevent collapse. Prevention of vertical collapse during the FP condition is possibly more important than in NP gait due to the nature and intensity of the unexpected perturbation. The ACLR subjects appear 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.

In Chapter III, no significant differences were reported for hip moments or power production between ACLD and CON subjects during FP stance. However, in the present investigation, the ACLR hip generated significantly more power (FIGURE 4.7C) but no differences in hip moment values were observed (FIGURE 4.3C) during early FP stance as compared to CON and pre-surgical ACLD values. As well, the ACLR subjects exhibited significantly more hip flexion during early and mid-stance as compared to CON (FIGURE 4.5C). 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 FIGURE 4.5C. In Chapter II it was 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 (Ferber, 2001). Since the ACLR subjects were already in a more flexed hip position, it is possible that a greater angular velocity of hip flexion and concomitant power generation was necessary to control the upper body in response to the forward movement of the force plate.

Summary

Data from Devita et al. (1997) show that the knee moment and kinematic patterns of acute ACLD subjects differ significantly from controls. The results from the present study suggest that the chronic ACLD group may have adapted to the ACL injury over time since they exhibited knee joint moment and kinematic patterns similar to control subjects. This conjecture is supported by the finding that 3 months following surgery these same subjects demonstrated a significantly different knee moment and were significantly more flexed at the knee and hip during the NP and FP conditions as compared to pre-surgical ACLD and CON values. The ACLR group also exhibited a hip moment pattern more characteristic of the CON group but significantly different from pre-surgical ACLD 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 and that the re-establishment of pre-injury gait patterns takes longer than 3 months to occur.

Bridge

The first three studies characterized the kinetic, kinematic, and muscle activation responses to an unexpected FP in ACL injured subjects 3 months following ACL reconstructive surgery as compared to pre-surgical ACLD values and non-injured young adults. However, no information has been provided on the contralateral uninjured limb of the subjects involved in this investigation. Studies involving bilateral gait adaptations to ACL injury and subsequent surgical repair are limited. Injury to one limb may result in contralateral limb accommodations in an

effort to maintain symmetry (Berchuck et al., 1990; Kowalk et al., 1997; Sadeghi et al., 2000). Therefore, the purpose of the fourth study was to investigate contralateral uninjured joint accommodations as a result of chronic ACLD and ACL surgical repair during NP and in response to an unexpected FP. Chapter V summarizes the similarities and differences in NP and FP gait patterns between the injured and non-injured limb of ACL injured subjects prior to and 3 months following surgical repair, as well as left and right limb comparisons in the CON group.

CHAPTER V

BILATERAL ACCOMMODATIONS TO ANTERIOR CRUCIATE LIGAMENT DEFICIENCY AND SURGERY

Introduction

Walking is a fundamental requirement for daily activity yet it is one of the most complex of all human activities. Successful locomotion requires a complex interaction between the central nervous system (CNS) and various muscles to maintain balance, support of the body against gravity, and propel the body forward in a smooth and rhythmical manner (Sadeghi et al., 2000; Winter, 1990a). Smooth and rhythmical gait is often associated with gait symmetry and gait asymmetry is commonly associated with gait pathology. While gait symmetry has been supported in the literature (Hamill et al., 1984; Menard et al., 1992), common consensus is that gait is asymmetrical even in healthy populations (Allard et al., 1996; Dickey & Winter, 1992; Crowe et al., 1993; Ounpuu & Winter, 1979; Sadeghi et al., 1997). Allard et al. (1996) reported total lower extremity joint symmetry for positive work during gait but significantly greater peak muscle power generation in the right limb of subjects. Allard et al. (1996) speculated that the between-limb differences in peak power should be interpreted as gait adjustments rather than

asymmetry and could be attributed to different limb functions during gait. Sadeghi et al. (1997) investigated bilateral muscle power patterns during gait in healthy subjects. Significant bilateral differences in power production were observed as a possible result of functional differences during the stance phase of gait. It was suggested that the lead leg was primarily used for propulsion while the trail leg was used for balance control. These data suggested that gait asymmetry in healthy subjects should not be considered pathological and challenged the use of unilateral limb evaluations or pooling of right and left limb data in gait evaluations. The bilateral assessment of gait mechanics in healthy populations may be most important especially when used as a comparison with injured populations.

Several studies have investigated possible joint accommodations to anterior cruciate ligament (ACL) injury using comparisons to the contralateral uninjured limb and to healthy controls (Berchuck et al., 1990; Czerniecki et al., 1988; Kowalk et al., 1997; Roberts et al., 1999; Rudolph et al., 1998; Tibone et al., 1986). Tibone et al. (1986) evaluated 18 ACL injured patients during free and fast walking, running, cutting, and stair climbing activities using the contralateral uninjured limb for comparison. No significant differences in joint angles were observed between limbs for any activity. Ground reaction force (GRF) data demonstrated symmetry during walking but a reduced vertical GRF during running and increased vertical GRF during fast walking for the injured limb. Rudolph et al. (1998) assessed chronic ACL deficient (ACLD) subjects during walking also using the contralateral uninjured limb for comparison. During walking, the involved limb exhibited lower peak vertical GRF, a greater knee flexion angle, a reduced knee extensor moment, and reduced knee power absorption compared to the contralateral uninjured limb. While data from these two investigations provide valuable information and suggest that ACL injury may lead to joint accommodation during gait, no comparisons were made to healthy uninjured populations.

Czerniecki et al. (1988) evaluated tibiofemoral rotation in ACL injured knees compared to the contralateral uninjured limb and healthy controls during treadmill walking and running using a triaxial electrogoniometer. Results revealed significant increases in bilateral tibiofemoral rotation with increasing gait speed but no significant differences in rotation between limbs or between groups. Kowalk et al. (1997) studied ACL injured subjects during stair ascent prior to and 6 months following reconstructive surgery. Data were compared between the ACL injured and uninjured limbs and to pooled limb data of healthy control subjects. No significant differences in any biomechanical parameter were observed between limbs or between groups pre-operatively. Post-operatively, however, the injured limb demonstrated significant reductions in peak knee extensor moment, peak power production, and work performed as compared to the uninjured limb and control values. Berchuck et al. (1990) examined ACLD subjects during walking and jogging using the contralateral uninjured limb and pooled data from healthy subjects for comparison. This study reported that the injured ACLD knee exhibited a sustained knee flexor moment during midstance compared to the uninjured limb and controls. This type of gait pattern was interpreted as a tendency to avoid or reduce the demand placed on the quadriceps and was termed a "quadriceps avoidance gait" possibly serving to reduce anterior tibial shear during gait. Berchuck et al. (1990) also demonstrated a bilateral increase in the hip extensor moment during normal gait and a bilateral reduction in the maximum knee extensor moment during jogging in ACLD patients as compared to controls.

While these investigations provide insight regarding possible joint accommodations to ACL injury and surgical repair, none of these studies considered gait asymmetry in the healthy populations. Therefore, the purpose of this investigation was to determine the effect of ACL injury and subsequent surgery on bilateral lower extremity joint kinematic, moment, and power

patterns in chronic ACLD subjects prior to and 3-months following surgical repair and in healthy subjects.

It was hypothesized that the ACLD and ACLR subjects would be more symmetrical in gait mechanics compared to the gait patterns of the uninjured control subjects during NP gait and in response to the FP.

Method

Subjects

Ten (5 males and 5 females) ACL deficient (ACLD) individuals aged 18-40 years were recruited as subjects for this study. All 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 (range 2.2yr – 16.1yr; mean 5.7 yr). All ACLD 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. All ACLD subjects were right limb dominant and all but 1 subject had injured their left limb. Demographic information regarding these subjects has been described in Chapter III.

Ten (5 males and 5 females) healthy uninjured young adults also participated in the study as control subjects (CON). All CON subjects were right limb dominant. Demographic information regarding these subjects has been described in Chapter II. Prior to participation, each subject signed a consent form (APPENDIX A) approved by the Human Subjects Compliance Committee at the University of Oregon.

Experimental Apparatus and Protocol

The experimental apparatus and protocol was the same as that described in Chapter II. The ACLD subjects completed the identical 48-walking trial test prior to, and 3 months

following, ACL surgical repair. Following the 48-walking trials, each subject performed the same protocol using the opposite limb.

Data Analysis

To obtain joint kinematic, moment, and power measures of interest for the trials, data analysis involved the following steps:

1. Partitioning of the stance phase of the gait cycle as defined from heel strike to toe off.
2. Interpolating joint (ankle, knee, hip) moment, power, and position as percent stance phase for each condition.
3. Averaging the 12 trials for each condition into an ensemble average.

Statistical Analysis

The purpose of this investigation was to determine the effect of ACL injury and subsequent surgery on bilateral lower extremity joint symmetry in chronic ACLD subjects prior to and 3-months following surgery and healthy controls. The overall goal was to determine global differences in lower extremity mechanics during the stance phase of gait and not to determine where in the stance phase differences may occur. Therefore, a point-by-point Pearson product-moment correlation coefficient over the entire stance period of gait was calculated between corresponding data points of bilateral lower extremity joint kinematic, moment, and power curves for each group during the non-perturbed (NP) and forward perturbation (FP) conditions. Derrick et al. (1994) first introduced this curve correlation technique and reported that temporal similarities are always indicative of a high correlation but are not sensitive to

amplitude differences. As such, the extensor angular impulse (EAI), positive work, and average joint angle were also analyzed using a three-way (group x limb x condition) analysis of variance (ANOVA). The independent variables were 1) the three groups (ACLD, ACLR, and CON), 2) limb (injured, non-injured, left, right), and 3) the two conditions (NP and FP). The dependent variables were joint (ankle, knee, hip) 1) moment, 2) power, and 3) position. A priori post-hoc analyses were then performed to detect differences between contralateral limbs and between groups. A maximum α level of 0.05 was used to indicate statistical significance.

Results

This study was conducted to determine the effect of ACL injury and subsequent surgical repair on bilateral lower extremity joint kinematic, moment, and power patterns in chronic ACLD prior to and 3 months following surgical repair, and in healthy subjects. In this section, descriptive measures of stance are presented first, followed by lower extremity joint moments, joint powers, and joint kinematics for non-perturbed (NP) and forward perturbation (FP) conditions.

Descriptive Measures of Stance

Time

Total time of stance for the NP and FP conditions were similar ($p>0.05$) between limbs and between the CON, ACLD, and ACLR groups (TABLE 5.1).

TABLE 5.1 Time of Stance for left (l) and right (r) limbs of Control (CON) and injured (i) and non-injured (n) limbs for Anterior Cruciate Ligament Deficient (ACLD) and Anterior Cruciate Ligament Reconstructed (ACLR) Groups for NP and FP Conditions (n=10)

Group/Limb	Time of NP Stance (ms)	Time of FP Stance (ms)
CONl	856.93 ± 73.23	1000.95 ± 75.94
CONr	863.06 ± 77.27	977.14 ± 58.33
ACLDi	865.08 ± 52.22	962.33 ± 77.00
ACLDn	838.97 ± 62.47	912.15 ± 73.33
ACLRi	853.22 ± 72.33	907.20 ± 72.24
ACLRn	858.00 ± 77.39	912.57 ± 58.54

Non-Perturbation Condition

TABLE 5.2 presents a comparison of CON, ACLD, and ACLR bilateral lower extremity Pearson product-moment correlation coefficient r-values and bilateral joint extensor angular impulse (EAI), positive work, and average angle (Ang) during NP stance.

Joint Moments

The shapes of the bilateral joint NP moment curves for each group were moderately to highly correlated with one another (range 0.72 – 0.99: TABLE 5.2; FIGURES 5.1-5.3). The CON, ACLD, and ACLR ankle NP moment curves were all highly correlated to the corresponding contralateral limb (range 0.98-0.99) and no significant ($p>0.05$) differences in ankle NP EAI were observed between limbs or between the CON, ACLD, or ACLR groups (TABLE 5.2; FIGURE 5.1).

The bilateral knee NP moment curves were highly correlated (range 0.94-0.96) for each of the 3 groups but magnitude differences in EAI were observed (TABLE 5.2; FIGURE 5.2). The ACLD non-injured (n) and ACLRn knee produced significantly ($p<0.05$) more NP EAI compared to the ACLD injured (i) knee and compared to bilateral CON knees (TABLE 5.2; FIGURE 5.2). The ACLRi knee produced significantly ($p<0.05$) more NP EAI compared to the CON right (r) knee (TABLE 5.2; FIGURE 5.2).

The bilateral CON hip NP moment curves were moderately correlated (0.72) while the contralateral ACLD (0.89) and ACLR (0.94) hip moment curves were highly correlated (TABLE 5.2; FIGURE 5.3). No significant ($p>0.05$) differences hip NP EAI were observed between limbs or between the 3 groups (TABLE 5.2; FIGURE 5.3).

Joint Powers

The shapes of the bilateral joint NP power curves for each group were similar and ranged from poor to highly correlated with one another (range 0.42 – 0.99: TABLE 5.2; FIGURE 5.1-5.3). The CON, ACLD, and ACLR ankle NP power curves were all highly correlated to the corresponding contralateral limb and no significant ($p>0.05$) differences in ankle positive work were observed between limbs or between the CON, ACLD, or ACLR groups (TABLE 5.2; FIGURE 5.4).

The bilateral CON knee NP power curves were highly correlated (0.93) but the contralateral ACLD (0.68) and ACLR (0.79) knee NP curves were only moderately correlated (TABLE 5.2; FIGURE 5.5). The ACLDn and ACLRn knees produced significantly ($p<0.05$) more positive NP work compared to the corresponding injured limb and compared to CON (TABLE 5.2; FIGURE 5.5). The ACLRn knee produced significantly ($p<0.05$) more positive NP work compared to the CONr knee (TABLE 5.2; FIGURE 5.5).

The bilateral CON hip NP power curves were poorly correlated (0.42) and the ACLD (0.70) and ACLR (0.75) hip NP power curves were moderately correlated with one another (TABLE 5.2; FIGURE 5.6). The CONr hip produced significantly ($p<0.05$) more positive NP work compared to CON left (l) hip (TABLE 5.2; FIGURE 5.6). No significant ($p>0.05$) differences in positive NP work were observed between ACLD and ACLR limbs (TABLE 5.2; FIGURE 5.6).

Joint Position

The shapes of the bilateral joint NP position curves for each group were similar and were highly correlated with one another (range 0.86 – 0.99: TABLE 5.2; FIGURE 5.1-5.3). The shapes of the bilateral ankle NP position curves were highly correlated (range 0.97-0.98) for the 3

groups but magnitude differences in average NP angle were observed (TABLE 5.2; FIGURE 5.7). The ACLDi, ACLRi, and ACLRn NP ankles were significantly ($p<0.05$) more plantarflexed compared to contralateral CON ankles (TABLE 5.2; FIGURE 5.7).

The shapes of the bilateral knee NP position curves were highly correlated for the 3 groups (range 0.93-0.99) but magnitude differences in average angle were observed (TABLE 5.2; FIGURE 5.8). The ACLDn and ACLRn NP knees were significantly ($p<0.05$) more flexed compared to the CONl knee (TABLE 5.2; FIGURE 5.8).

The bilateral hip NP position curves were highly correlated (range 0.86-0.96) for the 3 groups but magnitude differences in average angle were observed (TABLE 5.2; FIGURE 5.8). The contralateral ACLD and ACLR NP hips were significantly ($p<0.05$) more flexed compared to contralateral CON hips (TABLE 5.2; FIGURE 5.8).

TABLE 5.2 Pearson Product Correlation Coefficient r-values and Bilateral Means (± 1SD) for Extensor Angular Impulse (EAI†), Positive Work ††, and Average Angle (Ang†††) for the Ankle (A), Knee (K), and Hip (H) of Control (CON), Anterior Cruciate Ligament Deficient (ACLD), and Anterior Cruciate Ligament Reconstructed (ACLR) Subjects during NP Condition (n=10)

	CON			ACLD			ACLR		
Variable	r-value	Left	Right	r-value	INJ	NON	r-value	INJ	NON
EAIA	0.99±0.01	75.54±3.80	78.80±4.84	0.99±0.01	75.20±6.57	72.88±6.73	0.98±0.01	74.28±12.00	76.54±6.63
EAIK	0.96±0.02	4.22±1.20	3.44±0.49	0.94±0.04	6.97±2.68	15.46±3.30*$_{lr}$+	0.94±0.04	9.92±3.34*$_{r}$	15.83±4.8*$_{lr}$+#$_{i}$
EAIH	0.72±0.12	9.98±4.57	12.93±5.30	0.89±0.07	16.15±8.05	13.42±6.79	0.94±0.02	11.36±5.82	8.51±4.63
WorkA	0.99±0.01	120.60±23.85	128.48±28.34	0.98±0.01	121.52±18.67	124.47±20.19	0.98±0.01	132.56±31.16	116.44±13.62
WorkK	0.93±0.05	8.57±2.84	6.30±2.47	0.68±0.20	14.37±9.53	22.38±6.62*$_{lr}$+	0.79±0.13	10.24±4.46*$_{r}$	19.08±6.53*$_{lr}$+
WorkH	0.42±0.36	16.80±4.65	23.77±5.31+	0.80±0.19	28.32±15.61	23.38±11.67	0.75±0.16	22.64±8.21	17.34±6.50
AngA	0.98±0.01	2.39±1.66	3.44±0.33	0.97±0.02	-2.15±1.52*$_{lr}$	-0.27±1.15	0.98±0.01	-1.09±1.08*$_{lr}$	-2.86±1.14*$_{lr}$
AngK	0.99±0.01	12.97±0.87	15.22±1.92	0.97±0.02	14.79±4.57	18.92±2.62*$_{l}$	0.93±0.04	17.63±4.42*$_{l}$	18.74±3.91*$_{l}$
AngH	0.89±0.07	10.32±2.71	11.24±2.63	0.86±0.12	14.65±2.17*$_{lr}$	15.71±1.82*$_{lr}$	0.96±0.03	15.85±2.19*$_{lr}$	15.95±2.95*$_{lr}$

† Values indicate extensor and plantarflexor moments (Nm/kg)

†† Values are Joules (J)

††† Positive values indicate flexion and dorsiflexion, negative values indicate extension and plantarflexion (degrees)

\+ Significantly different than opposite limb ($p < 0.05$)

*$_{l}$ Significantly different than CON left limb ($p < 0.05$)

*$_{r}$ Significantly different than CON right limb ($p < 0.05$)

#$_{i}$ Significantly different than ACLD injured limb ($p < 0.05$)

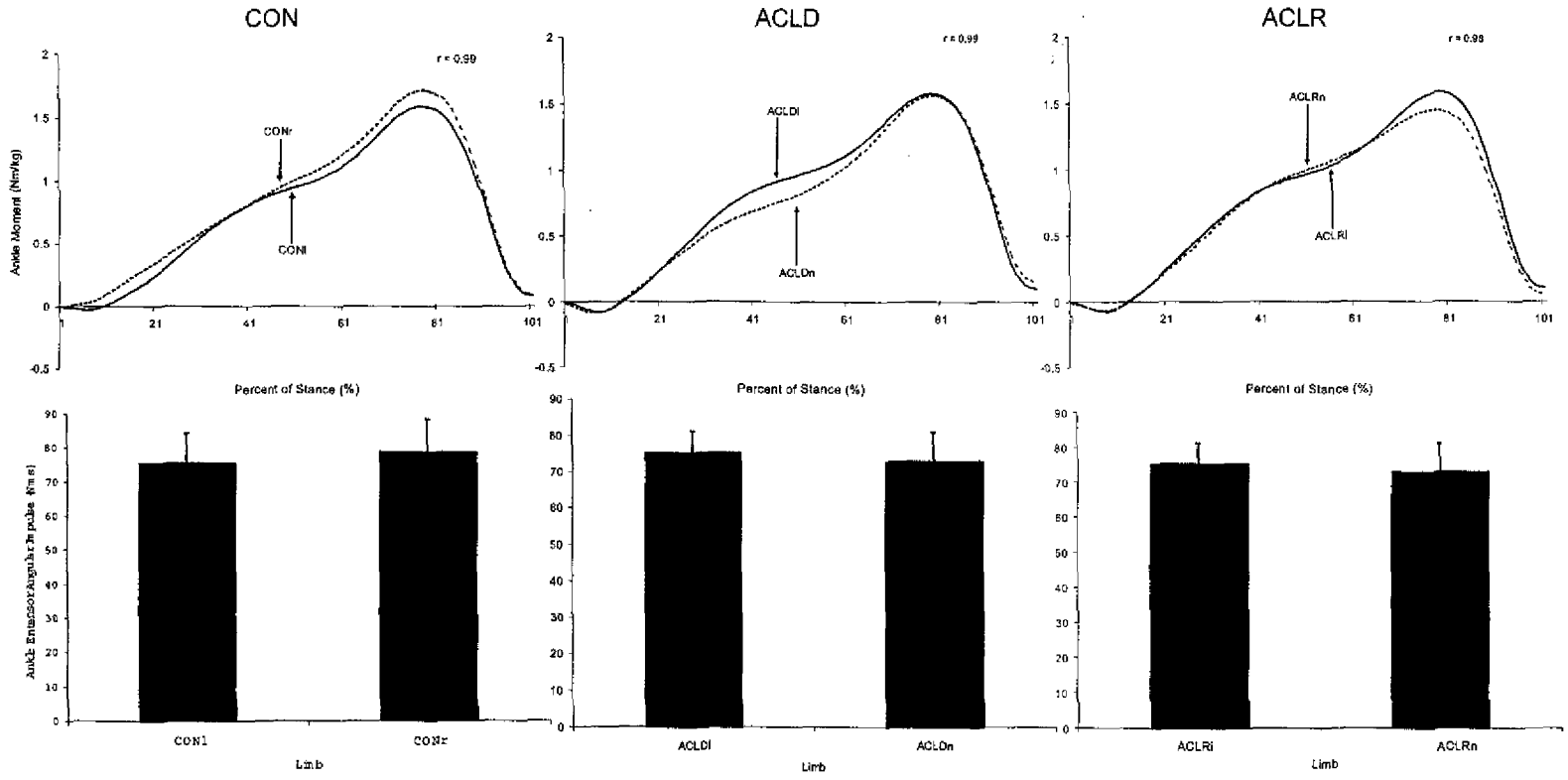

FIGURE 5.1 Bilateral ankle moment curves (top graphs) and extensor angular impulse (bottom graphs) of control (CON: left graphs), anterior cruciate ligament deficient (ACLD: middle graphs), and anterior cruciate ligament reconstructed (ACLR: right graphs) subjects during NP condition. Positive values indicate plantarflexor moments, negative values indicate dorsiflexor moments.

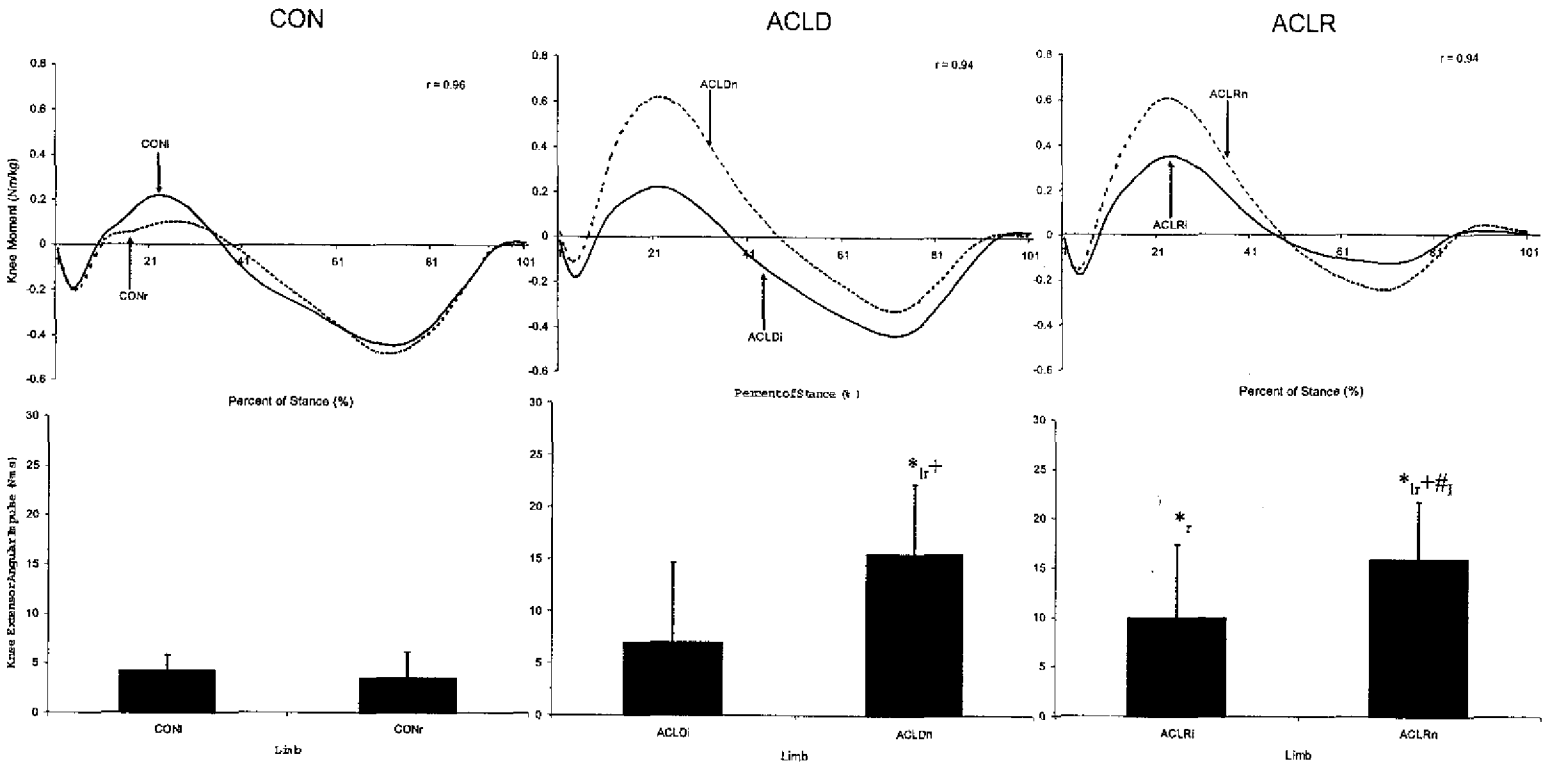

FIGURE 5.2 Bilateral knee moment curves (top graphs) and extensor angular impulse (bottom graphs) of control (CON: left graphs), anterior cruciate ligament deficient (ACLD: middle graphs), and anterior cruciate ligament reconstructed (ACLR: right graphs) subjects during NP condition. Positive values indicate plantarflexor moments, negative values indicate dorsiflexor moments.

\+ Significantly different than opposite limb ($p < 0.05$)

$*_l$ Significantly different than CON left limb ($p < 0.05$)

$*_r$ Significantly different than CON right limb ($p < 0.05$)

$\#_i$ Significantly different than ACLD injured limb ($p < 0.05$)

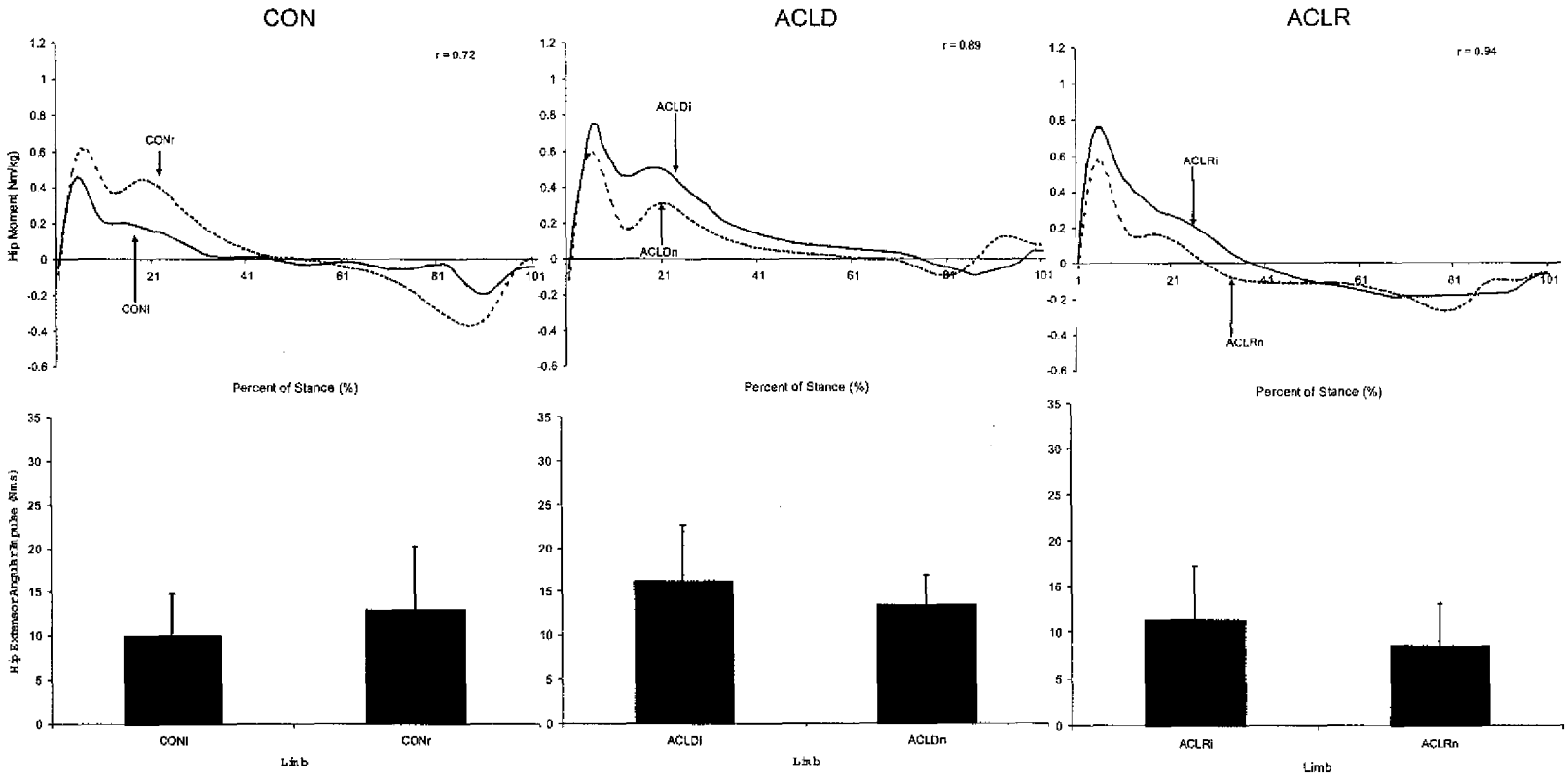

FIGURE 5.3 Bilateral hip moment curves (top graphs) and extensor angular impulse (bottom graphs) of control (CON: left graphs), anterior cruciate ligament deficient (ACLD: middle graphs), and anterior cruciate ligament reconstructed (ACLR: right graphs) subjects during NP condition. Positive values indicate plantarflexor moments, negative values indicate dorsiflexor moments.

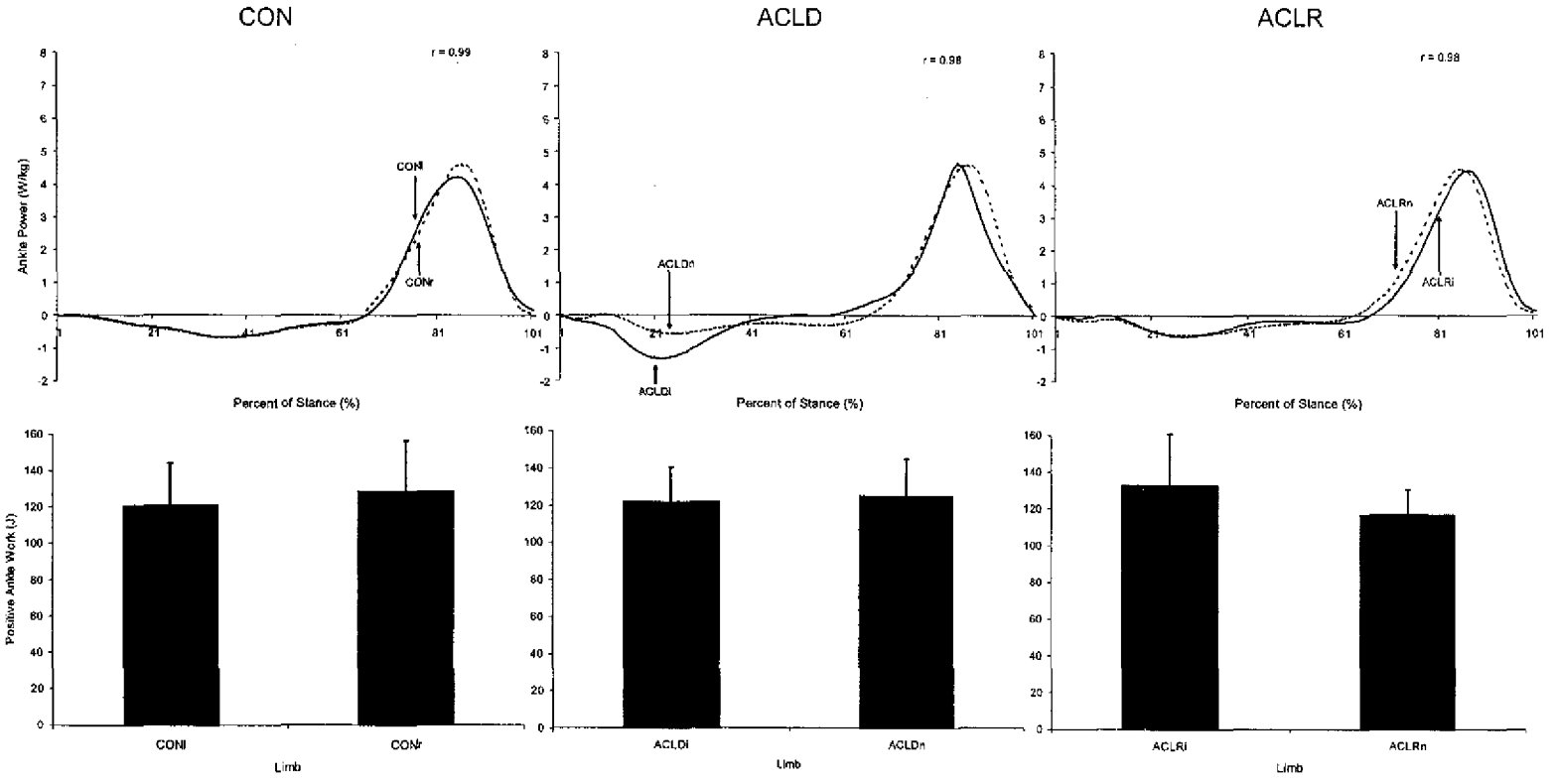

FIGURE 5.4 Bilateral ankle power curves (top graphs) and extensor angular impulse (bottom graphs) of control (CON: left graphs), anterior cruciate ligament deficient (ACLD: middle graphs), and anterior cruciate ligament reconstructed (ACLR: right graphs) subjects during NP condition. Positive and negative values are energy generation and absorption by the muscles.

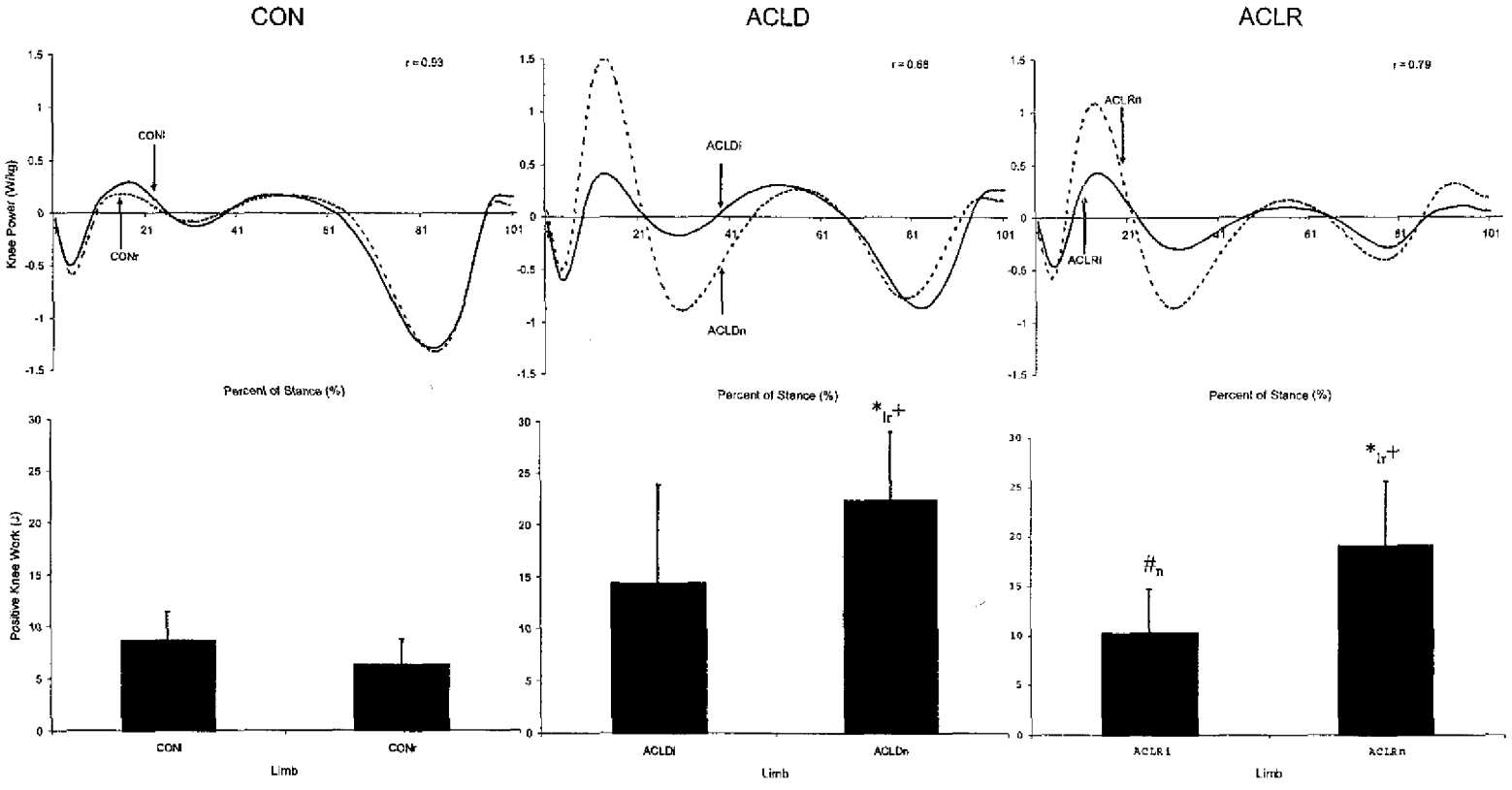

FIGURE 5.5 Bilateral knee power curves (top graphs) and extensor angular impulse (bottom graphs) of control (CON: left graphs), anterior cruciate ligament deficient (ACLD: middle graphs), and anterior cruciate ligament reconstructed (ACLR: right graphs) subjects during NP condition. Positive and negative values are energy generation and absorption by the muscles.

+ Significantly different than opposite limb ($p < 0.05$)

$*_l$ Significantly different than CON left limb ($p < 0.05$)

$*_r$ Significantly different than CON right limb ($p < 0.05$)

$\#_n$ Significantly different than ACLD non-injured limb ($p < 0.05$)

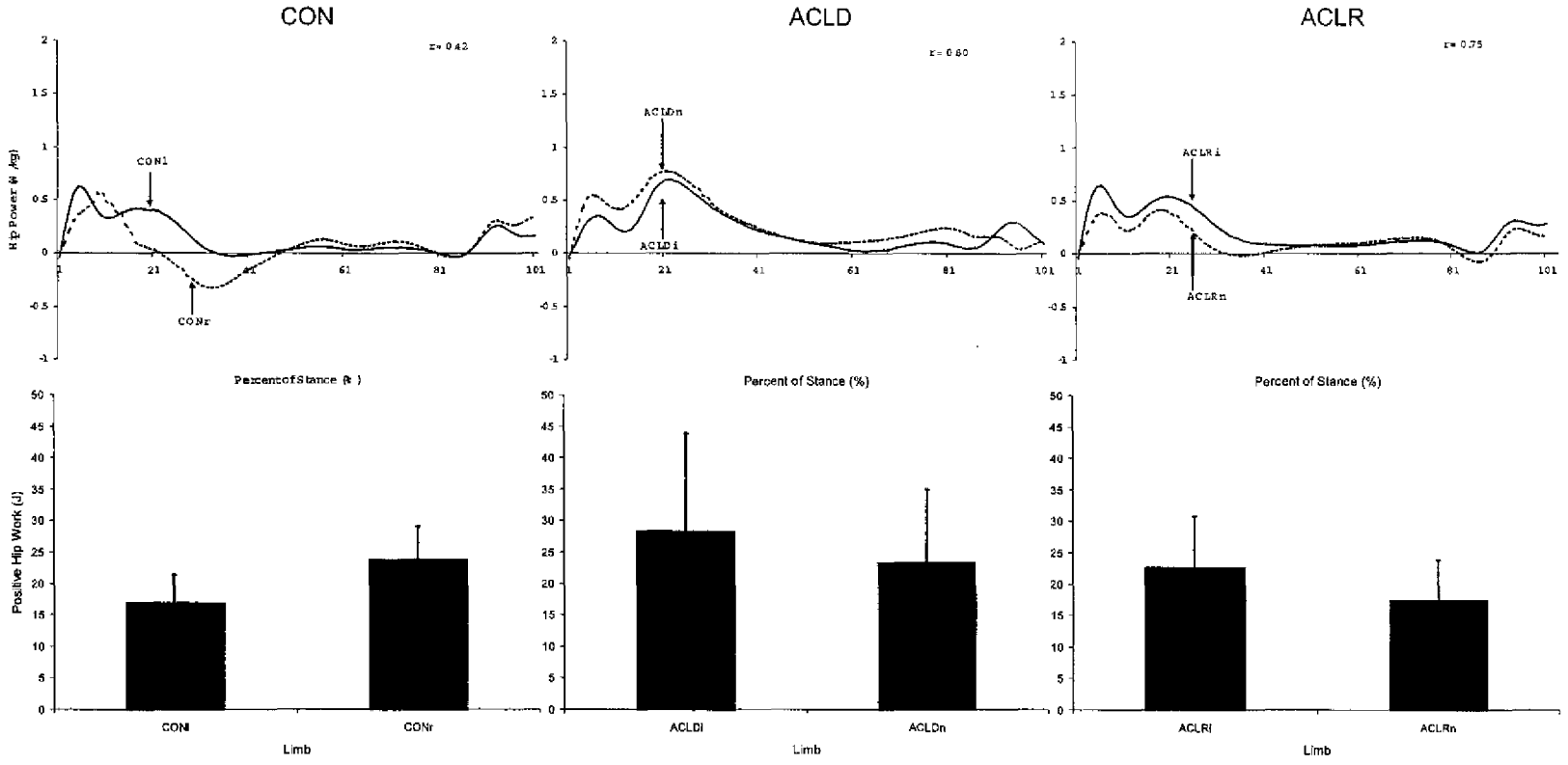

FIGURE 5.6 Bilateral hip power curves (top graphs) and extensor angular impulse (bottom graphs) of control (CON: left graphs), anterior cruciate ligament deficient (ACLD: middle graphs), and anterior cruciate ligament reconstructed (ACLR: right graphs) subjects during NP condition. Positive and negative values are energy generation and absorption by the muscles.

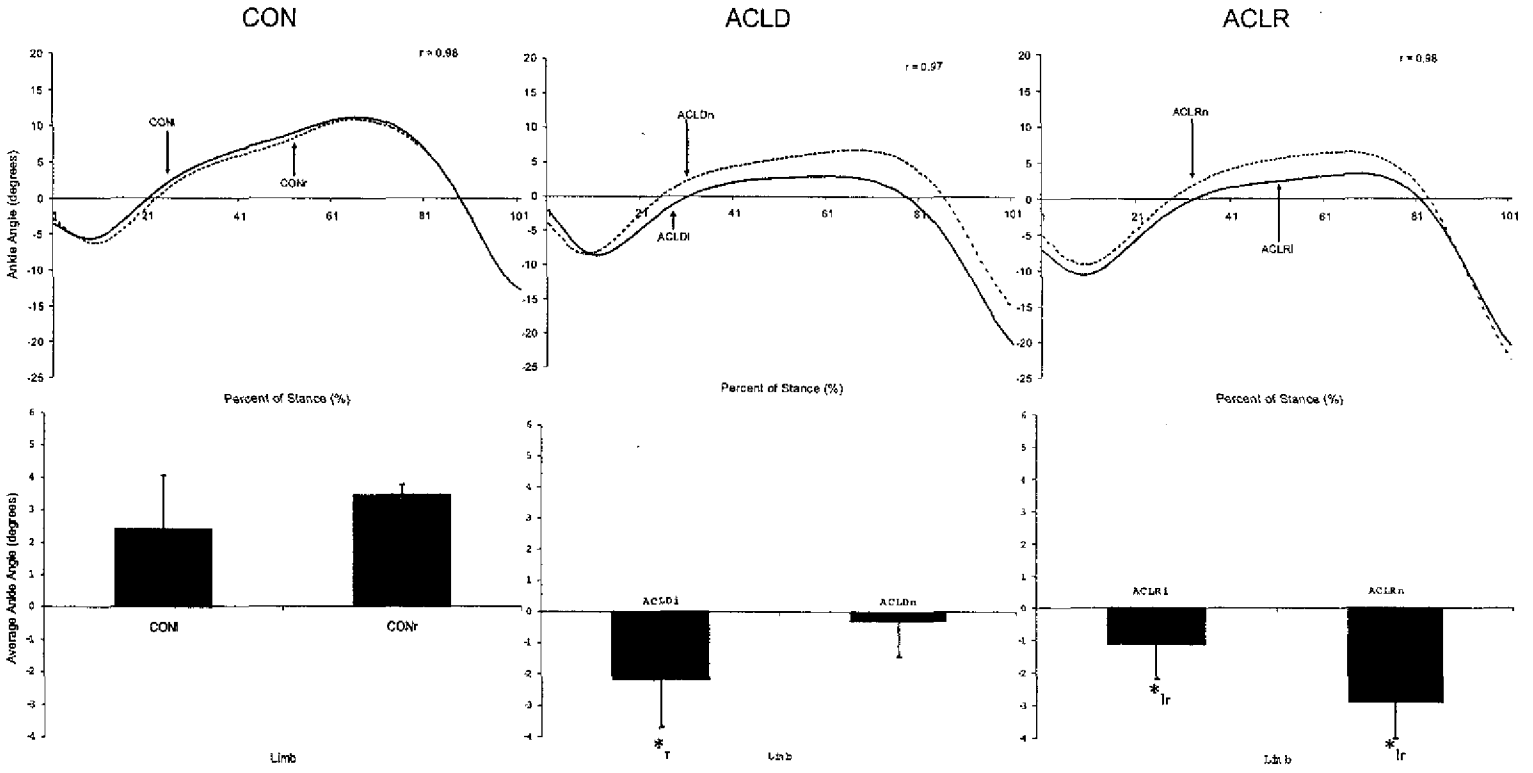

FIGURE 5.7 Bilateral ankle position curves (top graphs) and extensor angular impulse (bottom graphs) of control (CON: left graphs), anterior cruciate ligament deficient (ACLD: middle graphs), and anterior cruciate ligament reconstructed (ACLR: right graphs) subjects during NP condition. Positive and negative values are flexed and extended positions.

*$_l$ Significantly different than CON left limb ($p < 0.05$)

*$_r$ Significantly different than CON right limb ($p < 0.05$)

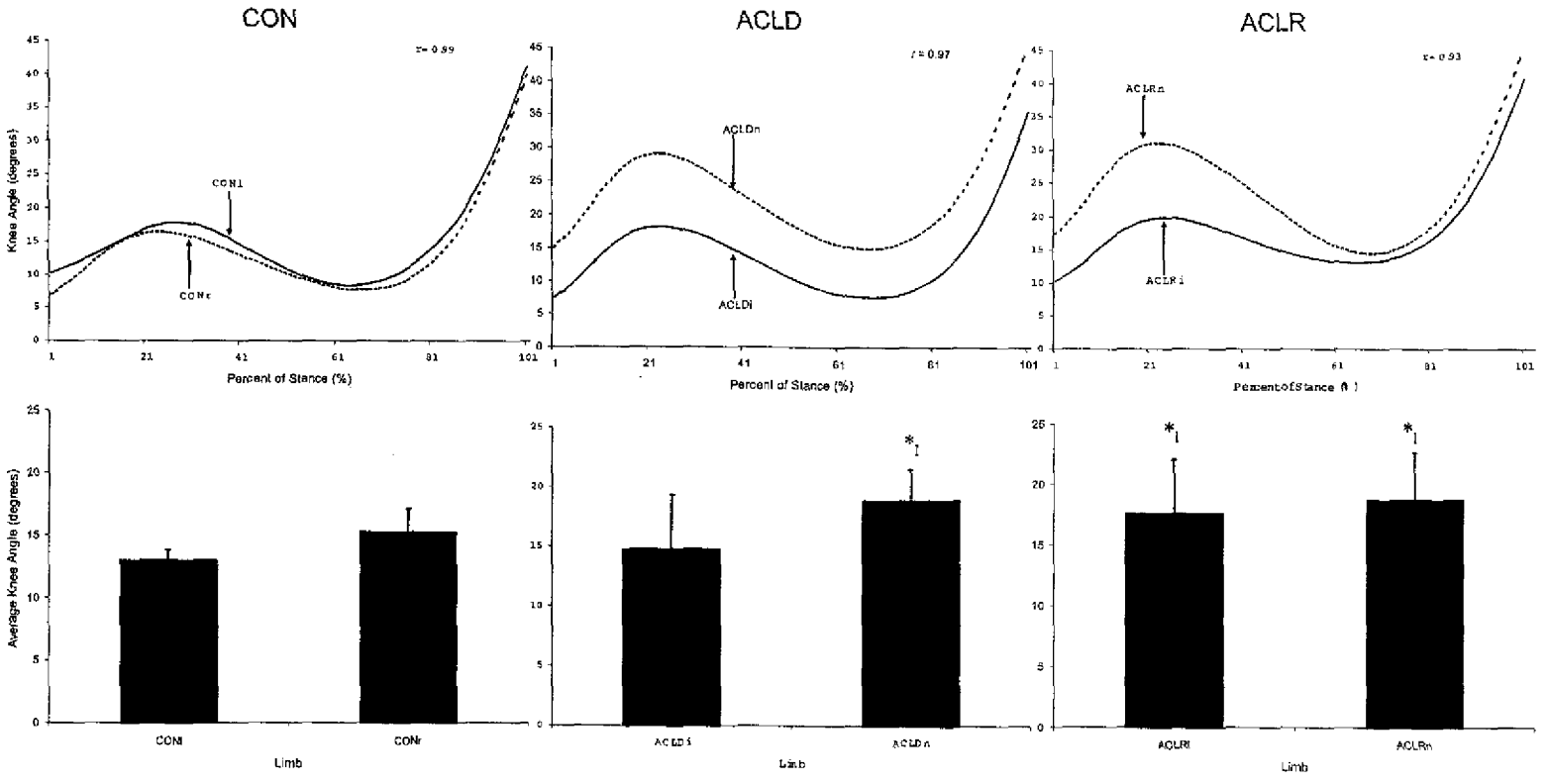

FIGURE 5.8 Bilateral knee position curves (top graphs) and extensor angular impulse (bottom graphs) of control (CON: left graphs), anterior cruciate ligament deficient (ACLD: middle graphs), and anterior cruciate ligament reconstructed (ACLR: right graphs) subjects during NP condition. Positive and negative values are flexed and extended positions.

$*_l$ Significantly different than CON left limb ($p < 0.05$)

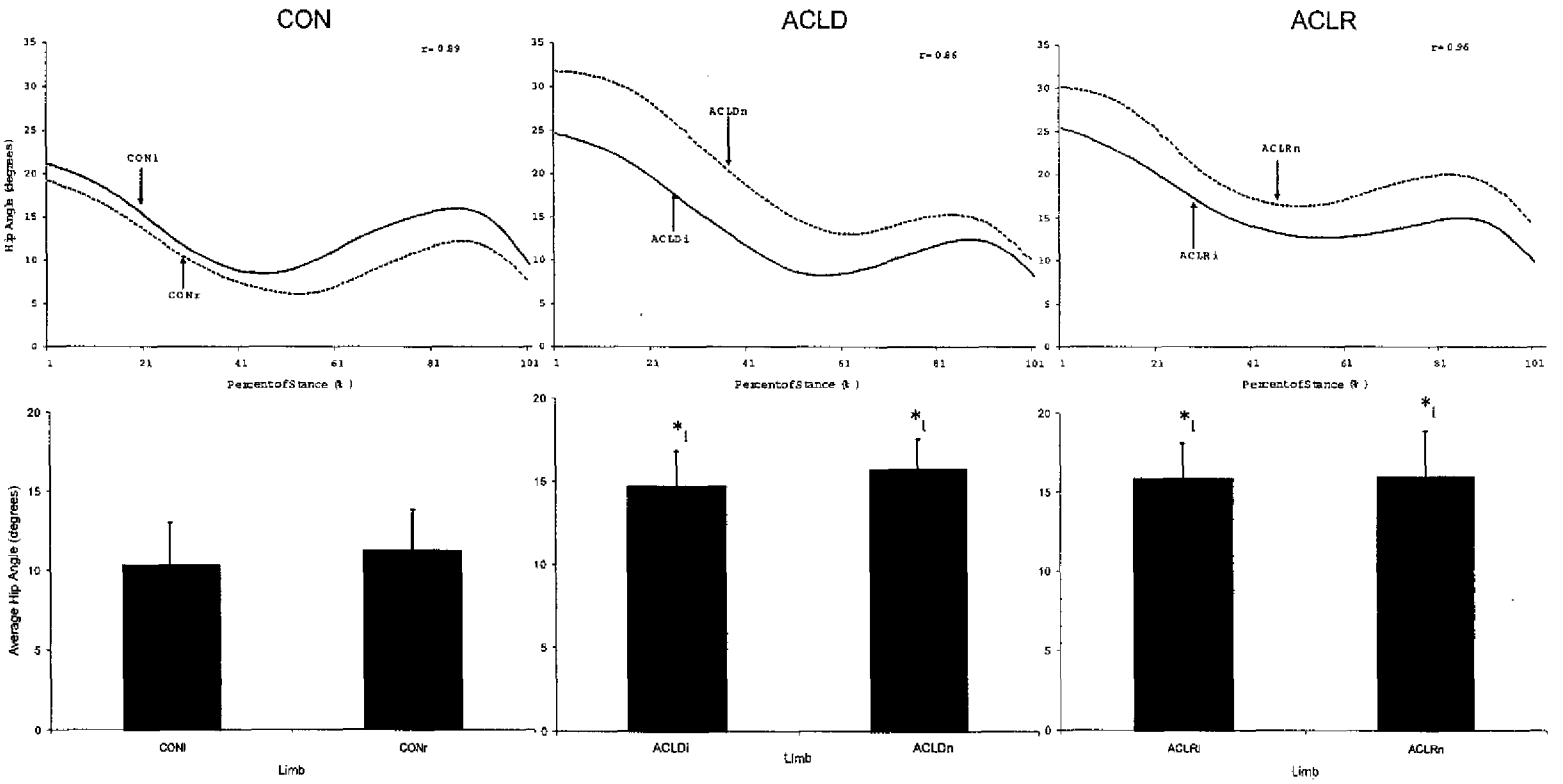

FIGURE 5.9 Bilateral hip position curves (top graphs) and extensor angular impulse (bottom graphs) of control (CON: left graphs), anterior cruciate ligament deficient (ACLD: middle graphs), and anterior cruciate ligament reconstructed (ACLR: right graphs) subjects during NP condition. Positive and negative values are flexed and extended positions.

$*_l$ Significantly different than CON left limb ($p < 0.05$)

$*_r$ Significantly different than CON right limb ($p < 0.05$)

Forward Perturbation Condition

TABLE 5.3 presents a comparison of CON, ACLD, and ACLR bilateral lower extremity Pearson product-moment correlation coefficient r-values and bilateral total joint extensor angular impulse (EAI), positive work (Pow), and average angle (Ang) during FP stance.

Joint Moments

The shapes of the bilateral joint FP moment curves for each group were similar and highly correlated with one another (range 0.88 – 0.99: TABLE 5.3; FIGURE 5.10-5.13). The CON, ACLD, and ACLR ankle FP moment curves were all highly correlated to the corresponding contralateral limb and no significant ($p>0.05$) differences in ankle EAI were observed between limbs or between the CON, ACLD, or ACLR groups (TABLE 5.2; FIGURE 5.1).

The bilateral knee FP moment curves were highly correlated for the 3 groups (range 0.88-0.94) but magnitude differences in EAI were observed (TABLE 5.3; FIGURE 5.11). The ACLDn and ACLRn knee produced significantly ($p<0.05$) more FP EAI compared to CON (TABLE 5.3; FIGURE 5.11).

The hip FP moment curves were all highly correlated (range 0.94-0.97) to the corresponding contralateral limb and no significant ($p>0.05$) differences in hip EAI were observed between limbs or between the CON, ACLD, or ACLR groups (TABLE 5.3; FIGURE 5.12).

Joint Powers

The shapes of the bilateral joint FP power curves for each group were similar and ranged from poor to highly correlated with one another (range 0.52 – 0.97: TABLE 5.3; FIGURE 5.13-5.14). The CON, ACLD, and ACLR ankle FP power curves were all highly correlated (range 0.96-0.97) to the corresponding contralateral limb and no significant ($p>0.05$) differences in positive FP ankle work were observed between limbs or between the CON, ACLD, or ACLR groups (TABLE 5.3; FIGURE 5.13).

The bilateral CON knee FP power curves were highly correlated (0.84) but the ACLD (0.52) and ACLR (0.53) knee FP power curves were poorly correlated between limbs (TABLE 5.3; FIGURE 5.14). No significant ($p>0.05$) differences in positive FP knee work were observed between limbs or between the CON, ACLD, or ACLR groups (TABLE 5.3; FIGURE 5.14).

The bilateral CON (0.80) and ACLR (0.82) hip FP power curves were highly correlated but the ACLD (0.69) hip FP power curves were moderately correlated between limbs (TABLE 5.3; FIGURE 5.15). No significant ($p>0.05$) differences in positive FP hip work were observed between limbs or between the 3 groups (TABLE 5.3; FIGURE 5.15).

Joint Position

The shapes of the bilateral joint FP position curves for each group were similar and were moderately to highly correlated with one another (range 0.74 – 0.99: TABLE 5.3; FIGURE 5.16-5.18). The shapes of the bilateral ankle FP position curves were highly correlated (range 0.90-0.96) for the 3 groups but magnitude differences in average angle were observed (TABLE 5.3; FIGURE 5.16). The ACLDi and ACLRn FP ankles were significantly ($p<0.05$) more plantarflexed compared to CON (TABLE 5.3; FIGURE 5.16).

The shapes of the bilateral knee FP position curves were highly correlated (range 0.92-0.98) for the 3 groups but magnitude differences in average angle were observed (TABLE 5.3; FIGURE 5.17). The ACLDn FP knee was significantly ($p<0.05$) more flexed compared to bilateral CON knees (TABLE 5.3; FIGURE 5.17).

The bilateral hip position curves were moderately correlated for the CON group (0.74) and highly correlated (range 0.84-0.89) for the ACLD and ACLR groups. However, the contralateral ACLD and ACLR FP hips were significantly ($p<0.05$) more flexed compared to bilateral CON hips (TABLE 5.3; FIGURE 5.18).

TABLE 5.3 Pearson Product Correlation Coefficient r-values and Bilateral Means (± 1SD) for Extensor Angular Impulse (EAI†), Positive Work ††, and Average Angle (Ang†††) for the Ankle (A), Knee (K), and Hip (H) of Control (CON), Anterior Cruciate Ligament Deficient (ACLD), and Anterior Cruciate Ligament Reconstructed (ACLR) Subjects during FP Condition (n=10)

	CON			ACLD			ACLR		
Variable	r-value	Left	Right	r-value	INJ	NON	r-value	INJ	NON
EAIA	0.97±0.01	56.04±8.77	58.51±9.64	0.99±0.01	51.11±5.94	50.09±8.01	0.97±0.02	56.30±10.98	54.36±5.88
EAIK	0.94±0.01	9.72±1.58	8.39±2.66	0.91±0.05	14.79±7.71	20.64±6.79*$_{lr}$	0.88±0.04	15.28±8.79	20.34±6.44*$_{lr}$
EAIH	0.97±0.01	22.57±4.79	22.66±7.25	0.96±0.02	23.60±6.47	23.76±3.44	0.94±0.02	19.40±5.46	15.27±5.33
WorkA	0.96±0.02	93.42±3.74	96.65±6.63	0.97±0.02	104.85±18.04	110.78±17.05	0.97±0.02	111.25±25.78	104.80±15.22
WorkK	0.84±0.06	9.22±2.17	5.76±3.36	0.52±0.35	6.96±4.24	5.76±4.36	0.53±0.31	5.12±4.56	7.33±5.62
WorkH	0.80±0.09	32.24±9.62	31.55±8.36	0.69±0.19	33.74±15.09	38.56±13.73	0.82±0.07	34.63±15.96	29.64±8.36
AngA	0.90±0.08	4.61±1.15	3.50±0.32	0.95±0.02	-1.18±1.21*$_{lr}$	0.58±1.79	0.96±0.03	0.58±1.83	-1.62±3.80*$_{lr}$
AngK	0.98±0.08	13.70±0.44	14.73±2.15	0.92±0.03	15.88±4.20	19.62±3.57*$_{lr}$	0.93±0.05	17.27±4.51	18.42±4.32
AngH	0.74±0.12	11.49±2.95	11.91±2.88	0.84±0.13	16.09±2.36*$_{lr}$	16.08±2.07*$_{lr}$	0.89±0.07	16.50±2.07*$_{lr}$	16.07±2.86*$_{lr}$

† Values indicate extensor and plantarflexor moments (Nm/kg)

†† Values are Joules (J)

††† Positive values indicate flexion and dorsiflexion, negative values indicate extension and plantarflexion (degrees)

*$_{l}$ Significantly different than CON left limb ($p < 0.05$)

*$_{r}$ Significantly different than CON right limb ($p < 0.05$)

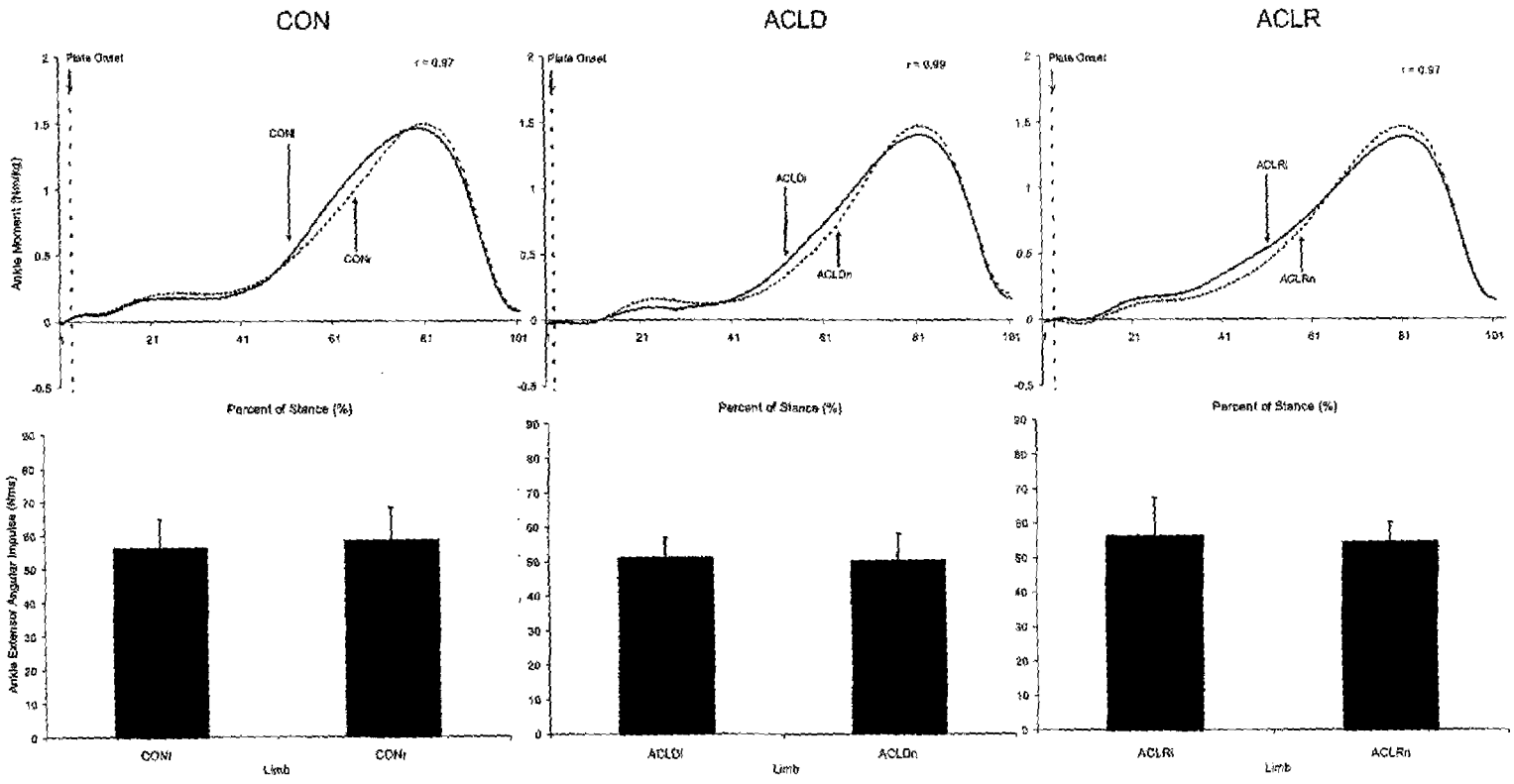

FIGURE 5.10 Bilateral ankle moment curves (top graphs) and extensor angular impulse (bottom graphs) of control (CON: left graphs), anterior cruciate ligament deficient (ACLD: middle graphs), and anterior cruciate ligament reconstructed (ACLR: right graphs) subjects during FP condition. Positive values indicate plantarflexor moments, negative values indicate dorsiflexor moments.

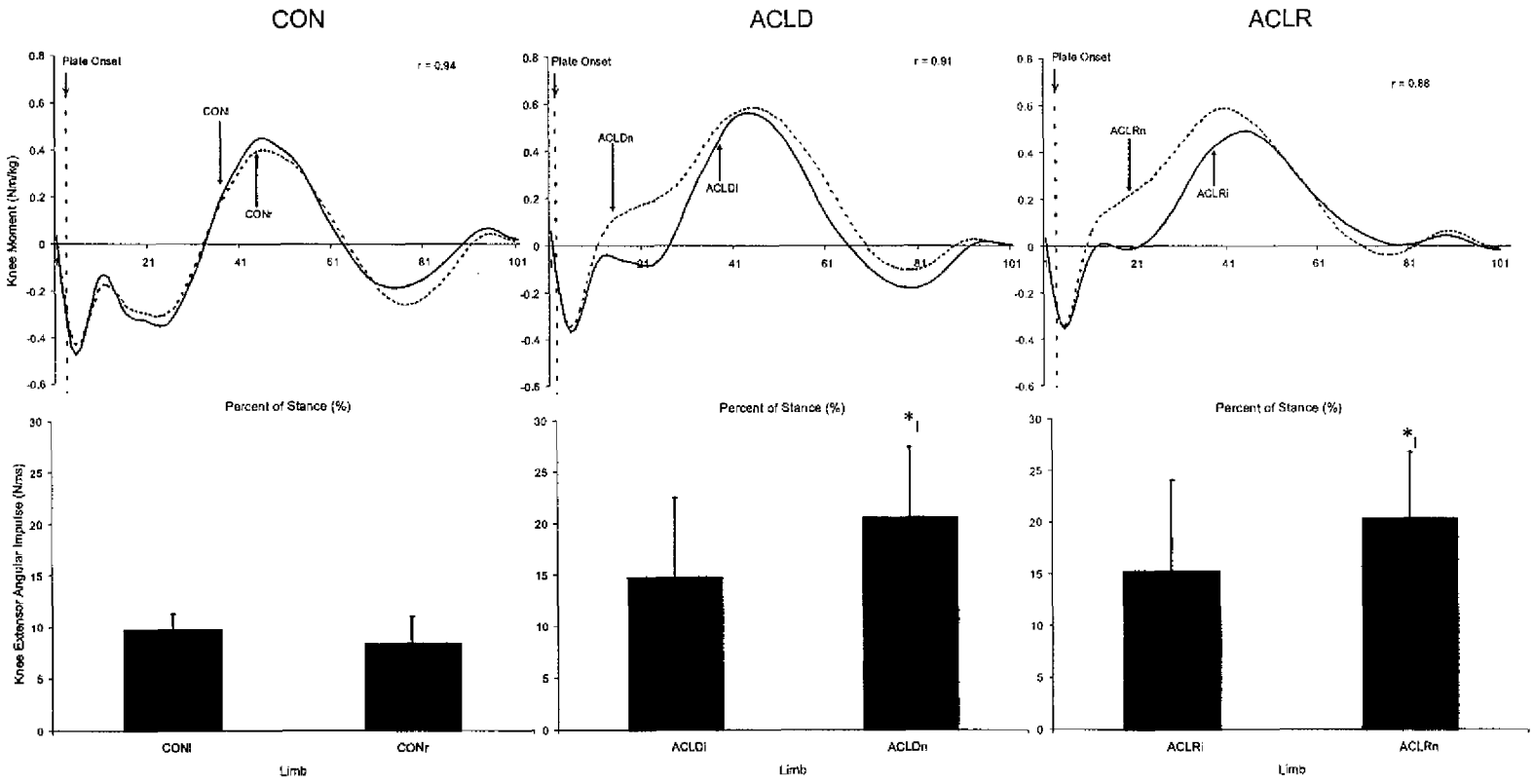

FIGURE 5.11 Bilateral knee moment curves (top graphs) and extensor angular impulse (bottom graphs) of control (CON: left graphs), anterior cruciate ligament deficient (ACLD: middle graphs), and anterior cruciate ligament reconstructed (ACLR: right graphs) subjects during FP condition. Positive values indicate plantarflexor moments, negative values indicate dorsiflexor moments.

$*_l$ Significantly different than CON left limb ($p < 0.05$)

$*_r$ Significantly different than CON right limb ($p < 0.05$)

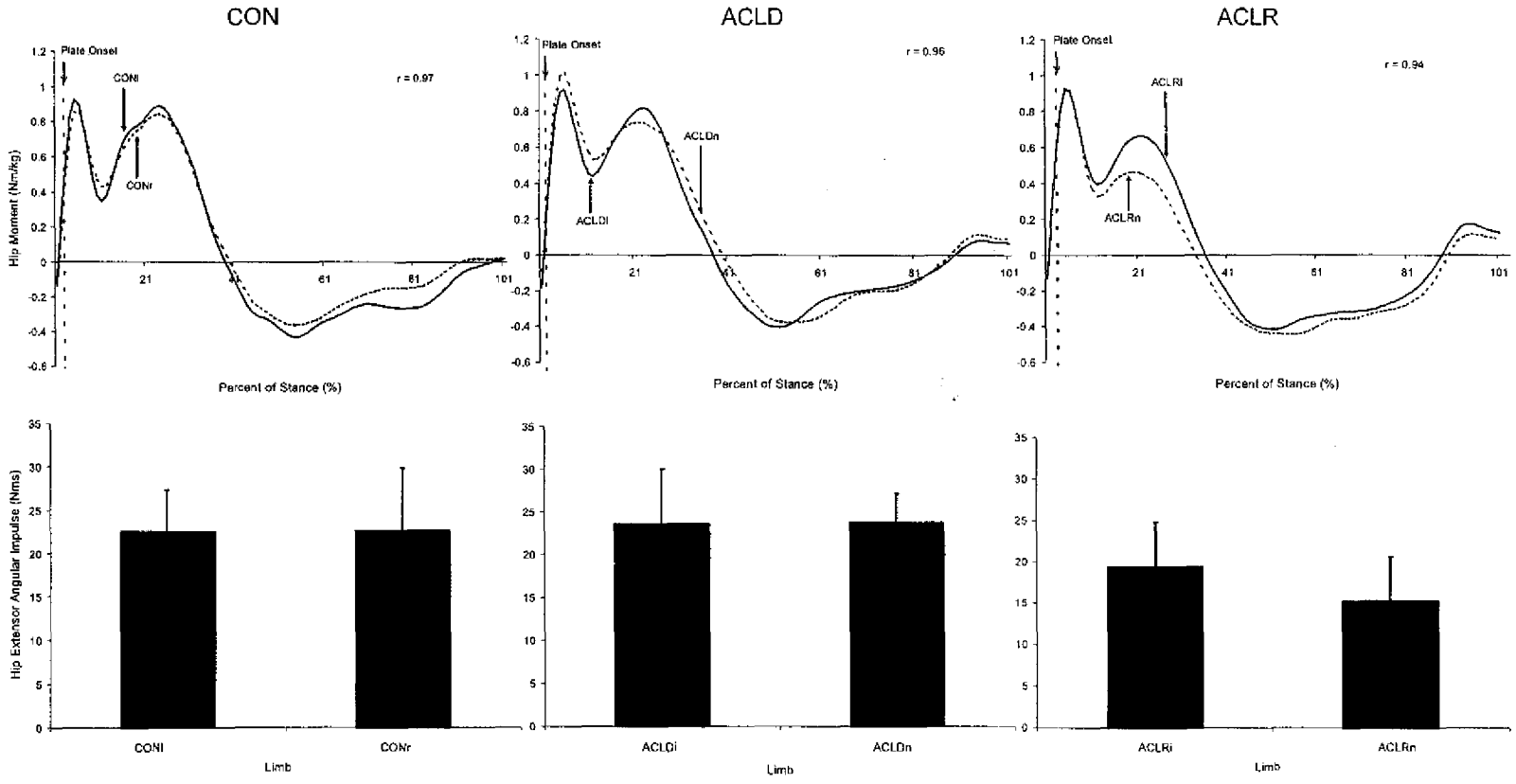

FIGURE 5.12 Bilateral hip moment curves (top graphs) and extensor angular impulse (bottom graphs) of control (CON: left graphs), anterior cruciate ligament deficient (ACLD: middle graphs), and anterior cruciate ligament reconstructed (ACLR: right graphs) subjects during FP condition. Positive values indicate plantarflexor moments, negative values indicate dorsiflexor moments.

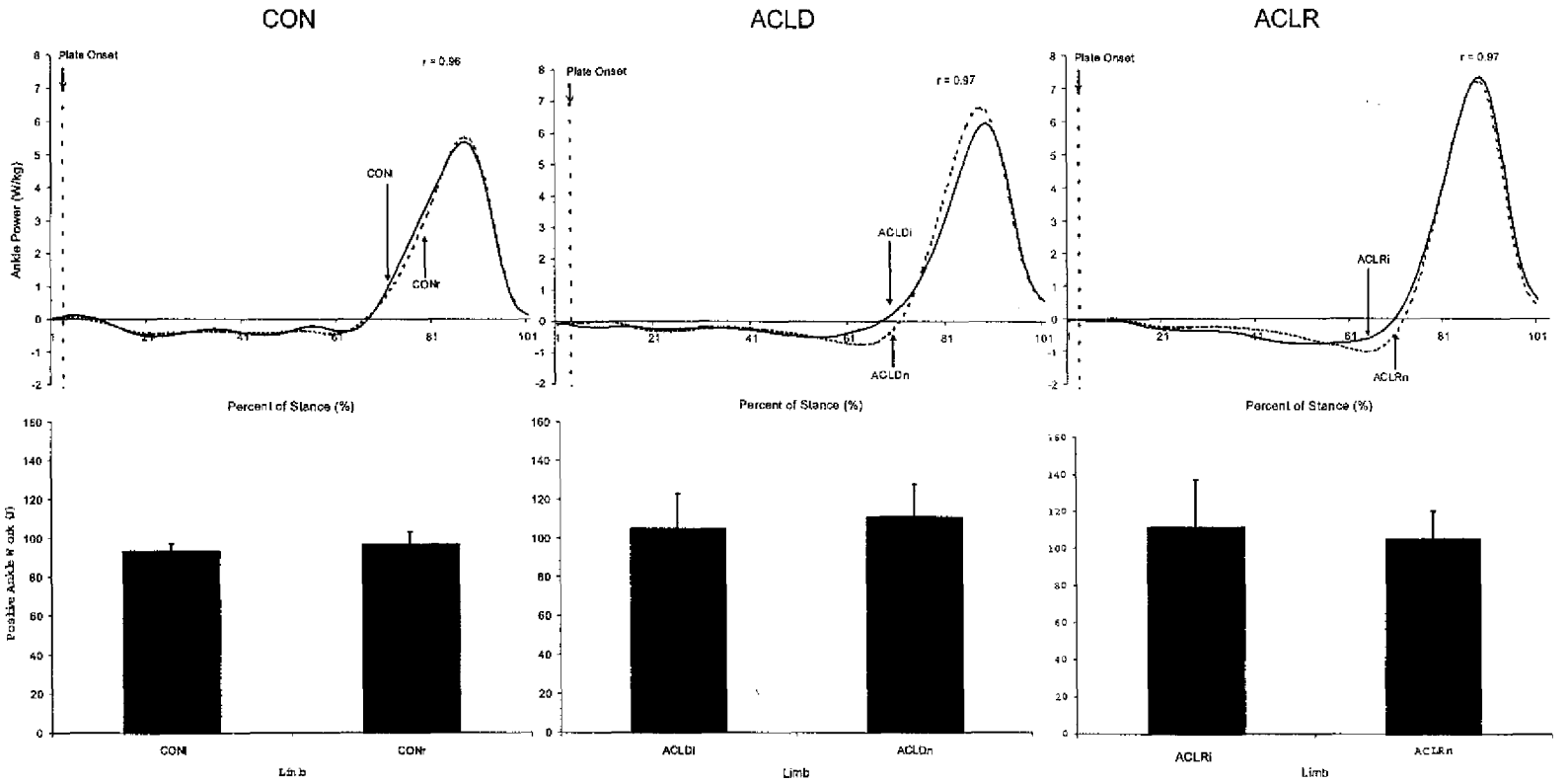

FIGURE 5.13 Bilateral ankle power curves (top graphs) and extensor angular impulse (bottom graphs) of control (CON: left graphs), anterior cruciate ligament deficient (ACLD: middle graphs), and anterior cruciate ligament reconstructed (ACLR: right graphs) subjects during FP condition. Positive and negative values are energy generation and absorption by the muscles.

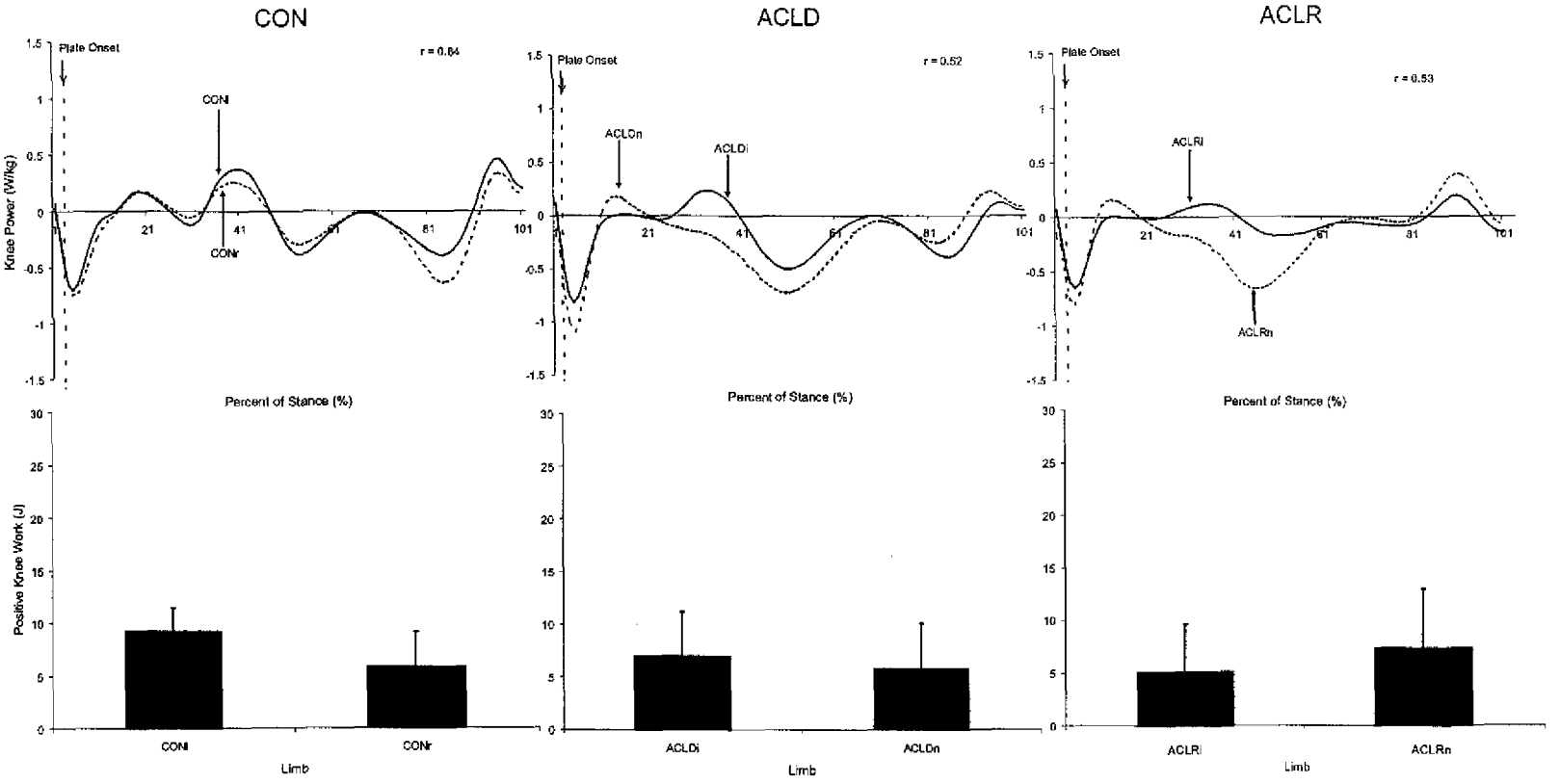

FIGURE 5.14 Bilateral knee power curves (top graphs) and extensor angular impulse (bottom graphs) of control (CON: left graphs), anterior cruciate ligament deficient (ACLD: middle graphs), and anterior cruciate ligament reconstructed (ACLR: right graphs) subjects during FP condition. Positive and negative values are energy generation and absorption by the muscles.

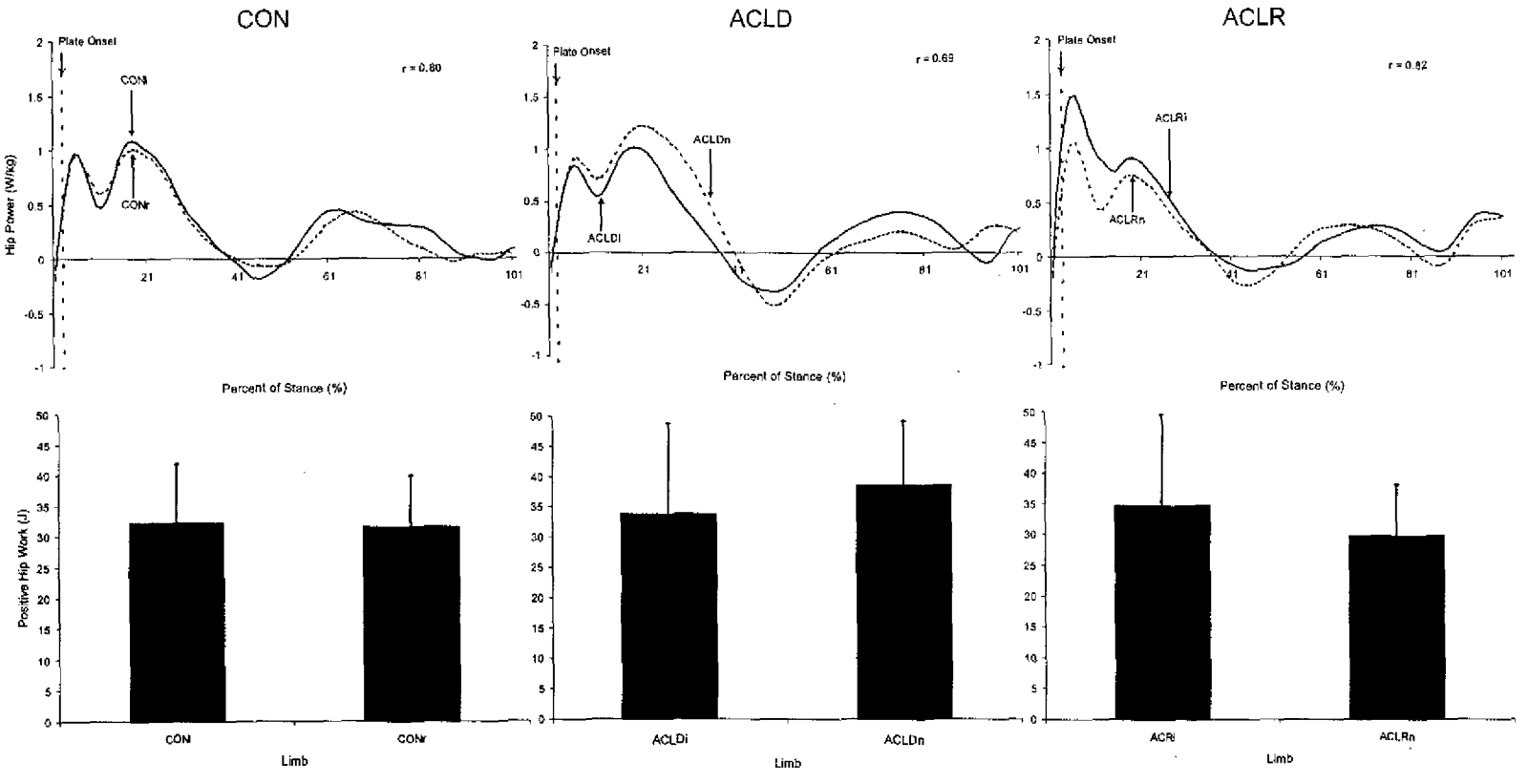

FIGURE 5.15 Bilateral hip power curves (top graphs) and extensor angular impulse (bottom graphs) of control (CON: left graphs), anterior cruciate ligament deficient (ACLD: middle graphs), and anterior cruciate ligament reconstructed (ACLR: right graphs) subjects during FP condition. Positive and negative values are energy generation and absorption by the muscles.

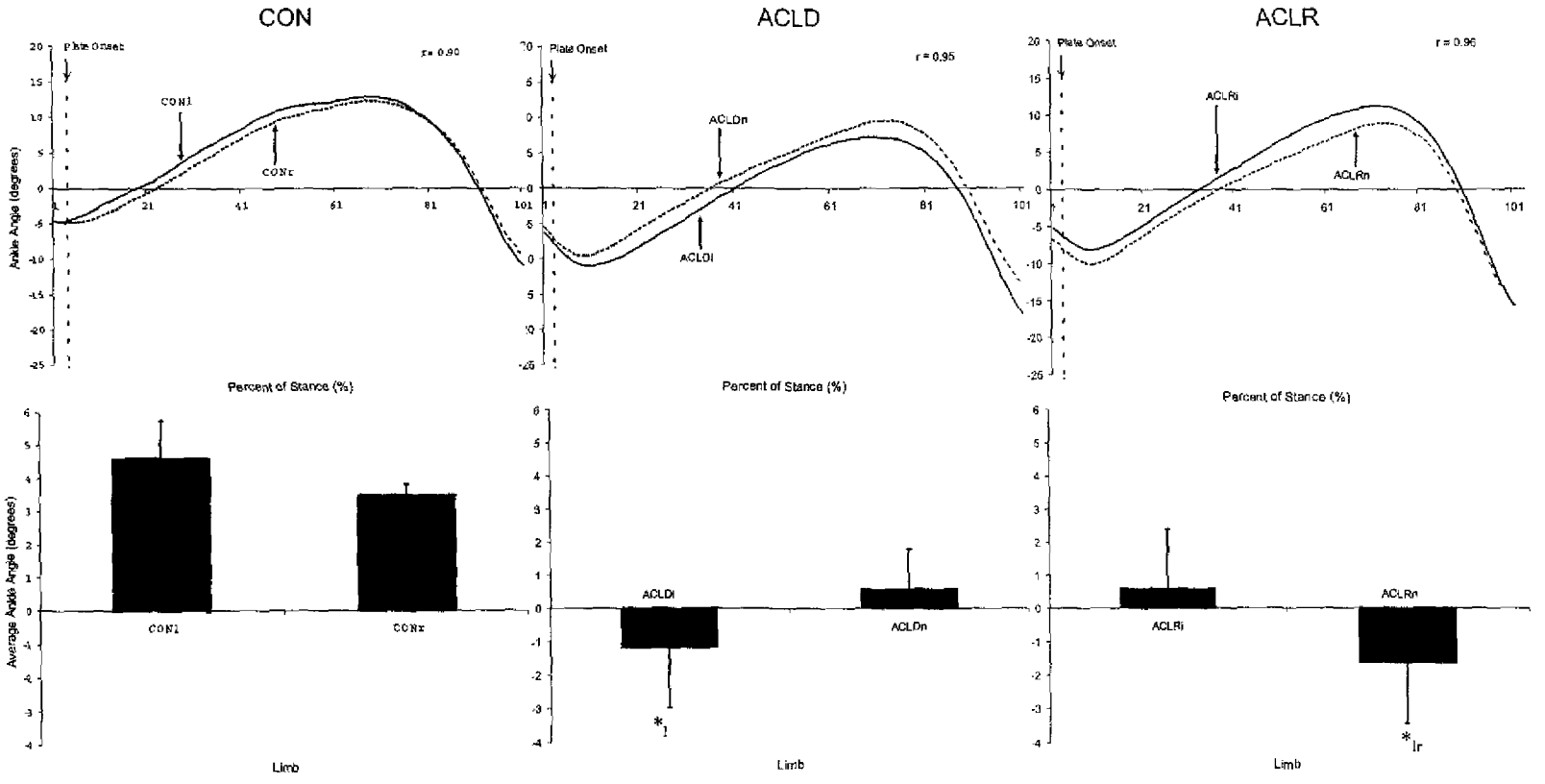

FIGURE 5.16 Bilateral ankle position curves (top graphs) and extensor angular impulse (bottom graphs) of control (CON: left graphs), anterior cruciate ligament deficient (ACLD: middle graphs), and anterior cruciate ligament reconstructed (ACLR: right graphs) subjects during FP condition. Positive and negative values are flexed and extended positions.

$*_l$ Significantly different than CON left limb ($p < 0.05$)

$*_r$ Significantly different than CON right limb ($p < 0.05$)

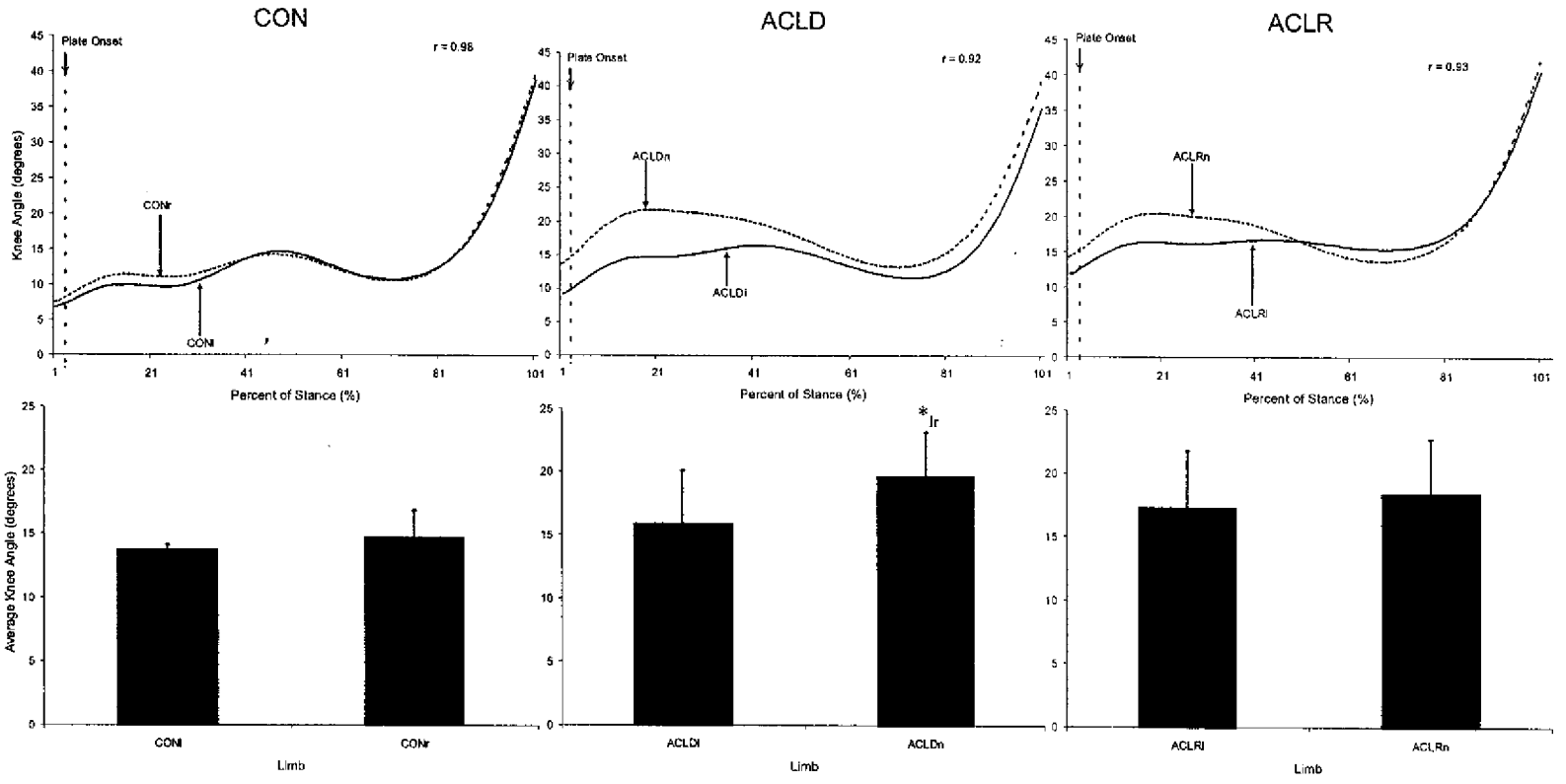

FIGURE 5.17 Bilateral knee position curves (top graphs) and extensor angular impulse (bottom graphs) of control (CON: left graphs), anterior cruciate ligament deficient (ACLD: middle graphs), and anterior cruciate ligament reconstructed (ACLR: right graphs) subjects during FP condition. Positive and negative values are flexed and extended positions.

*$_l$ Significantly different than CON left limb ($p < 0.05$)

*$_r$ Significantly different than CON right limb ($p < 0.05$)

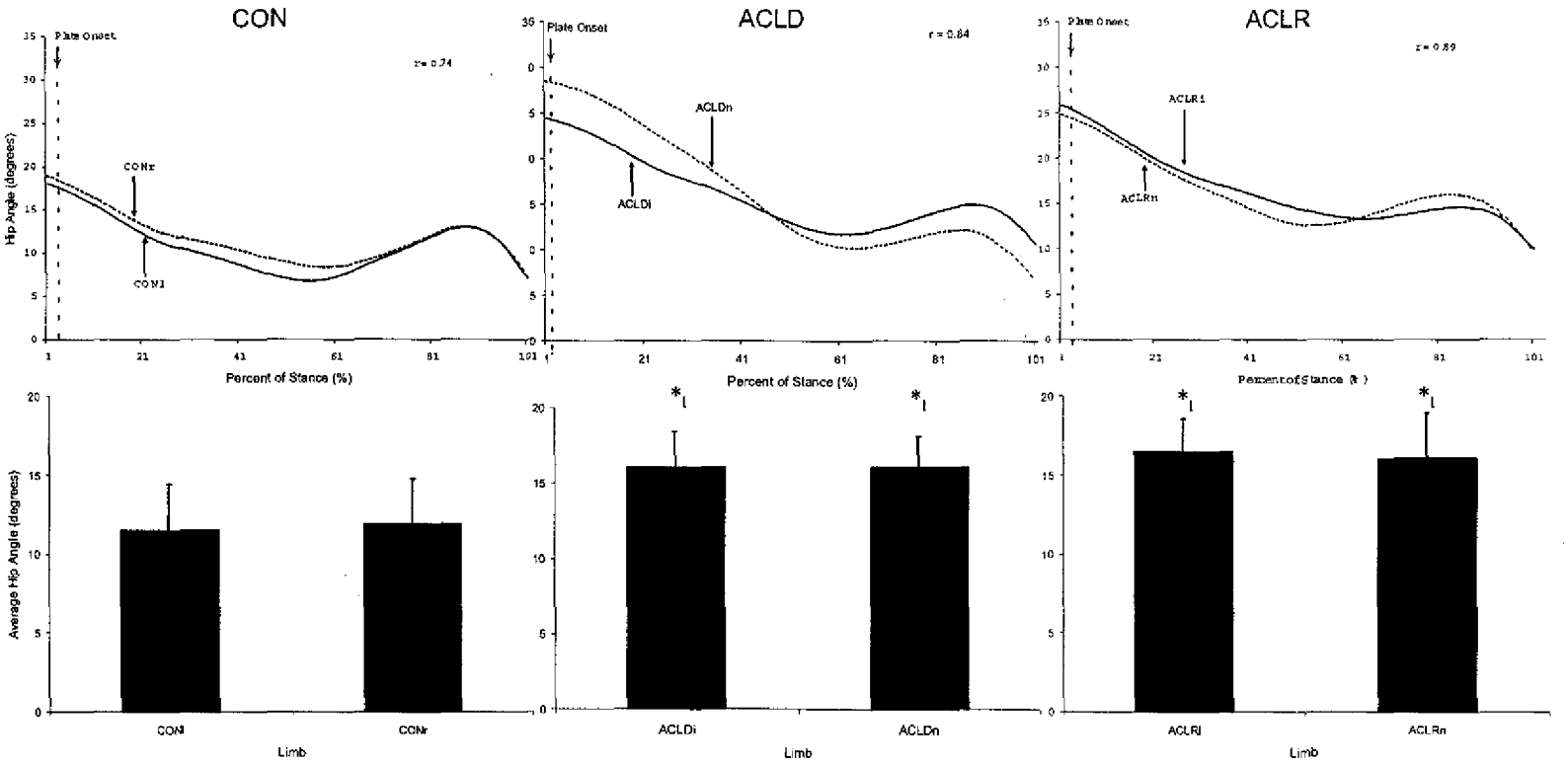

FIGURE 5.18 Bilateral hip position curves (top graphs) and extensor angular impulse (bottom graphs) of control (CON: left graphs), anterior cruciate ligament deficient (ACLD: middle graphs), and anterior cruciate ligament reconstructed (ACLR: right graphs) subjects during FP condition. Positive and negative values are flexed and extended positions.

$*_l$ Significantly different than CON left limb ($p < 0.05$)

$*_r$ Significantly different than CON right limb ($p < 0.05$)

Hypotheses Results

It was hypothesized that the ACLD and ACLR subjects would be more symmetrical in gait mechanics compared to the gait patterns of the uninjured control subjects during NP gait and in response to the FP. These hypotheses were partially confirmed as results demonstrated that during NP gait the control subjects exhibited hip asymmetry (FIGURES 5.3 & 5.6) and knee symmetry (FIGURES 5.2 & 5.5) while the ACLD and ACLR subjects exhibited hip symmetry (FIGURES 5.3 & 5.6) and knee asymmetry (FIGURES 5.2 & 5.5). In response to the FP, the CON group exhibited lower extremity joint symmetry (FIGURES 5.10 – 5.16) while the ACLD and ACLR group exhibited knee power asymmetry (FIGURE 5.14).

Discussion

The purpose of this study was to determine the effect of ACL injury and subsequent surgery on bilateral lower extremity joint kinematic, moment, and power patterns during non-perturbed and perturbed gait. While gait symmetry has been supported in the literature (Hamill et al., 1984; Menard et al., 1992), others have reported that gait is asymmetrical in healthy populations (Allard et al., 1996; Sadeghi et al., 1997, 2000). Few studies have investigated possible gait asymmetry with respect to joint accommodations due to ACL injury (Berchuck, et al., 1990; Kowalk et al., 1997; Roberts et al., 1999; Rudolph et al., 1998). Of those studies, none have conducted a comprehensive bilateral lower extremity gait analysis utilizing a population of chronic ACLD subjects prior to and following surgical repair nor has any study investigated the effects of unexpected gait perturbations. It is possible that contralateral joint accommodations could occur as a result of the injury itself and in response to surgical repair.

Non-perturbed Gait

Control Group Hip Asymmetry

Bilateral CON ankle and knee moment, power, and position patterns during NP stance were similar in both temporal and magnitude measures (TABLE 5.2). These findings are consistent with previous literature (Allard et al., 1996; Eng & Winter, 1995; Sagedhi et al., 2000). However, significant bilateral differences in CON hip positive work and low to moderate between-limb correlation coefficient values were observed for the CON hip power (r=0.42) and moment (r=0.72) curves (TABLE 5.2; FIGURE 5.6). It has been previsouly reported that hip power patterns in healthy individuals were asymmetrical due to different limb functions during

stance (Sadeghi et al., 2000). These authors studied gait asymmetry in healthy adults by measuring consecutive gait cycles for the purpose of identifying functional differences in the lead or trail leg. Sadeghi et al. (2000) reported that the lead leg was mostly responsible for propulsion while the trail leg was mostly responsible for control and stability during the stance phase of gait. However, all subjects involved in the study were right limb dominant and the right limb was always the lead limb. Therefore, it is unknown whether subjects would have exhibited similar functionally related asymmetry if the non-dominant limb were used as the lead limb. In the present investigation, all CON subjects were right limb dominant but consecutive gait cycles were not studied. Thus, it is difficult to match these results with those of Sadeghi et al. (2000). However, since each limb was used as the lead limb, perhaps the different hip moment and power patterns exhibited by the CON group were due to limb dominance. In the present study, the small CONl hip flexor moment and negligible CONl hip power production that was observed during mid-stance could be characteristic of the non-dominant hip. In contrast, the greater hip moment and significantly greater work performed by the CONr hip during early and late stance could be more characteristic of the dominant hip.

Control Group Hip-Knee Trade-off

It has been reported that changes in hip moment patterns are equally matched by alterations in knee moment patterns during gait (Ferber, 2001, Winter, 1987). Winter (1987) hypothesized that such a deterministic trade-off between the hip and the knee indicated a stride dependent control of the head-arm-trunk (HAT) segment to maintain the total support moment to prevent the body from collapsing due to gravitational forces. Although bilateral CON hip moment patterns were asymmetrical and bilateral CON knee moment patterns were symmetrical, results from the present investigation support the ipsilateral hip-knee trade-off premise put forth

by Winter (1987). Examination of FIGURES 5.2 and 5.3 indicate that, during early NP stance, the CONr hip exhibited a greater extensor moment as compared to the CONl hip and the CONr knee demonstrated a reduced extensor moment as compared to the CONl knee (TABLE 5.2; FIGURE 5.3 & 5.4). Perhaps this reciprocal trade-off was necessary due to the coordination between posterior muscles (hip extensors/knee flexors) and anterior muscles (hip flexors/knee extensors) as a result of muscle bi-articular function.

Examination of TABLE 5.2 reveals that the total joint EAI remained similar for both limbs (CONr: 89.74±9.57; CONl: 95.17±10.63) suggesting that the total moment of support was maintained regardless of differences between bilateral joint moment patterns. Other studies have also reported similar bilateral support moments in healthy subjects (Winter, 1987), in subjects who exhibited pathological gait patterns (Winter, 1989, 1990b), and in ACLR subjects performing jumping maneuvers (Ernst et al., 2000). However, no study has investigated bilateral lower extremity joint accommodations during gait in ACL injured subjects prior to and 3 months following reconstructive surgery.

ACLD and ACLR Hip Asymmetry

Contrary to the CON group, no between-limb differences for hip positive work in the ACLD and ACLR groups were observed during NP stance (TABLE 5.2). As well, the correlation coefficient values for the ACLD and ACLR groups hip moment (r=0.89/0.94, respectively) and power (r=0.80/0.75, respectively) patterns were relatively higher compared to the bilateral CON moment (r=0.72) and power (r=0.42) values (TABLE 5.2; FIGURES 5.3, 5.6). These data suggest that the ACLD and ACLR groups demonstrated hip joint symmetry and that bilateral hip accommodations occurred, possibly in response to ACL injury and surgical repair. Berchuck (1990) reported that chronic ACLD subjects demonstrated a bilateral increase in hip

extensor moments compared to healthy adults during NP gait, and Kowalk et al. (1997) reported no significant differences in bilateral maximum hip joint moment or power production values during stair ascent as compared to controls.

ACLD and ACLR Knee Asymmetry

It is possible that symmetrical hip moment and power patterns were an adaptation to asymmetry exhibited in the ACLD/ACLR group knee moment and power patterns. In the present study, no significant between-limb differences in CON knee EAI were observed and bilateral CON knee moment ($r=0.96$) and power ($r=0.93$) curves were highly correlated (TABLE 5.2; FIGURES 5.2 & 5.5). However, in the ACLD and ACLR groups, moderate to high between-limb correlation coefficient values for knee moment and power patterns, but significant bilateral differences in EAI and work, were evident during NP stance (TABLE 5.2; FIGURE 5.2). The ACLDn and ACLRn knee produced significantly more EAI compared to the contralateral ACLDi and ACLRi knee (TABLE 5.2; FIGURE 5.2). Derrick et al. (1994) reported that high correlations indicate similarities in the temporal relationship between two curves. However, such correlations are not sensitive to amplitude differences. Therefore, the significant between-limb differences observed in the ACLD/ACLR knee EAI and positive work values could be interpreted as bilateral knee asymmetry. Furthermore, the ACLDn/ACLRn knee extensor moments during early NP stance are greater in magnitude than those in healthy adults (Allard et al., 1996; Eng & Winter, 1995; Kadaba et al., 1990; Sadeghi et al., 1997; Winter, 1987). Therefore, the results from this investigation suggest that the greater knee extensor moment and positive work demonstrated by the ACLDn/ACLRn knee was compensation for ACL injury and surgery possibly for the purpose of providing propulsion during gait.

Winter (1980) suggested that the overall moment of support revealed the tendency of the lower limb to push away from the ground and propel the body forward during gait. As well, it was previously discussed that changes in hip moment patterns are equally matched by alterations in knee moment patterns during gait. In FIGURES 5.2 and 5.3 it can be observed that during early NP stance, the greater ACLDn/ACLRn knee moment is matched by a reduced hip moment when compared to the contralateral ACLDi/ACLRi knee and hip. Furthermore, the reduced ACLDi/ACLRi knee moment is matched by a greater hip moment compared to the contralateral ACLDn/ACLRn knee and hip. Examination of TABLE 5.2 also demonstrates that there is little differences in the sum of the total EAI for the contralateral limbs or as compared to the CON group (ACLDi: 98.32 Nms; ACLDn: 101.76 Nms; ACLDn: 101.76 Nms; ACLRn: 100.88 Nms). These data suggest that the total moment of support was maintained regardless of differences in bilateral joint moment patterns and ACL injury. It is possible that the greater ACLDn/ACLRn knee produced greater EAI in an effort to maintain the moment of support and provide compensatory propulsion during stance.

Response to Unexpected Forward Perturbations During Gait

Control Group Symmetry

Bilateral lower extremity joint moments, powers, and angles for the CON group during FP stance were generally similar in pattern (r=0.74-0.98) and no significant between-limb differences were observed for any variable (TABLE 5.3). These data are in contrast to NP gait where asymmetrical hip moment and power patterns were observed. In Chapter II it was reported that the muscles surrounding the hip were found to be most important in maintaining control of the HAT segment and preventing collapse of the lower extremity as an initial response to the FP

(Ferber, 2001). These data suggest that, regardless of limb, reactive hip adjustments to unexpected perturbations result in symmetrical bilateral responses.

ACLD and ACLR Group Symmetry and Asymmetry

In Chapter IV, it was reported that there were no significant differences in hip moment values during FP stance between the CON, ACLD, and ACLR groups (Ferber, 2001). During FP in the present study, bilateral accommodations to injury and surgery can be observed in that the ACLD and ACLR groups exhibited symmetrical bilateral hip moment patterns (r=0.94 – 0.96) and no differences in bilateral hip EAI were observed (TABLE 5.3; FIGURE 5.12). These data suggest that, in response to an unexpected FP, reactive balance adjustments result in similar bilateral responses regardless of injury status.

During FP stance, the CON group demonstrated similar contralateral knee power patterns (r=0.84) while the ACLD (r=0.52) and ACLR (r=0.53) subjects exhibited asymmetry in knee power patterns (TABLE 5.3; FIGURE 5.14). However, the ACLD and ACLR subjects exhibited similar bilateral knee FP moment (0.88 – 0.91) and position (r=0.92 – 0.93) curve correlation coefficient values (TABLE 5.3). Since joint power is calculated as the product of joint moment and angular velocity, it is sensitive to small changes in either measure. It is possible that ACL injury and subsequent surgery resulted in knee joint instability that was compensated for by small changes in knee joint position and joint moment generation and that these small changes were observed as alterations in knee power patterns. Alterations in bilateral knee power patterns may be more critical during the FP condition for the purpose of preventing re-injury and vertical collapse.

Summary

The present study investigated bilateral symmetry in healthy subjects and chronic ACL injured subjects prior to and 3 months following reconstructive surgery. During NP gait, healthy adults demonstrated asymmetrical hip moment and power patterns whereas ACLD and ACLR subjects exhibited symmetrical hip but asymmetrical knee mechanics. In response to the FP, healthy adults exhibited lower extremity joint symmetry but the ACLD and ACLR group exhibited asymmetrical knee moment and power patterns. These findings suggest that ACL injury and surgery result in bilateral joint accommodations and that, when investigating ACL injured populations, bilateral control population data should be used in addition to non-injured limb data.

CHAPTER VI

SUMMARY AND RECOMMENDATIONS

It has been suggested that neuromuscular adaptations occur as a result of anterior cruciate ligament (ACL) injury and that these adaptations may depend on the time since injury and surgical repair. The ability of an ACL injured individual to react and maintain equilibrium in response to an unexpected gait perturbation is critical in prevention of reinjury. However, the underlying neurological and biomechanical process by which locomotion occurs is complex, especially when gait is disrupted or perturbed. The purpose of this dissertation was to investigate how normal gait patterns may change as a result of chronic ACL deficiency (ACLD) and subsequent surgical repair and to determine the effect of unexpected forward perturbations on these individuals compared to healthy controls. The intent was to further our understanding of adaptations that may occur as a result of chronic ACLD and surgical repair and to better understand lower extremity mechanics in response to an unexpected forward perturbation.

The first study characterized the typical lower extremity postural responses in healthy young adults when reacting to an unexpected forward perturbation occurring at heel strike. The purpose of this investigation was to determine the effect of unexpected forward perturbations during gait on lower extremity joint moment, power, and kinematics and muscle EMG patterns in

healthy subjects. The muscles surrounding the hip were found to be most important in maintaining control of the upper body and in the preventing collapse as an initial response to the forward perturbation. Distinct lower extremity joint moment and power patterns were observed in response to the perturbation but, similar to non-perturbed gait, an overall positive moment of support was maintained. It was therefore suggested that reactive balance control was a coordinated and synchronized effort of the lower extremity joints in an effort to maintain dynamic equilibrium during an unexpected forward perturbation.

The second study investigated how normal gait patterns may change as a result of chronic ACLD and to determine the effect of unexpected forward perturbations on chronic ACLD subjects as compared to healthy controls. It has been reported that chronic ACLD patients tend to develop a quadriceps avoidance gait pattern indicated by a sustained knee flexor moment throughout stance (Berchuck et al., 1990). However, other investigators have not demonstrated the quadriceps avoidance phenomenon in chronic ACLD patients (Roberts et al., 1999; Rudolph et al., 1998). The chronic ACLD subjects in the present investigation did not demonstrate a quadriceps avoidance gait pattern during non-perturbed gait or in response to the forward perturbation. However, the ACLD subjects appeared to accommodate to chronic ACL deficiency through alterations of hip and ankle joint kinematic, kinetic, and muscle power patterns during non-perturbed gait. In response to the same forward perturbation, the ACLD subjects demonstrated a greater knee extensor moment, in an effort to prevent vertical collapse and maintain balance, as compared to healthy controls.

The third study examined the same ACLD subjects 3 months following ACL reconstructive (ACLR) surgery. The purpose was to determine how normal gait patterns and the response to an unexpected forward perturbation may change as a result of surgery as compared to pre-surgical values and healthy controls. Three months following surgery, these same subjects

demonstrated a significantly different knee moment pattern and were significantly more flexed at the knee and hip during non-perturbed and perturbed gait compared to pre-surgical values and the control group. The ACLR group did, however, exhibit a hip moment pattern more characteristic of the control group but significantly different from pre-surgical ACLD 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 and that any re-establishment of pre-injury gait patterns takes longer than 3 months to occur.

The fourth study examined bilateral joint accommodations as a result of ACL injury and reconstructive surgery as compared to healthy subjects. During NP gait, healthy adults demonstrated asymmetrical hip moment and power patterns whereas ACLD and ACLR subjects exhibited symmetrical hip but asymmetrical knee mechanics. In response to the FP, healthy adults exhibited lower extremity joint symmetry but the ACLD and ACLR group exhibited asymmetrical knee moment and power patterns. These findings suggest that ACL injury and surgery result in bilateral joint accommodations and that when investigating ACL injured populations, bilateral control population data should be used in addition to non-injured subjects.

Strengths of the Study

This investigation has several strengths. First, group-related differences in postural responses reached not only statistical significance, but the mean power value for comparisons was 83%. Thomas et al. (1991) indicated that the greater the power, the more meaningful the group differences. This suggests the results of this investigation are meaningful and warrant clinical consideration.

Second, few studies have quantified reactive gait alterations due to unexpected gait perturbations and, of those performed, only electromyographic and kinematic information has been presented. This investigation was the first to calculate joint moments and powers associated with an unexpected forward perturbation and, as such, these data provide a more comprehensive understanding of how each of the lower extremity joints contributes to dynamic balance control when gait is perturbed.

Third, there is controversy surrounding the development of a quadriceps avoidance gait pattern in chronic ACLD subjects. If a quadriceps avoidance pattern were to develop, the ACLD knee would contribute little, if any, to the vertical support of the body throughout stance, and the hip and ankle joint must compensate to prevent collapse. The development of a quadriceps avoidance gait pattern could be considered to indicate a pathological gait pattern. The results of this investigation suggest that development of a quadriceps avoidance gait pattern may be less common than previously reported.

Fourth, investigations involving ACLR subjects are limited and suggest that time since surgery plays an important role in the return of normal gait patterns (Bulgheroni et al., 1997; Bush-Joseph et al., 2001; Cicotti et al., 1994; Devita et al., 1998; Ernst et al., 2000; Timoney et al., 1993). However, time between injury and surgery differed significantly in these investigations. Data from this study 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.

Lastly, few studies have investigated possible bilateral adaptations during gait as a result of ACL injury and surgical repair. Data from this investigation suggest that ACL injury and surgery result in bilateral joint accommodations and that, when investigating ACL injured populations, bilateral control population data should be used in addition to non-injured limb data.

These data are especially important from a clinical perspective since data from the non-injured contralateral limb are often used as the criteria for determining when an ACL injured individual has re-established pre-injury measures.

Limitations of the Study

The first concern regarding the experimental paradigm involves the artificial nature of the perturbation itself. A realistic slip-perturbation is a kinematic event marked by the loss of frictional contact between the foot and the ground. The perturbation used in this study was a kinetic event and, as such, was due to an external force being applied to the subject. This type of design was due to the constraints in the experimental setup. It is therefore difficult to compare the results of this investigation to a realistic slip paradigm.

Second, Bothner et al. (2001) investigated standing postural responses to backward perturbations and reported that platform deceleration has a quantifiable impact on lower extremity postural responses particularly at the knee and ankle joints. Bothner (2001) decomposed the net joint moment data into relative contributions from the force plate itself and those due to muscular force generation. Since the data provided in this experiment are simply the net joint moment, it is difficult to discern the influence of the force plate movement on reactive balance adjustments. Therefore, the joint moment data should be further decomposed to discern postural adjustments due to force plate movement and those due to muscular force generation.

Third, EMG data for the non-perturbed condition were normalized to the maximum within-trial amplitude and, therefore, EMG amplitude relative to the CON, ACLD, and ACLR groups was not available. With this normalization method, one is limited to discussing EMG pattern characteristics. Since several significant between-group joint moment differences were

observed, corroborating EMG information would be useful. In the same respect, EMG data were collected using surface electrodes. Given the numerous muscles in the human body, analysis of the selected muscles alone is not sufficiently comprehensive to observe the contribution of all muscles that produce joint moments.

Fourth, it has been demonstrated that errors in marker placement (Stagni et al., 2000) and alignment of center of pressure and foot coordinates (McCaw & Devita, 1995) can significantly affect lower extremity joint moment calculations. Joint moment data from this investigation should therefore be considered as approximations of true values.

Fifth, since data were only collected 3 months following surgery, it is difficult to speculate about when the ACLR subjects would have regained pre-injury gait characteristics. Data 6 months following surgical reconstruction would have been useful to answer questions regarding re-establishment of pre-injury gait characteristics and adaptation to reconstructive surgery.

Finally, biomechanical adaptations to chronic ACLD and following reconstructive surgery depend on several factors including patient compliance with rehabilitation protocols, surgical procedure, and patient characteristics. The present study was limited to physically active subjects who had suffered an ACL rupture 2 or more years prior to testing. All subjects had suffered at least one episode of giving-way and, thus, had decided to undergo reconstructive surgery. The surgical procedure consisted of an arthroscopically assisted, endoscopic, bone-patellar tendon-bone reconstruction using the central one-third of the patellar tendon. The present results may only apply to ACL-injured individuals with identical or, at least, similar characteristics.

Recommendations for Future Research

Previous investigations have demonstrated that acutely injured ACL subjects exhibit a sustained knee extensor moment compared to non-injured control subjects (Devita et al., 1997). However, the results from the present investigation suggest that ACLD subjects, given time, may re-establish knee moment patterns similar to pre-injury characteristic and uninjured subjects. A follow-up study that documents gait mechanics from soon after ACL injury until 1-2 years post-injury would provide a better understanding of the injury recovery process.

While a longitudinal study would lend important information, it is time consuming and suffers from possible subject attrition. Therefore, a study involving ACLD subjects who were at different stages of ACL injury recovery would also be useful. Wexler et al. (1998) investigated 30 ACLD patients divided into three groups according to time between injury and testing: 0 to 2.5 years (early), 2.5 to 7.5 years (intermediate), and greater than 7.5 years (chronic). In this fashion, time-related adaptations to ACLD using a cross-sectional approach can be better determined.

Third, ACL reconstructive surgery has been demonstrated to significantly alter lower extremity joint mechanics regardless of time between injury and surgery. However, a future study that investigated the ACLR subjects 4 weeks, 12 weeks, and 36 weeks after surgery would better determine time-related adaptations to ACL surgery. However, as was demonstrated in the present investigation, time between injury and surgery would have to be accounted for.

APPENDIX A

INFORMED CONSENT FORM

Comparison of perturbation response between anterior cruciate ligament deficient and uninjured individuals

You are invited to participate in a research study conducted by Reed Ferber, a Doctoral student at the University of Oregon in the Department of Exercise and Movement Science. The investigator hopes to determine whether or not subjects with anterior cruciate ligament (ACL) injuries have delayed muscle onsets and alter their gait kinematics compared to individuals with uninjured knees under simulated slip conditions. You have been selected as a possible participant because you have either a unilateral ACL deficiency, have undergone a surgical repair and your physician has permitted your participation, or you have no knee dysfunction. The results will contribute to a better understanding of the effects of ACL injury on balance control during walking.

The experiment will be held in the Motor Control Lab of the University of Oregon. In this experiment, you will be asked to perform multiple walking trials across a platform into which two movable platforms have been placed. On random trials, one of the platforms will move forward at 10 centimeters per second as you step onto it. You will not be informed in advance whether or not the plate will move. Your task will be to maintain your balance while walking across the walkway. Your response to the perturbations will be recorded using small disc sensors placed on the surface of the skin. Signals from these sensors will be recorded into a computer for analysis. Video record of your trials will also be used. Small reflective markers will be placed over some of your joints to identify joint movement on the video record. You will be asked to wear shorts and a sleeveless shirt so that the markers can be videotaped clearly. You will also be asked to wear the harness we provide to prevent falling.

There will be a total of 48 walking trials. The first twelve will be without platform movement. The following 36 will consist of a number of trials in which one of the platforms may move. A rest period of approximately 5 minutes will be provided after every twelve trials. Longer rest periods can be taken as desired. This walking experiment will last approximately one and a half hours.

There is minimal risk that you may fall when your balance is perturbed. Using a relatively mild perturbation speed and displacement minimizes the risk. The risk is also minimized by having you wear a harness and by providing a handrail to grasp all along the walkway. The harness will prevent you from falling to the floor in the case that you should slip.

To reduce the risk of skin irritation to the applied sensors, hypoallergenic gel and tape will be used. Incidence of skin response to the tape or gel is low or non-existent. Also, you may get tired or uncomfortable during some of the tasks. To minimize this, the test will be paused or stopped at your request.

So that you remain anonymous in our files, all data will be coded with letters and numbers and kept locked in the principal investigator's office. All data and videos will be destroyed five years after the completion of the project. Your name will not appear in the investigator's files.

Coding is done to keep subject names anonymous. Information obtained in connection with this study that can be identified with you will remain confidential and will be disclosed only with your permission. We may wish to use the video tape recording of your movement for educational purposes in the future.

Your identity will not be disclosed. If you would like to give your permission for use of the video recording for educational purposes (such as classes or conferences), please place your initials by "yes" below. If you do not wish to give us permission at this time, please initial by "no". Video recordings will not be taken for any commercial use.

yes _____ no _____

As your permission is voluntary, your decision will not affect your relation with the Motor Control Lab or the Orthopaedic and Fracture Clinic. If you decided to participate, you are free to withdraw your consent and discontinue participation at any time without penalty.

You or your insurer are responsible for any medical expenses resulting from injuries to you caused by you participation in this research project. If you are a UO student or employee covered by a UO medical plan, the terms of that plan may apply to any such injuries. Should you suffer injury as a result of participating in this project, you would be free to file a claim against the State of Oregon pursuant to ORS 30.260-.275. Questions regarding claims should be directed to the Assistant to the President for Legal Affairs (541)346-3843, University of Oregon, Eugene, OR 97403. Any such incidents should also be reported to the Committee for the Protection of Human Subjects (541)346-2510 at the same address. If the Project or the University were to be legally at fault and liable, the largest possible recovery would be $200,000 to any claimant and $500,000 to all claimants for any single incident.

If you have any questions, please feel free to contact Reed Ferber at (541) 346-1033 or his faculty advisor Louis R. Osternig, Ph.D. at (541) 346-3384. If you have any questions regarding your rights as a research subject, contact Human Subjects Compliance, University of Oregon, Eugene, OR 97403, (541) 346-2510. You will be given a copy of this form to keep for your files.

Your signature indicates that you have read and understand the information provided above, that you willingly agree to participate, that you may withdraw your consent at any time and discontinue participation without penalty, that you will receive a copy of this form, and that you are not waiving any legal claims, rights, or remedies.

Participant Signature _____ Date _____

Witness Signature _____ Date _____

Primary Investigator Signature _____ Date _____

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