MUSCLE FUNCTION AND QUALITY AFTER ANTERIOR CRUCIATE LIGAMENT (ACL) RECONSTRUCTION

By

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A THESIS PRESENTED TO THE GRADUATE SCHOOL
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This document is dedicated to my family and friends.

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The anterior cruciate ligament (ACL) is an important dynamic stabilizer of the knee. The incidence of ACL rupture and surgical reconstruction is high in the United States. Whether knee extensor strength and voluntary activation are hindered after reconstruction is debated. Thus the purpose of our study was to evaluate bilateral knee-extensor strength in individuals with unilateral ACL reconstruction (ACLR); and to examine voluntary activation of the quadriceps femoris in individuals who have received ACLR as compared with healthy controls. Muscle quality via isokinetic strength testing (180°/s and 60°/s) of the quadriceps musculature, quadriceps voluntary activation, and thigh circumference were assessed. Central activation ratio (CAR) calculated via twitch interpolation was used to determine voluntary activation deficits in the quadriceps. Measurements of 24 college-age unilateral ACLR individuals and 23 healthy participants were evaluated. Thirteen females (age 20.2 $\pm$ 1.1 years, height 162.5 $\pm$ 5.7 cm, weight 592.5 $\pm$ 61.4 N, years post-surgery 2.5 $\pm$ 1.5 years) and 11 males (21.3 $\pm$ 2.5 years,

177.9 ± 7.4 cm, 772.0 ± 112.1 N, years post-surgery 3.3 ± 1.8 years) participated in the ACLR group. Eleven females (age 21.5 ± .82 years, height 165.5 ± 4.8 cm, weight 606.2 ± 114.1 N) and 12 males (21.8 ± 1.1 years, 179.3 ± 4.9 cm, 855.9 ± 146.9 N) were in the control group. A knee-extensor strength deficit as well as a lower CAR of the quadriceps was found in the ACLR limb compared to the contralateral limb. No difference in voluntary activation was revealed among the ACLR limb, healthy limb, and control limb. In addition, no difference in thigh circumference existed between the ACLR and contralateral limb. Therefore, the strength deficits found in the ACLR leg are attributable to lower voluntary activation compared to the contralateral leg, given that no difference was found in thigh circumference between legs. Further research is needed to conclude whether isokinetic strength is a predictor of re-injury; and to examine the underlying mechanism central inhibition and neural drive to the quadriceps femoris. Clinicians should consider the deficits in muscle quality when returning the patient to a pre-injury activity level.

CHAPTER 1
INTRODUCTION

The anterior cruciate ligament provides stability to the tibiofemoral joint mainly by resisting anterior translation of the tibia on the femur. A high incidence of anterior cruciate ligament (ACL) rupture is evident each year, and a great deal of investigation has been devoted to injury mechanism and associated risk factors of an ACL injury. ACL rupture can occur as a result of a contact mechanism, often placing the knee under valgus stress; or by a noncontact mechanism in which a sudden deceleration, cutting maneuver, or improper landing takes place. Intrinsic and extrinsic factors that predispose an individual to ACL injury have been identified. Intrinsic factors (which cannot be changed) include lower-extremity malalignment, a smaller ACL, physiological laxity, increased quadriceps angle, increased pelvic width, tibial rotation, foot alignment and hormonal influence. Proposed extrinsic factors include improper landing mechanics, muscular imbalances, neuromuscular recruitment patterns, flexibility, shoe-surface interface, and field conditions.

Surgical repair after ACL rupture is typical for the general public and imperative for athletes to return to high-level competition. Rehabilitation (whether accelerated or conventional) focuses on range of motion, strength, neuromuscular control, and functional activity progression in order to achieve preinjury activity level. Return-to-activity criteria are generally based on comparing the ACL reconstructed knee to the uninvolved side. Different rehabilitation philosophies have recently been challenged, especially whether early weightbearing exercise is advantageous or detrimental to the

ACL graft. Even some time after the ACL has been repaired and a rehabilitation program completed, thigh musculature atrophy along with proprioception, voluntary activation, and knee extensor strength deficits may be encountered. Strength and muscle-quality deficits are apparent in some clinical populations, although it is unclear in ACLR individuals. Isokinetic testing and muscle quality via twitch interpolation are commonly used to assess strength and voluntary activation; and both techniques allow for reliable, quantitative measures. The reviewed studies solely evaluate strength or voluntary activation.

Given that the investigations to date are not comprehensive, the purpose of our study was to perform a thorough evaluation of knee-extensor function in individuals with unilateral ACLR. More specifically, muscle quality via isokinetic strength testing (180°/s and 60°/s) of the quadriceps musculature, quadriceps femoris voluntary activation, and thigh circumference were assessed. Central activation ratio (CAR) calculated via twitch interpolation was used to determine voluntary activation deficits in the quadriceps. Our findings may prove valuable to clinicians evaluating progress and directing or redirecting rehabilitation. In addition, the results may allow the clinician to predict the probability of future functional knee instability or aid in prevention of re-injury based on deficits in muscle quality.

CHAPTER 2
REVIEW OF LITERATURE

General Introduction

The anterior cruciate ligament (ACL) provides static stability of the tibiofemoral joint, resisting anterior translation of the tibia on the femur, internal and external rotation of the tibia on the femur, and hyperextension. The ACL also plays an important role in dynamic knee stability, preventing anterior translation of the tibia relative to the femur while non-weightbearing (Bach & Hull, 1998; Fleming et al. 2001). The ACL is a commonly injured ligament of the knee (Boden et al. 2000; Johnson, 1983). Typical mechanisms for ACL injury include direct contact (usually as a result of a valgus force), or noncontact mechanisms (including sudden deceleration, cutting maneuvers involving a quick change of direction, or landing improperly). Not surprisingly, ACL injuries are common in soccer, football, and basketball: these sports require repeated decelerating and cutting maneuvers. Of the common mechanisms, non-contact injuries reportedly make up 70% or more of ACL injuries (Boden et al. 2000; Griffin et al. 2000; McNair et al. 1990). Because of the high number of ACL tears that occur each year, risk factors associated with ACL injury have been studied extensively over the past decade (Beckett et al. 1992; Griffin et al. 2000; Johnson, 1983; Kaufman et al. 1999; Loudon et al. 1996; Woodford-Rogers et al. 1994). Several potential risk factors have been identified. Some investigatorsindicate that foot structure (mainly hyperpronation of the subtalar joint) exposes the ACL to disadvantageous biomechanical patterns during gait (DeLacerda, 1980; Donatelli, 1996). DeLacerda (1980) concluded that abnormal pronation of the

subtalar joint in the stance phase of gait was a contributing factor in overuse injuries of the lower extremity. Loudon et al. (1996) reported that factors of knee recurvatum, excessive navicular drop, and excessive subtalar joint pronation were significant discriminators of an ACL-injured group compared to a noninjured group.

Interestingly, ACL injury rates between males and females are quite different. Epidemiological data indicate that the incidence of ACL injury is substantially higher in females compared to their male counterparts, (4-to 6-fold higher, in some studies) (Arendt & Dick, 1995; Gwinn et al. 2000; Hutchinson & Ireland, 1995; Lindenfeld et al. 1994). Etiology of the gender difference in injury rate was the focus of numerous studies. A multifactorial solution dependent on intrinsic and extrinsic factors is the most plausible explanation for higher injury rates in females. Although the quest to prevent ACL injuries continues, 1 in every 3,000 people in the general population rupture their ACL in the United States each year (Miyaskaka et al. 1991). In athletics, the incidence of ACL rupture is 3 of 100 athletes over the course of the season (McCarroll et al. 1995). Approximately 175,000 ACL reconstructions are performed annually and the associated cost of surgery is over 2 billion dollars (Gottlob et al. 1999). Accordingly, rehabilitation techniques and return-to-play criterion have evolved. Examining the mechanisms of ACL injury, intrinsic and extrinsic factors related to injury, rehabilitation, the use of isokinetics, and interpolated twitch as a measure of muscle quality should facilitate a comprehensive preview of post surgery outcomes in a population of ACL reconstructed individuals. Though these factors have been examined independently in several studies, a simultaneous and thorough evaluation on the same group of individuals has not been performed.

Mechanisms of Injury

The ACL becomes vulnerable with excessive anterior tibial translation or rotation of the femur on the tibia, especially when the knee is close to full extension (Boden et al. 2000; Kirkendall & Garrett, 2000). McNair et al. (1990) reported that the non-contact ACL injury mechanism usually involved a slight knee flexion angle accompanied by excessive internal rotation of the tibia at the instant of foot strike. Under this condition, the extensor mechanism (which uses the quadriceps to produce a large eccentric force) places the knee in a vulnerable position (Boden et al. 2000). Large anterior shear forces are placed on the proximal tibia, especially at low knee-flexion angles (<30°) in which the patellar tendon/tibia angle is largest (Boden et al. 2000). This patellar tendon/tibia angle is reportedly larger in women, indicating greater shear stress of the knee (Nisell, 1985).

Biomechanics of ACL Rupture

The ACL (providing ligamentous constraint to stabilize the tibiofemoral joint) runs from the anterior intercondylar region of the tibia through the intercondylar notch of the femur, and attaches on the posteromedial aspect of the lateral femoral condyle. The bands extend posterior, superior, and lateral as it runs through the intercondylar notch. The two distinct bundles of the ACL are the anteromedial and posterior medial bundle. Because of the different attachment sites of the two bands, in full extension, the posterolateral band is taut; and in full flexion, the anteromedial band is taut. The anteromedial band of the ACL was found to be under increased strain as the knee transitions from a non-weightbearing to a weightbearing position (Fleming et al. 2001). Anterior shear and internal torque on the tibia combined with compressive force produced by body weight strained these fibers as well (Fleming et al. 2001). Zavatsky &

Wright (2001) evaluated ACL injury mechanisms and the corresponding knee flexion angles at which ruptures occur. These researchers constructed a sagittal-plane knee model in which a critical strain criterion was used to model the onset and progression of ACL rupture (Zavatsky & Wright, 2001). Zavatsky & Wright (2001) found that at low knee flexion angles (<20°) the shorter posterior ACL fibers rupture first followed by the anterior fibers due to excessive anterior tibial translation. However at higher flexion angles, the longer anterior fibers of the ACL would rupture first then progressing to the posterior fibers.

Intrinsic Factors Related to ACL Injury

Several researchers have proposed predisposing intrinsic and extrinsic risk factors of ACL injury based on anatomical, neurological, and muscular characteristics. Intrinsic factors associated with ACL injury include structural differences such as malalignment of the lower extremity, a narrowed intercondylar notch, a smaller ACL, physiological laxity, increased quadriceps angle, increased pelvic width, tibial rotation, foot alignment and hormonal influence (Arendt & Dick, 1995; Hutchinson & Ireland, 1995). Femoral notch height, width, height to width ratio and overall shape have been considered to contribute to incidence of ACL injury (Tillman et al. 2002; Shelbourne et al. 1995). A smaller A-shaped notch may not pinch a normal sized ACL, however congenitally smaller ACL bands may result (Ireland, 1994). The quadriceps femoral angle (Q-angle) has also been considered in the potentiality of ACL injury. The Q-angle is characterized as the acute angle between the line of the anterior superior iliac spine and midpoint of the patella and the line connecting the midpoint of the patella and to the tibial tuberosity. Greater Q-angles are frequently seen in females and have been concluded to produce medial stress as the quadriceps pulls laterally on the patella (Shambaugh et al. 1991). In addition,

Loudon et al. (1996) found genu recurvatum, excessive navicular drop, and excessive subtalar joint pronation to be significant discriminators between female ACL injured participants and non-injured participants. Boden et al. (2000) evaluated hamstring flexibility and measured genu recurvatum in addition to assessing the mechanism of injury in ACL injured individuals. Both flexibility and genu recurvatum measures were significantly greater in the ACL group compared to matched healthy control participants. The hamstring muscles utilize both passive and active properties to produce a posterior force on the proximal tibia when activated to counteract anterior translation, therefore acting as a dynamic stabilizer of the ACL (Boden et al. 2000). The authors concluded that the hamstrings contribute to passively protect the ACL might be reduced in patients with above-average flexibility because the ACL group had greater hamstring laxity. Hormonal fluctuations may also play a role. More specifically, estrogen, progesterone, relaxin and estrodial are also hypothesized to have effects on muscle function and tendon and ligament strength (Sarwar et al. 1996). Although the examination of intrinsic factors may help to identify the causes of ACL injury, intrinsic risk factors are not modifiable. Therefore, interventions cannot be performed to reduce the influence of intrinsic factors. An examination of modifiable (extrinsic) factors seems a more logical approach.

Extrinsic Factors Related to ACL Injury

Extrinsic factors related to ACL injury include improper landing mechanics, muscular imbalances, neuromuscular recruitment patterns, flexibility, shoe surface interface, and playing surface (Huston et al. 2000; Arendt & Dick, 1995). Extrinsic factors that may contribute to the higher female injury rate include baseline level of conditioning and coordination, decreased muscle strength normalized for body weight compared to males, neuromuscular differences, knee stiffness, landing technique, and

postural control (Huston & Wojtys, 1996; Hewett, 2000). Regarding muscular coordination, electromyographic power spectra analysis performed by White et al. (2003) revealed that females exhibited increased quadriceps to hamstrings coactivation compared to matched male subjects. White et al. (2003) suggested the increased quadriceps coactivation might generate greater anterior tibial load in dynamic conditions. Huston & Wojtys (1996) reported different muscle recruitment patterns in which females tended to first contract their quadriceps when responding to anterior tibial translation. Conversely, males have the tendency to recruit the hamstring muscles first.

Hamstring recruitment acts to decrease the load on the ACL, providing better protection as a result (Huston et al., 2000). Fortunately, neuromuscular firing patterns can be trained, correcting the quadriceps dominant technique used by female athletes. Hewett et al. (1996) found a significant increase in hamstring torque and correction of hamstring strength imbalances with neuromuscular training including plyometrics, stretching and strengthening in female athletes. In addition, Huston et al. (2000) investigated knee stiffness (which can add to joint stability) to determine if gender differences existed. Knee stiffness is dependent on the number of active actin-myosin cross-bridges along with the excitation of the muscle to protect the ligament from excessive strain (Huston et al. 2000). Males were able to increase active knee stiffness 4-fold compared to the twofold increase presented by females (Wojtys et al. 1998).

Many non-contact ACL injuries occur upon landing from a jump, therefore evaluating landing characteristics has been the primary focus of several researchers. Hewett et al. (1996) found notable differences in the kinematics of landing characteristics between genders. Hewett et al. (2002) concluded that female athletes rely on ground

reaction forces to direct muscle contraction characterizing females as ligament dominant instead of being muscle dominant, as males tend to be for joint control strategies. Optimal landing characteristics are geared to reduce compressive loads. Better landing mechanics accompanied by decreased landing peak force, increased knee flexion angle at landing, and decreased abduction and adduction moments were achieved after implementing a six week program of jump training in the female athletes which may contribute to lower injury rates. Hewett and colleagues (1996) found during a follow-up study on knee injury rates after the program. Despite the ability to improve performance by manipulating extrinsic factors, injuries are inevitable and are often followed by rehabilitation in order to return the injured individual to their normal level of activity.

Rehabilitation

Several approaches for rehabilitation after ACL injury and reconstruction have been proposed, all of which are geared for the patient to reestablish a pre-injury activity level. During weightbearing conditions, compressive forces about the knee are indicated to reduce anterior-posterior laxity and contribute to joint stiffness compared to a non-weightbearing condition (Fleming et al. 2001; Torzilli et al. 1994). Based on this finding, using weightbearing conditions in rehabilitation would seem to be the optimal approach to regain knee stability and function. Shelbourne and Nitz (1990) proposed an accelerated rehabilitation program to minimize muscle atrophy and quickly regain knee function. The authors' premise was that physical therapy utilizes weightbearing therapeutic exercise based on the suggestion that open kinetic chain tasks imposes greater amounts anterior shear stress upon the ACL (Shelbourne & Nitz, 1990). However, Fleming et al. (2001) challenges this rehabilitation paradigm. Fleming et al. (2001) examined the function of the ACL in vivo during non-weightbearing and weightbearing

conditions by applying external loads about the knee. ACL strain was measured with an implanted differential variable reluctance transducer and a knee-loading fixture with 6 degrees of freedom to apply the anterior and posterior shear forces, internal and external rotational torques, and varus and valgus moments. The authors reported that the application of internal rotational torques and anterior shear forces at the knee resulted in ACL strain in the non-weightbearing conditions and increased strain was experienced during the transition from non-weightbearing to weightbearing conditions. Externally applied torques and varus-valgus moments strained the ACL during weightbearing conditions. Strain of the ACL increased significantly during transition between the weightbearing and non-weightbearing condition. Fleming and colleagues suggested that weightbearing exercise in rehabilitation of ACLR does not protect the ACL graft from strain. The authors proposed that the significant increase in ACL strain during transition is due to the location of the compressive force vector applied and anterior tibial shift due to an inclined tibial plateau. The compressive force was located between the ankle and hip, producing a compressive force vector posterior to the knee and eliciting an extensor moment. The quadriceps knee musculature responds to counterbalance this posterior force. The second theory proposed by the authors was that the anterior shift occurring close to full extension was due to a posteriorly inclined tibial plateau causing the tibia to slide anteriorly (Fleming et al. 2001; Torzilli et al. 1994).

Thigh muscular atrophy and associated strength deficits have been frequently reported in the literature after ACL rupture and reconstruction. Hurley et al. (1997) evaluated the effects of joint damage on muscle function, proprioception, and rehabilitation; and reported that thigh musculature strength and knee stability were

reported to decrease following ACL rupture. Kobayashi et al. (2004) found that knee flexor concentric strength of the involved knee after reconstruction reached 90% of uninvolved limb at 6 months after surgery, whereas quadriceps strength of the involved limb took longer to reach the same level compared to the uninvolved limb. The extensor concentric strength did not reach 90% strength of the uninvolved limb until one year after surgery (Kobayashi et al. 2004). Osteras et al. (1998) also evaluated isokinetic knee muscle strength 6 months after ACL reconstruction and similar results were found. About 82% of the ACLR knees reached 90% of knee flexor strength of the uninvolved knee, whereas only 12% of the ACL knees fulfilled the recommended quadriceps strength parameter for return-to-activity. Wilk et al. (1994) reported only 16% of the subjects in the ACLR group reached 90% of the quadriceps isokinetic strength compared to the contralateral limb. This quadriceps lag revealed by these investigators should be considered in the release of an athlete to full participation in activities. Ciccotti et al. (1994) demonstrated the importance of post surgical rehabilitation of ACL rupture focusing on vastus lateralis, bicep femoris, and tibialis anterior training in order to increase muscle activity along with coordinated responses quadriceps and hamstring during functional activities. Coombs & Cochrane (2001) evaluated isokinetic knee flexor strength in ACLR patients who underwent repair using doubled semitendonosis and gracilis grafts. Average eccentric flexion peak torque was significantly less in the involved limb compared to the uninvolved side at 3, 6, and 12 months after surgery. A deficit remained for knee flexion strength, more specifically eccentric and concentric average peak torque remained less in the involved limb compared to the uninvolved limb even at 12 months after surgery. Mattacola et al. (2002) reported negligible differences

between ACLR and age and activity matched controls for stability index and peak flexion torque. However, significant differences were found between the involved ACLR participants and matched involved limb controls for peak extension torque. The ACLR participants produced significantly less extension torque compared to the controls. Furthermore, side-to-side differences were found in which the involved ACLR limb produced significantly less extension torque than the uninvolved limb.

Anderson et al. (2002) examined recovery of concentric and eccentric strength before and after ACL reconstruction via isokinetic testing. Patients underwent similar rehabilitation protocols, in which the subjects had full range of motion by month 4 and were released to full activity 4-6 months postoperatively. Torque continued to increase considerably 6 months after the surgery and up to a year in both ACL reconstructed knees with patellar tendon grafts and reconstructions using hamstring tendon grafts. Muscle function of the quadriceps and hamstrings improved during recovery in both the reconstructed and uninvolved limbs; furthermore graft type had no effect on recovery. The quadriceps progressed the slowest in the ACL reconstructed knee, only reaching 83% relative torque compared to the uninjured limb when measurements were taken one year after surgery. Keays et al. (2003) evaluated the relationship between knee isokinetic strength and functional stability before and after ACL reconstruction in which semitendonosis and gracilis tendons were used in the repair. Similar to Anderson et al. (2002), all subjects followed a uniform rehabilitation protocol in the study. Significant positive correlations between quadriceps strength indices and functional stability were evident both before and after surgery. However, no correlations were detected at significant levels for hamstring strength and functional stability. Given that the

hamstrings play a more important role in decelerating tibial translation, muscular reaction time and motor unit recruitment may be better muscle parameters to relate to knee functional stability (Kaeys et al. 2003). Side-to-side differences were significant in postoperative measures; both quadriceps and hamstrings muscle groups had greater strength in the uninvolved limb compared to the reconstructed side.

Rehabilitation concerns after ACL reconstruction are focused on regaining thigh musculature strength and knee stability compared to the uninjured limb. Hiemstra et al. (2000) observed knee extensor strength deficits in an ACLR group at least one year after surgery using isokinetic testing compared to age and activity matched healthy controls. Therefore, clinicians should be aggressive in training proprioception and strength once the strength of the graft is sufficient. Ernst et al. (2000) investigated knee extensor strength using functional tasks as opposed to non-weightbearing isokinetic strength testing. Ernst et al. (2000) found knee extensor moment deficits by evaluating single leg vertical jumps and lateral step-ups via motion analysis and force platform system technology in ACLR patients and matched healthy controls. Hip and ankle compensations were suggested to take place due to knee strength deficits during vertical jump landing.

Criticism of isokinetic testing due to its non-functional application has been made based on the claim that this form of testing does not reflect actual sport specific motion. Sport movements often exceed the maximum speed of the dynamometer and in lower extremity dominant sports a weightbearing exercise would a be more functional testing position than the isokinetic non-weightbearing position. However, isokinetics remain valuable objective measures for isolated muscle groups.

Isokinetics in Rehabilitation

Isokinetics are widely used by therapists to evaluate and strengthen both healthy and injured patients. Hislop & Perrine (1967) introduced the term isokinetic exercise, as they investigated muscle force with the use of a dynamometer. Hislop & Perrine (1967) characterized muscle performance as a function of force, work, power, and endurance. The force output of a muscle and the torque generated by a muscle (usually referred to as strength) are the function of the tension created by the contracting muscle (Hislop & Perrine, 1967). During isokinetic exercise, the velocity of the movement is controlled, while the muscle maintains a state of maximum contraction throughout the entire range of motion.

The objective of isokinetic exercise is to mechanically apply resistance that matches the maximal muscle loading throughout full range of motion, even at biomechanically disadvantaged positions (Thistle et al. 1967). The advantage of isokinetic exercise over isotonic exercise is clear; the load utilized during isotonic exercise cannot exceed the maximum load of the contracting muscle's weakest angular position. Isokinetic exercise employs the concept of accommodating resistance; the isokinetic dynamometer matches the maximal force produced by the involved muscle throughout the range of motion. The ability to isolate the joint during movement is another advantage of isokinetic exercise. Kannus (1994) concluded that isokinetics are useful (with proper education and strict adherence to the test instructions) to document the progress of muscular rehabilitation and studying dynamic muscle function. The movements associated with isokinetics are not close to actual human performance tasks given that the actual motion exceeds the fastest available testing speeds of the dynamometer. In addition, the isokinetic training effect is specific to that type of

movement and there would not be an associated crossover effect to functional movements (Kannus, 1994).

Despite the limitations, clinicians often use isokinetic strength exercises in rehabilitation of lower extremity injuries. In addition, return-to-activity criteria are commonly based on the strength exhibited by the injured leg compared to that of the uninjured leg. This would be a valid standard assuming no strength differences exist between healthy limbs before injury. Few bilateral differences in lower extremity strength exist in most sedentary individuals or athletes participating in bilaterally symmetrical lower extremity activities. For example, soccer athletes usually have tendencies to use one leg more than the other for dribbling or shooting. As a result, soccer can be characterized as an asymmetrical lower extremity activity. If bilateral strength differences exist, then appropriate adjustments should be made for return to activity standards especially for one side foot-dominant sports.

Rothstein et al. (1987) found that knee extension and flexion peak torque, work and power measurements can be reliably obtained via an isokinetic dynamometer. Chow et al. (1997) similarly concluded that isokinetic dynamometry is a valuable resource for clinicians as long as the limitations of the machine are taken into consideration. These limitations include a) torque "overshoot" and "oscillation" before constant angular velocity is reached, b) a decrease in the duration of constant angular velocity occurs as the preset angular velocity increases, c) errors in torque measurement can occur without correcting gravitational and inertial effects, and d) inconsistencies among the research dealing with the reliability of strength data collected between different machines, inter-day and with-in day testing (Chow et al. 1997). Pincivero et al. (1997) concluded that

isokinetic testing at specific speeds were highly reliable when testing isokinetic strength and muscular endurance for the quadriceps and hamstrings.

Based on the reliability of isokinetic strength testing, several researchers have used the testing procedure to evaluate the quadriceps and hamstring muscles in healthy and clinical populations. Strength differences are observed in certain athletic populations (Mognoni et al. 1994; Oberg et al. 1986; Zakas et al. 1995). Mognoni et al. (1994) examined isokinetic knee and hip torques in young (16-18 years age) soccer players and found that knee extensor torques were higher in the nondominant limb at 60, 180, 240, and 300°/sec ($p<.05$). Oberg et al. (1986) found that male soccer players possessed statistically higher torque levels compared to their male nonsoccer player counterparts. No differences were found between contralateral muscle groups in dominant and nondominant legs in any of the test groups, nor was a difference between the supporting and nonsupporting legs in soccer players found. Zakas et al. (1995) measured isokinetic peak torques at 60 and 180°/s among basketball and soccer players of different divisions given that the sports require different training and playing techniques. Relative to body weight, no differences were detected for hamstring and quadriceps muscle strength or hamstring to quadriceps strength ratios within the different basketball and soccer divisions. Yoon et al. (1991) were unable to find strength differences between limbs during isokinetic testing in healthy young adults. Kannus (1988) measured peak and total-work ratios of hamstring and quadriceps muscles in a group of ACL insufficient knees using isokinetic testing at 60 and 180°/s. The injured limb of all participants had a significantly higher hamstring to quadriceps ratio in each test compared to the healthy limb. Natri et al. (1996) measured peak torques isokinetically at speeds of 60 and 180°/s

and peak work at 180°/s in a group of ACLR patients. A significant deficiency in thigh strength (especially in extension) was evident in the involved limb; furthermore the deficit was larger at the slower testing speed. Calmels et al. (1978) found no significant differences between right or left sides during isokinetic testing in 158 healthy paarticipants. Similarly, Maupas et al. (2002) reported no significant differences between the left and right side among 40 healthy male and female participants. However, isokinetic peak torque values were influenced by gender and speed of the motion. Males had significantly greater peak torque values compared to their female counterparts; at faster speeds of the concentric mode, muscle strength decreased. Overall, isokinetic peak torque values are influenced by age, sex, test position, angular velocity, and gravity effect torque (Maupas et al. 2002; Miyashita & Kanehisa, 1979).

Knapik et al. (1983) and Yoon et al. (1991) found that maximal torque occurred later in the range of motion as the angular velocity increased during knee-flexion efforts. Results of Yoon et al. (1991) were consistent with previous studies; the point at which peak torque occurred was dependent on the speed of the motion. Kannus and Beynnon (1993) and Brown et al. (1995) also found that peak torques are affected by the angular velocity; peak torque occurs later in the range of motion with increasing velocity. Therefore, clinicians should take this into consideration when evaluating muscular performance. The recorded peak torque may not represent the maximal torque for the patient, especially at higher angular velocities given that the limb may pass the optimal joint position for muscular performance (Kannus & Beynnon, 1993). Clarity of bilateral knee peak torque measures in clinical populations (especially ACLR patients) has yet to be achieved.

Clinicians and those interested in investigating isokinetic torques in the hamstring and quadriceps musculature should take gravity compensation of the limb into consideration when testing. Fillyaw et al. (1986) compared isokinetic knee flexor and extensor moments to assess hamstring to quadriceps strength ratio at peak torque angles with and without correcting for gravity at 60 and 240°/s. Uncorrected gravity underestimated quadriceps torque and overestimated hamstring muscle torque and the ratio between the two at both speeds. Uncorrected hamstring to quadriceps peak torque ratio increased as speeds went from 60 to 240°/s, however the gravity corrected ratio significantly decreased. Finucane et al. (1994) determined the error associated with the gravity-correction procedure of the KIN-COM dynamometer as a limb segment was weighed at different lever arm positions. The dynamometer recorded the rotational component of gravitational forces for the weight suspended from the lever arm accurately. The results of Aagaard et al. (1998) revealed that gravity correction influenced the ratio of hamstring to quadriceps torque when the extension velocity varied. When corrections for gravity were made, constant conventional hamstring to quadriceps ratios were maintained for various speeds. Functional ratios of hamstrings to quadriceps strength were calculated; extension ratios were based upon eccentric hamstring and concentric quadriceps moments, and flexion ratios were based on concentric hamstring and eccentric quadriceps moments. A potential 1:1 hamstring to quadriceps strength relationship was demonstrated for knee extension at the faster speed of 240°/s for the functional extension ratio. The authors suggested that the hamstring muscles have a significant functional capacity for providing dynamic stability at the knee joint as the hamstring eccentrically contracts (Aagaard et al., 1998). Due to the importance of

gravity correction during isokinetic testing, we used gravity compensation for all subjects. Isokinetic testing provides an excellent measure of the gross strength of muscle, however, it does not provide any information regarding the quality of the muscle. Therefore, additional measures must be made to evaluate muscle quality in order to further evaluate the muscle function.

Interpolated Twitch

Muscle quality has been evaluated in healthy and clinical populations via interpolated twitch techniques. The technique of twitch interpolation is commonly used to determine whether human muscles are activated fully during maximal voluntary efforts (also referred to as extrapolation) (Hales & Gandevia, 1988). Inferences can be made of the level excitability of motorneurons or neural drive with the measurement of voluntary activation via twitch interpolation (Herbert & Gandevia, 1999). Muscle function can be optimally tested by comparing a maximal voluntary isometric contraction, which relies on central neural drive to the muscle, with a maximal superimposed electrically evoked contraction independent of the central nervous system (Milner-Brown et al. 1973).

Supramaximal twitch interpolation is characterized by a single percutaneous tetanic pulse delivered during a maximal voluntary isometric contraction, which elicits muscle force known as an interpolated twitch (Herbert & Gandevia, 1999). Any rise in force due to the stimulus indicates that not all motor units were activated. The ratio of voluntary maximal effort to the electrically evoked involuntary maximal contraction is known as the CAR, and is used to assess the central inhibition in the muscle of an individual. CAR is calculated by dividing the maximum force before stimulation by the peak force recorded after the stimulation then multiplying this value by 100%. Central activation failure may alter force production by the muscle. If the CAR is equal between both

limbs, force production should not be affected. Incomplete motor unit activation of a muscle is an indicator of inhibition of the neural drive within the central nervous system during a maximal isometric contraction (Hunter et al. 1998).

Twitch interpolation is a reliable measure when completed in a thorough manner. Reproducibility of measurements during maximal voluntary activation was assessed by Allen et al. (1995). Maximal voluntary torques of the bicep brachii did not significantly vary significantly within a subject between sessions, however there were consistent differences in the level of maximal voluntary activation between subjects. Herbert & Gandevia (1999) evaluated interpolated twitch in the human adductor pollicis motorneuron pool and concluded that twitch interpolation may not be a sensitive measure of excitability of the motorneurons at near-maximal forces. These authors also suggested that large reductions in excitation of the motorneuron pool might be indicated by increases in the amplitude of interpolated twitches observed in fatigue and various pathologies. Sheild & Zhao's (2004) review of twitch interpolation techniques expressed that sensitive and high-resolution measurements of force are required to detect small activation deficits. Consideration of the site of stimulation, stimulation intensity, and the number of interpolated stimuli are important when using twitch interpolation techniques (Sheild & Zhao, 2004). Even with highly sensitive twitch interpolation techniques, healthy adults were unable to fully activate some musculature with maximal effort (Dowling et al., 1994; Allen et al., 1995). Roos et al. (1999) found no difference in the ability of males to activate the quadriceps to a high degree (94-96%). Stackhouse et al. (2000) measured CARs of the quadriceps during maximal voluntary contractions in healthy adults; all reached 95% or more. Rutherford et al. (1986) used the twitch

superimposition technique to study activation of the quadriceps in healthy young adults and patients with musculo-skeletal disorders. Most participants fully activated the quadriceps, however inhibition was seen in subjects with previous history of knee or joint injury and patients with muscle pain and joint pathology.

Hurley (1997) summarized deficits in quadriceps activation and its effect on rehabilitation in patients with traumatic and arthritic knee damage. Arthritic damage to the knee joint resulting in the inability to fully activate the muscle may lead to muscle weakness and atrophy impeding rehabilitation. Severity of joint damage secondary to ACL rupture influenced reduced muscle activation causing quadriceps weakness. ACLR was again suggested to increase quadriceps voluntary activation (Hurley, 1997). Hurley (1997) suggested that joint damage results in abnormal articular afferent information, which decreases alpha-motor neuron excitability and reduces voluntary quadriceps activation. This in turn decreases gamma-motor neuron excitability and results in decreased proprioception. Severe joint damage with large reduction in activation may prevent reaching the threshold for stimulation. Rehabilitation can increase alpha-motorneurone excitability as well as gamma-motorneuron excitability, improving proprioception (Hurley, 1997).

Muscle weakness is suggested to exceed what is expected by atrophy as a result of disuse alone (Elmqvist et al. 1988; Spencer et al. 1984). Instead, Elmqvist et al. (1988) and Spencer et al. (1984) indicate that the inability to voluntarily activate the muscle completely accounted for the muscle weakness. Urbach et al. (1999) investigated quadriceps muscle activation in ACL ruptured patients based on previous findings of voluntary activation deficits associated with other knee injuries with the aim to make

concrete conclusions regarding muscle quality in ACL deficient knees. Given that minor deficits of quadriceps activation were found in patients with unilateral ACL rupture (Hurley, 1997; Snyder-Mackler et al. 1994), Urbach et al. (1999) wanted to determine if these activation deficits were attributable to the rupture or the insignificant differences that can be found in normal healthy humans (Dowling et al. 1994). Urbach et al. (1999) found that patients with symptomatic, isolated ACL deficiency have only a statistically significant deficiency of voluntary quadriceps activation compared with an age, gender, and activity-matched healthy control group. In addition, the deficit in the ability to fully activate the muscle voluntarily in the involved quadriceps results in a crossover effect to the uninvolved quadriceps and is affected to the same extent. This diminished muscle strength of the uninvolved limb was explained solely by a deficit in voluntary activation. A uniform decline was found in the quadriceps muscle of the injured limb compared to the healthy limb. A deficit in voluntary activation during maximal isometric effort was evident, indicating that the atrophy was due to not using the musculature. The injured limb deficit compared to the healthy controls was explained by the voluntary-activation deficit and a true muscle weakness. Urbach et al. (1999) proposed an important consideration that the bilateral deficit in voluntary activation might challenge the validity of functional muscle tests when the uninjured extremity serves as reference. Urbach et al. (2001) later investigated voluntary quadriceps activation after ACLR to determine if voluntary activation could be reversed by repair of the ACL. Twelve male subjects with an isolated ACL tear and 12 matched control subjects before operation and two years after reconstruction of the ACL were evaluated. Prior to surgery, a similar bilateral deficit was found in voluntary quadriceps activation compared to the healthy controls.

Quadriceps voluntary activation improved significantly bilaterally 2 years after ACL reconstruction but remained less than the controls. In a similar study, Snyder-Mackler et al. (1994) evaluated reflex inhibition of the quadriceps femoris muscle after ACL injury and reconstruction. A burst-superimposition technique was used to assess the strength of the quadriceps muscle in a group of ACLR within 6 months post rupture, and two groups of subjects who had a torn ACL for an average of three months (subacute) and two years (chronic), both of which did not undergo ACLR. The ACLR and chronic ACL rupture groups did not present quadriceps activation deficits in the involved limb, whereas reflex inhibition of quadriceps contraction was evident in the subacute ACL rupture group. Based on mixed results concerning quadriceps activation deficits after ACLR, our study evaluated CAR in a group of unilateral ACLR averaging 2 to 4 years after reconstruction.

The purpose of our study was to perform a comprehensive evaluation of knee-extensor function in individuals with unilateral ACLR. More specifically, muscle quality via isokinetic strength testing and voluntary activation of the quadriceps femoris was assessed. Evaluation of muscle quality by measuring strength and CAR in ACLR patients may enable the clinician to determine the probability of functional knee instability, aid in prevention of re-injury as well as deciding when the appropriate time to return the patient to a pre-injury activity level.

We hypothesized that a decreased peak isokinetic extension torque would be present in the ACLR limb compared to the healthy limb as well as a smaller thigh circumference in the ACLR limb. In addition, a deficit in CAR of the ACLR limb compared to both the healthy side and control limb was suspected.

CHAPTER 3
METHODS

Participants

Twenty-four college-aged male and female unilateral ACL reconstructed individuals were recruited to participate in the study. Eligible individuals must have undergone surgical repair of one ACL at least six months prior to participation in which either an autograft or allograft patellar tendon or hamstring tendon graft was used. In addition participants were required to complete physical rehabilitation programs and resume pre-injury activity levels prior to involvement. Exclusion criteria required participants to be free of any additional lower extremity injury that hindered their physical activity within 6 months prior to testing. Twenty-three college-aged male and female control participants were also recruited for the study. The control participants had to be free of lower extremity injuries for a minimum of 6 months and could not have undergone surgery to the lower extremity. Participants were informed regarding the experimental protocol and signed an informed consent agreement under the established guidelines of the Institutional Review Board of the University of Florida before participation.

Instrumentation

A Landice treadmill (Model L8, New Jersey) was used for a warm-up before the strength and muscle activation testing. A flexible measuring tape was used to measure thigh circumference. A KINCOM (Chattanooga Group Inc.) isokinetic dynamometer collecting data at 40 Hz was used for all strength measures. A muscle stimulator,

GRASS, Model S48, utilizing two CONMED reusable neuromuscular stimulation electrodes was used to deliver electrical impulses to the quadriceps femoris muscle percutaneously. To amplify the electrical impulse from the GRASS stimulator to the muscle, a GRASS stimulator isolation unit (Model SIU8T) was used. LabVIEW realtime software was utilized to record the electrical impulses and the associated torque produced by the contracting muscle. A custom Quick Basic program was written to calculate the CAR (Appendix A). Excel ® 2000 was used for data reduction. Statistical analyses of knee extension torques, CAR, and thigh circumference were completed using SPSS © 2003. All equipment specifications appear in Appendix B.

Procedure

ACLR Participants

After signing the informed consent document, a secondary measure of mid-thigh circumference was collected bilaterally to evaluate the potential for thigh muscle atrophy after ACL construction. The same investigator made all measurements with a flexible measuring tape to obtain the largest circumference as the participant stood while the thigh was relaxed. Prior to isokinetic and CAR testing each participant warmed-up for 5 minutes on a treadmill at his/her own preferred walking pace. Bilateral knee extension torques and joint positions were assessed using the isokinetic dynamometer (Figure 3-1). Participants were in a seated position, with the chair back reclined to 78° and seat length set to 18 cm. During testing, a constant hip flexion angle of 85° was maintained. In addition, participants were placed in 90° of knee flexion and the dynamometer head was aligned with the axis of rotation of the knee at the lateral femoral epicondyle. Each participant was stabilized on the chair with two padded diagonal chest straps, a padded waist strap, an ipsilateral limb strap over the thigh, and a force transducer pad positioned

over the shin approximately 7.6 cm (3 in) above the lateral malleolus. Gravity correction of each limb was completed. Subjects were instructed to grasp the chest straps for support and to keep their trunk in contact with the back of the chair during testing. Subjects were given similar and consistent verbal encouragement to extend and flex the leg as hard and fast they could throughout the entire range of motion for all isokinetic testing conditions.

Figure 3-1. Participant positioning for isokinetic and CAR testing

Familiarization with the dynamometer was completed by performing 2 warm-up sessions including three submaximal repetitions at 180°/s. The isokinetic speed was set at 180°/s for the first two trials in which 3 maximum effort repetitions were performed

with a rest period of 2 minutes between trials. The subsequent two trials were performed at a slower speed of 60°/s in which 3 maximum efforts were performed with a 3-minute rest period between trials. Similar testing protocols have been implemented when assessing isokinetic knee strength (Anderson et al. 2002; Coombs & Cochrane, 2001; Keays et al. 2003; Maupas et al. 2002; Yoon et al. 1991). CAR was assessed on the isokinetic dynamometer using the isometric mode. CAR was determined while the subject performed maximum effort isometric knee extension as an interpolated supramaximal tetanus twitch is delivered to the quadriceps. The participant was positioned in the same seat arrangement as the isokinetic testing except the knee was placed at 90° of knee flexion angle for isometric testing. Two self-adhesive electrodes were placed in a bipolar configuration on the quadriceps muscle longitudinally. One self-adhesive electrode was placed over the proximal rectus femoris tendon, the other was placed over the prominent vastus medialis muscle, just above superior patellar pole. Prior to electrode placement, rubbing alcohol was swabbed over the electrode pad sites to remove any oil on the skin. A GRASS S48 stimulator and GRASS stimulus isolation unit were used to deliver the electrical impulse with a duration of 1/10000 second. The GRASS S48 Stimulator was set at the following parameters: frequency 50-60 Hz, 2 trains per second, train duration 120 ms, stimulus rate 100 pulse per second, and stimulus duration 6 ms. The voltage used for a CAR test was dependent on the voltage required for maximal motor recruitment of the muscle, determined by the titration process. Each participant was titrated to determine the voltage required for full muscle activation of the quadriceps. Titration enabled the participant to be familiarized with the sensation associated with the electrical impulse starting at the baseline of 10 volts and increasing

the voltage by increments of 10 volts. Once the stimulation level reached 80 volts, the LabVIEW realtime software program was used to capture the interpolated twitch and resulting force produced by the elicited muscle contraction. Data were sampled in LabVIEW at 15,000 Hz. As the recorded force curve plateaued (when the force did not increase with increased voltage), titration was finished and the maximal voltage required to recruit all motor units was determined. The voltage delivered to the human quadriceps to elicit full muscle activation was comparable to other investigations (Stackhouse et al. 2000; Rutherford et al. 1986). Once the titration was completed, the tetanic stimulus was applied to the muscle producing an electrically evoked contraction during a maximal isometric effort. To evaluate the CAR of the quadriceps, we asked each participant to perform a maximum effort isometric knee extension. Participants were given verbal encouragement to kick out as hard as possible while the KINCOM force transducer recorded the force. Once the maximum force was observed, a train of electrical impulses was delivered to the quadriceps. A 3-minute rest period was given between the 2 isometric testing trials. All tests were performed bilaterally in randomized limb order.

Control Participants

The control participants underwent equivalent procedures except the isokinetic testing was eliminated from the process. Thigh circumference was measured first and the same positioning procedure for the KINCOM was used. CAR testing followed a 5-minute warm-up on the treadmill. All CAR procedures were identical to the procedure the ACLR participants experienced.

Data Reduction

Peak isokinetic knee extension torque was calculated using a customized QuickBasic program (Microsoft ® QuickBasic 4.5). The peak torque over the two trials

(6 repetitions) during the isokinetic tasks (60º/s and 180º/s) was used in statistical analyses. A separate customized QuickBasic program was utilized to find CAR for each isometric knee extension trial. CAR was calculated using the following equation: CAR = maximum force before stimulation / peak force recorded after the stimulation X 100% (Figure 3-2).

$$CAR = \frac{a}{b} \text{ X } 100\%$$

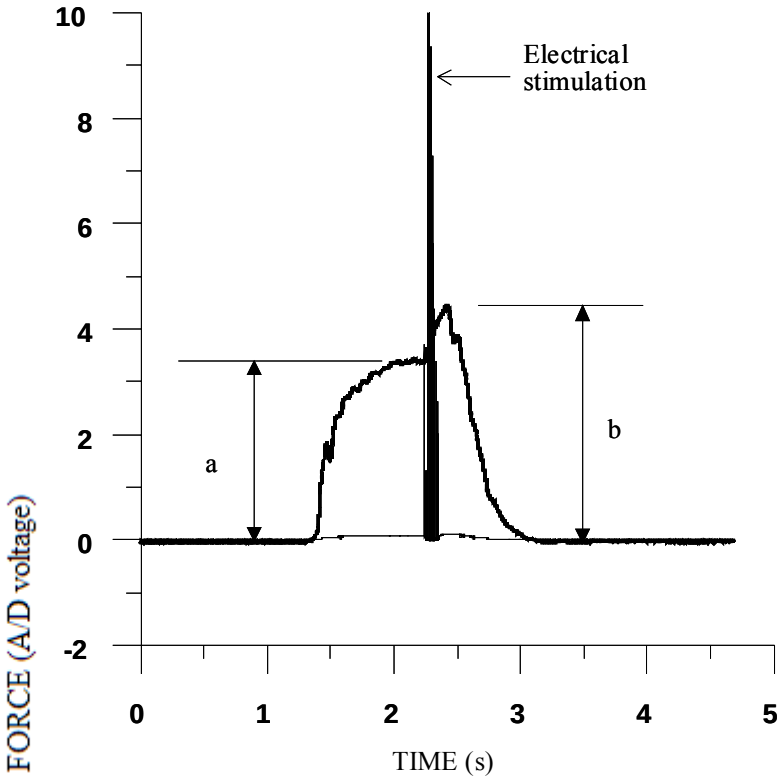

Figure 3-2. CAR calculation. Note that "a" is the prestimulation knee extensor force and "b" is the poststimulation knee extensor force.

Statistical Analysis

Paired sample t-tests were performed to ensure the ACLR and control groups were similar for comparison. A 2x2 multivariate analysis of variance (MANOVA) with repeated measures on lower extremity was conducted on the ACLR group to determine if gender and lower extremity (uninjured and ACLR) had a significant effect on isokinetic

peak extensor torque (60°/s and 180°/s), CAR, and thigh circumference. A separate 2x2 MANOVA with repeated measures on lower extremity side was also performed on the control group to determine if gender and lower extremity (right and left) have a significant effect on CAR and thigh circumference. Given that isokinetic strength data for ACLR individuals are available, the values obtained in this were compared to previous studies. To evaluate the influence of gender and leg on CAR and thigh circumference, two separate 2x3 ANOVAs were performed analyzing the ACLR and control participants simultaneously. The leg variable was divided into the healthy ACLR limb, the injured ACLR limb, and the control limb. After a paired sample t-test confirmed the limbs were not significantly different, the average of the left and right limb was calculated for control participants and used as the control limb in the ANOVAs. The level of significance for all tests was set at .05.

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CHAPTER 4
RESULTS

Of the 24 ACLR participants, 18 had patellar tendon autografts, 2 had hamstring tendon autografts, and 4 had allografts. T-tests were performed to confirm that the control and ACLR groups were matched by age, height, and weight. The results revealed that males in the ACLR group were appropriately matched to the control group by age ($t(21) = -.599$, $p = .555$), height ($t(21) = -.524$, $p = .605$) and weight ($t(21) = -1.530$, $p = .141$). The females in the ACLR group were appropriately matched by height ($t(22) = -1.372$, $p = .184$) and weight ($t(22) = -.370$, $p = .715$) to the control group, however the controls were 1.3 years older ($t(22) = -3.145$, $p = .005$). Means and standard deviations of age, height, and weight for the ACLR and control group are presented in Table 1.

Table 4-1. Subject Characteristics

	Age (yrs + SD)	Height (cm + SD)	Weight (N + SD)	Time post surgery (yrs + SD)
Female ACLR (n=13)	20.2 ± 1.1*	162.5 ± 5.7	592.5 ± 61.4	2.5 ± 1.5
Male ACLR (n=11)	21.3 ± 2.5	177.9 ± 7.4	772.0 ± 112.1	3.3 ± 1.8
Female Control (n=11)	21.5 + .82 *	165.5 + 4.8	606.2 + 114.1	
Male Control (n=12)	21.8 + 1.1	179.3 + 4.9	855.9 + 146.9	

[1] Significantly different ($p < .05$)

The 2x2 MANOVA performed on the ACLR group revealed main effects for gender (Wilks' $\Lambda = .357$, $F(4,19) = 8.541$, $p < .001$, multivariate $\eta^2 = .643$) and lower extremity side (Wilks' $\Lambda = .613$, $F(4,19) = 2.995$, $p = .045$, multivariate $\eta^2 = .387$) indicating that both have a significant influence on the dependent variables. However, a significant interaction between lower extremity and gender in the MANOVA was not observed (Wilks' $\Lambda = .881$, $F(4,19) = .641$, $p = .640$, multivariate $\eta^2 = .119$). Although a

main effect for gender was found in the 2x2 MANOVA for controls, in which males had larger thigh circumference compared to females (Wilks' $\Lambda = .741$, $F(2,20) = 3.499$, $p = .05$, multivariate $\eta^2 = .259$), extremity side (Wilks' $\Lambda = .897$, $F(2,20) = 1.147$, $p = .338$, multivariate $\eta^2 = .103$) and the interaction of side and gender (Wilks' $\Lambda = .860$, $F(2,20) = 1.634$, $p = .220$, multivariate $\eta^2 = .140$) were not significant. Univariate ANOVAs were computed as follow-up tests when appropriate.

Strength

Peak extension torque was normalized by the participant's body weight for analysis. Between subjects comparison revealed that peak extension torque significantly differed in which males were able to generate greater peak extensor torque compared to females at speeds of 60 °/s and 180 °/s ($F(1,22) = 20.758$, $p < .001$, partial $\eta^2 = .485$; $F(1,22) = 24.546$, $p < .001$, partial $\eta^2 = .527$, respectively). Within subjects comparison revealed that peak extension torques at speeds of 60 °/s and 180 °/s ($F(1,22) = 4.850$, $p = .038$, partial $\eta^2 = .181$; $F(1,22) = 7.624$, $p = .011$, partial $\eta^2 = .257$, respectively) were lower in the ACLR leg compared to the healthy leg. Means and standard deviations of peak extension torque for the ACLR subjects are reported in Table 4-2 and Table 4-3.

Table 4-2. Normalized Knee-Extension Peak Torque (N*m/BW) Values for the ACLR Participants' Involved Knee, Uninvolved Knee, and Totals (Mean + SD) at 60°/s

	Female (n=13)	Male (n=11)	Totals (n=24)
Involved	.18 + .03	.25 + .05	.21 + .05 *
Uninvolved	.19 + .04	.26 + .05	.22 + .05 *
Totals	.19 + .04 †	.26 + .05 †	

[2] Significantly different gender peak extension torques ($p < .001$)
[3] Significantly different peak extension torques for involved and uninvolved limb ($p < .05$)

Table 4-3. Normalized Knee-Extension Peak Torque (N*m/BW) Values for the ACLR Participants' Involved Knee, Uninvolved Knee, and Totals (Mean + SD) at 180°/s

	Female (n=13)	Male (n=11)	Totals (n=24)
Involved	.12 + .03	.19 + .04	.16 + .05 *
Uninvolved	.13 + .03	.21 + .05	.17 + .06 *
Totals	.13 + .03 †	.20 + .05 †	

[4] Significantly different gender peak extension torques ($p < .001$)
[5] Significantly different peak extension torques for involved and uninvolved limb ($p < .05$)

CAR

Between subjects comparison revealed that CAR for the ACLR participants did not significantly differ based on gender ($F(1,22) = .876$, $p = .359$, partial $\eta^2 = .038$). Within subjects comparison revealed that CAR ($F(1,22) = 4.432$, $p = .047$, partial $\eta^2 = .168$) was significantly lower in the ACLR leg compared to the healthy leg. Means and standard deviation of CAR for the ACLR and control groups are reported in Table 4. Between subject comparisons revealed that gender did not significantly influence CAR values in the control group ($F(1,21) = 2.009$, $p = .171$, partial $\eta^2 = .087$). Mean CAR values for the control participants are reported in Table 5. Two separate 2x3 ANOVAs were performed to determine if the ACLR group differed from the control group among CAR and thigh circumference variables. The paired t-test performed on the control group revealed that the left and right limbs were not significantly different ($t(22) = 1.19$, $p = .246$). Given that a bilateral difference of the control limbs did not exist, an average of the right and left thigh circumference and CAR values were calculated and used as the control limb in the ANOVAs. Between subjects comparison for CAR measurements failed to reveal main effects for gender [$F(1,65) = 3.031$, $p = .086$] and lower extremity [$F(2,65) = .785$, $p = .460$] or a significant interaction of gender and lower extremity [$F(2,65) = .089$, $p = .915$].

Table 4-4. CAR (Mean + SD) for the ACLR Injured, ACLR Uninjured, and Control Limbs

	Female	Male	Totals
ACLR Involved	92 + 6.5	90 + 7.8	91 + 7.0 *
ACLR Uninvolved	95 + 3.9	92 + 7.6	93 + 5.9 *
Control Limb (n=23)	94 + .03	91 + .06	92 + .05

[6] Significantly different ($p < .05$)

Table 4-5. CAR (Mean + SD) for the Control Limbs

	Female (n=11)	Male (n=12)	Totals
Left	94 $\pm$ 3.1	91 $\pm$ 5.1	92 $\pm$ 4.5
Right	93 $\pm$ 3.4	91 $\pm$ 7.4	92 $\pm$ 5.8
Totals	94 + 3.3	91 + 6.2	

Thigh Circumference

Between subjects comparison revealed that gender did not influence thigh circumference in the ACLR participants ($F(1,22) = .612$, $p = .442$, partial $\eta^2 = .027$). Furthermore, the within subject comparison revealed that thigh circumference did not differ between healthy and ACLR legs ($F(1,22) = 3.237$, $p = .086$, partial $\eta^2 = .128$). Means and standard deviations for thigh circumference for the ACLR and control groups are reported in Table 6. The between subject comparisons in the control group analysis revealed that gender had a significant influence on thigh circumference ($F(1,21) = 6.349$, $p = .020$, partial $\eta^2 = .232$) in which males had larger thighs compared to females. Means and standard deviations of thigh circumference values for the control subjects are reported in Table 7. The paired t-test performed on the control group revealed that the left and right limbs were not significantly different ($t(22) = .682$, $p = .502$). Therefore, the average of the right and left control limbs were used for the ANOVA comparison. The ANOVA comparing the healthy and control group revealed only a significant difference for gender in the between subjects comparison. Thigh circumference in the males was

greater compared to the females [F(1,65) = 5.741, p = .019]. However, neither a main effect was found for lower extremity [F(1,65) = 2.548, p = .086] nor an interaction of gender and lower extremity [F(2,65) = 1.189, p = .311].

Table 4-6. Thigh Circumference (m) for the ACLR Injured, ACLR Uninjured, and Control Limbs

	Female	Male
ACLR Involved	.51 + .05	.53 + .05
ACLR Uninvolved	.52 + .04	.53 + .05
Control Limb (n=23)	.52 + .06 *	.57 + .04 *

[7] Significantly different (p< .05)

Table 4-7. Thigh Circumference (m) for the Control Limbs

	Female (n=11)	Male (n=12)	Totals
Left	.52 + .06	.58 + .04	.55 + .06
Right	.52 + .06	.57 + .04	.55 + .05
Totals	.52 + .06 *	.57 + .04 *	

[8] Significantly different (p< .05)

CHAPTER 5
DISCUSSION

Our study evaluated college-aged males and females who have undergone ACL reconstruction (who were on average 3 years post-operation) and compared the clinical group to healthy control participants. Both patellar tendon and semitendinosis grafts were used; furthermore the reconstruction procedure in the ACLR patients was not uniform for the patients. All patients were able to return to pre-injury activity levels. Deficits in isokinetic knee extensor strength in the ACLR limb and lower voluntary activation compared to the contralateral limb were revealed.

Strength

Regaining thigh strength is crucial to maintaining dynamic support of the knee, especially after injury. Moreover, appropriate activation of the knee-extensors and flexors are of equal importance when joint stability is challenged. Overall, deficits of 7% and 8% were found in knee-extensor strength of the ACLR leg compared to the healthy leg at 60°/s and 180°/s, respectively. When the female and male patients were separated, females displayed 9% and 7% deficits in the ACLR knee-extensor strength compared to healthy knee-extensor strength at 60°/s and 180°/s, respectively. Male patients had deficits of 6% and 8% in the strength of the ACLR knee-extensors compared to the healthy leg at 60°/s and 180°/s, respectively. Knee-extensor strength deficits found in our patient population are comparable to other studies arriving at similar findings. Rosenberg et al. (1992) reported isokinetic strength deficits of 18% in ACLR patients (1 to 2 years

after surgery) in the knee-extensors at isokinetic speeds of 60°/s compared to the contralateral limb.

Hamstring muscle activation is important in protecting ACL by slowing down anterior tibial translation. Fortunately, female and male patients who have undergone ACLR are reported to regain hamstring strength within 6 months to a year after surgery (Kobayashi et al. 2004). Based on the results of Wilk et al. (1994), Natri et al. (1196) and the more recent work of Kobayashi et al. (2004), hamstring strength deficits were not as large as the quadriceps deficits. Kobayashi et al. (2004) found that 36 ACLR patients recovered 90% of isokinetic knee-flexor strength within 6 months after surgery. Extensor strength recovered more slowly compared to the knee-flexors. The quadriceps strength lagged to deficits of 27% at 60°/s and 12% at 180°/s. Similarly, Wilk et al. (1994) reported two-thirds of the patients reached 90% of knee flexor strength in the ACLR limb compared to the healthy limb at 180°/s; whereas less than a tenth could reach 90% of the knee-extensor strength of the healthy knee. Natri et al. (1996) reported mean peak torque deficits of the ACLR knee of 15% and 9% compared to a hamstring deficit of only 7% and 5% at speeds of 60°/s and 180°/s, respectively. Harter et al. (1990) reported maximum quadriceps torque deficits of 14% at isokinetic speeds of 120°/s in the ACLR limb compared to the contralateral limb in patients with mean age of 23 years and post-operative periods of at least 2 years. Even larger quadriceps strength deficits were reported by Heimstra et al. (2000). A global deficit of 25% was found in the knee-extensors in ACLR patients 2.5 years post-surgery (using either hamstring or patellar tendon graft types) compared to matched healthy controls.

Based on the results of these studies and our research, it appears that knee-extensor strength deficits are long-term consequences. The plausible causes of the deficits may be due to an incomplete rehabilitation of the ACLR knee or the inability to fully activate the muscle as a result of the initial and post-operative joint damage. Measuring CAR may be used to assess a deficit in the ability to voluntarily activate the muscle during maximum efforts.

CAR

The ability to voluntarily activate muscles and make appropriate adjustments in order to execute a coordinated task is important. Incomplete motor unit activation of a muscle is an indicator of inhibition of the neural drive within the central nervous system during a maximal isometric contraction (Hunter et al. 1998). Joint damage may interfere with the ability to fully activate a muscle (Hurley, 1997; Rutherford et al.1986). Furthermore, muscle atrophy or weakness may be encountered, introducing an additional challenge to rehabilitation (Hurley, 1997; Elmqvist et al. 1988; Spencer et al. 1984). Urbach et al. (1999) reported that the diminished muscle strength in the involved limb in ACL deficient patients was explained solely by a deficit in voluntary activation due to muscle weakness. Furthermore, a crossover effect was found in the uninjured limb (voluntary activation was reduced to the same extent as the ACLR limb) when compared to healthy controls. The crossover effect decreased around 2 years after ACL reconstruction although a deficit remained (Urbach et al., 2001). Snyder-Mackler et al. (1994) failed to find voluntary activation deficits in the 20 ACLR patients who participated, however no controls were used in the design of the study. Our study revealed that the CAR for the ACLR limb was significantly lower than the contralateral limb (when only ACLR patients were analyzed). A mean lower CAR in the ACLR limb

(2% for the females and 3% for the male patients) compared to the healthy contralateral limb was revealed. All subjects who participated in the study by Urbach et al. (2001) were male. Comparable CAR values of 91% were found for our male control subjects. However, ACLR patients had higher CAR values compared to the patients in the study by Urbach et al. (2001). CAR values of 90% and 92% were found in the involved and contralateral limb, respectively in our study, whereas Urbach et al. (2001) reported CAR values of 85% and 84%, respectively. Voluntary quadriceps muscle activation was reduced in the ACLR limb, however we did not find a crossover effect; no difference was found in the ACLR group compared to the healthy controls.

No differences among the ACLR leg, healthy leg, and control leg was revealed in CAR when simultaneously compared. The strength deficits found in the ACLR group may therefore be attributed to the lower CAR in the involved limb. Overall the voluntary activation of the healthy control participants averaged 92%. The CAR values are similar to the values of the voluntary activation of the quadriceps reported by Stackhouse et al. (2000), in which all healthy adults reached 95%. Similarly, Roos et al. (1999) found that males activated the quadriceps to a high degree (94-96%).

The procedure implemented to assess maximal voluntary activation in the present study was similar to Stackhouse et al., 2000; Rutherford et al., 1986. Although the twitch interpolation technique to determine voluntary activation is a sensitive measure at maximal efforts, most reliability research is on various muscle groups. Allen et al. (1995) reported consistent reproducibility of measurements within participants on the of the bicep brachii. Herbert & Gandevia (1999) evaluated interpolated twitch in the human adductor pollicis motorneuron pool. Limited research is available on the reproducibility

of voluntary activation. Further research should target the reliability on quadriceps femoris muscle group.

Thigh Circumference

No gender differences for thigh circumference were found in ACLR group. However, thigh circumference of the males was larger (M = .57, SD = .04) compared to the females (M = .52, SD = .06) in the control group. The ANOVA revealed similar results between the healthy and control group, in which males had a larger thigh circumference than the females. The 5% larger thigh circumference exhibited by the male participants compared to that of the females may not be clinically significant. Male participants were also larger (178.6 cm, 814.0 N). Although measuring thigh circumference with a flexible measuring tape is an easy and inexpensive technique for a clinician, magnetic resonance imaging of the cross-sectional area is a more accurate measure of the muscle and correlated strength (Arangio et al., 1997). According to findings of Arangio et al (1997), thigh circumference in the injured limb underestimated thigh atrophy and was not correlated with strength. As a clinician, underestimates of atrophy by thigh circumference should be considered when addressing muscle weakness in rehabilitation.

Limitations

A limitation of this study is having female ACLR patients that were on average 1.3 years younger than the control females. However, this age difference may not be clinically significant. The mean age was 20.2 and 21.5 years and for the ACLR and control participants, respectively.

A majority of the ACLR patients had patellar tendon autografts in the present study. Although previous studies have reported no difference in strength of the knee-

extensors after ACLR between patellar tendon and semitendinosis grafts (Harter et al., 1990), the same conclusions have yet to be made regarding graft type and voluntary activation of the quadriceps. All the patients in the study by Urbach et al. (2001) had the ACL repaired using a semitendinosis tendon grafts. Gaps remain in the research concerning voluntary activation of the quadriceps after ACLR.

Mean time after reconstruction was 2.5 years for females and males 3.3 years. The knee extensor deficit appears to be a permanent consequence of the injury. Therefore, a generalization may be made regarding long-term knee-extensor strength deficits in ACLR patients even after completing rehabilitation and returning to pre-activity levels. Further research is essential to determine whether isokinetic strength and voluntary activation are predictors of re-injury. Clinicians should consider any deficits in muscle quality when returning the patient to a pre-injury activity level based.

Conclusion

The ACLR patients included in our study were on average 2.5 to 3.3 years after surgical reconstruction of the ACL and were able to return to pre-injury activity levels. Overall, lower isokinetic knee-extensor strength of 7% and 8% and was found in the ACLR patients at speeds of 60 °/s and 180 °/s, respectively. Lower voluntary quadriceps activation was revealed in the ACLR limb compared to the contralateral healthy limb. CAR and thigh circumference were not significantly different among the ACLR knee, contralateral limb, and control limbs. Gender differences were found in thigh circumference in which males had larger circumferences compared to females. The knee-extensor strength deficits found in ACLR patients appear to be long-term effects as a result of the joint damage experienced. Muscle quality is not optimal in the ACLR patient, however when compared to healthy controls the ACLR patients are not

significantly different and therefore may not be at an increased risk for re-injury. Monitoring strength values alone may not be enough to evaluate the progression of recovery, given that the underlying mechanism of the strength deficits may be attributed to the inability to reach maximum voluntary activation. The strength deficits revealed in the ACLR leg are attributable to lower voluntary activation compared to the contralateral leg given that no difference was found in thigh circumference between legs. Further research is required to conclude the mechanism underlying central inhibition and neural drive to the quadriceps femoris. Twitch interpolation may be a valuable tool for determining activation deficits and addressing the progression of rehabilitation after joint damage and surgery. A well planned and executed therapy program including functional rehabilitation that targets muscle re-education and proprioceptive activities appears to critical in overall joint health and recovery.

APPENDIX A
QUICKBASIC PROGRAM FOR CAR

```
' ** Filename: CAR3.bas                          11/2/03
'** This program computes the central activation ratio & preES peak torque.
'** Smoothed force data sampled at 15,000 Hz. (BiStreng)

DIM Vex, Force
DIM NoF AS DOUBLE           ' Number of frame
DIM Dir AS STRING           ' Directory
DIM Subj AS STRING          ' first and last initials
DIM File AS STRING          ' Current file name
DIM Trial AS STRING         ' trial # 1 or 2
DIM Side AS STRING          ' Side of the leg (left or right)
DIM fBeginES AS DOUBLE      ' frame -- begin ES
DIM fEndES AS DOUBLE        ' frame -- end ES
DIM fBeginMVIC AS DOUBLE    ' frame -- begin MVIC
DIM fEndMVIC AS DOUBLE      ' frame -- end MVIC
DIM fEnd AS INTEGER         ' frame -- end
DIM k AS DOUBLE
CLS
k = 0: Flag = 0: Switch = 0
fBeginMVIC = 0: fBeginES = 0
ForceMVIC = 0: ForceMVIC2 = 0: ForceMVIC90 = 0: ForceMVIC15 = 0: ForceES = 0
Dir = "c:\ESS_Res\BiStreng\Data\"
OPEN Dir + "subjlist.txt" FOR INPUT AS #3
'OPEN Dir + "OneSubj.txt" FOR INPUT AS #3
FrameR = 15000
DO UNTIL EOF(3)
INPUT #3, Subj                  ' Read in subject ID
PRINT Subj
dt = 1 / FrameR                 ' time interval = 1/sample rate
OPEN Dir + "\" + Subj + "\" + Subj + "x.car" FOR OUTPUT AS #2
FOR q = 1 TO 2
IF q = 1 THEN Side = "L"
IF q = 2 THEN Side = "R"
FOR r = 1 TO 2
  IF r = 1 THEN Trial = RTRIM$(LTRIM$(STR$(1)))
  IF r = 2 THEN Trial = RTRIM$(LTRIM$(STR$(2)))
File = Subj + Side + Trial
'IF File = "A10R2" THEN GOTO 100
IF File = "A12L2" THEN GOTO 100
```

```
IF File = "A13R2" THEN GOTO 100
OPEN Dir + Subj + "\" + File + ".dat" FOR INPUT AS #1
PRINT "Processing ...";
PRINT File
DO UNTIL EOF(1)                                    ' 1st time
k = k + 1
  INPUT #1, Ves, VForce
  VForce = ABS(VForce)
'  PRINT USING "#########"; k;
'  PRINT USING "###.###"; Ves; VForce
'    WHILE INKEY$ = "": WEND
IF Flag = 0 THEN
  IF Ves > .5 THEN              ' Locate the frame for elec stim begin
    fBeginES = k
    Flag = 1
'    WHILE INKEY$ = "": WEND
  END IF
END IF
IF Switch = 0 THEN
  IF VForce > .1 THEN             ' Locate the frame for MVIC begin
    fBeginMVIC = k
    Switch = 1
    'WHILE INKEY$ = "": WEND
  END IF
END IF
IF Flag = 1 THEN
  IF VForce < .1 THEN             ' Locate the frame for MVIC end
    fEndMVIC = k
    Switch = 0
    'WHILE INKEY$ = "": WEND
  END IF
END IF
IF k > fBeginMVIC AND Flag = 0 THEN
  IF VForce > ForceMVIC THEN
    ForceMVIC = VForce                 ' Max force before elec stim
    fForceMVIC = k
  END IF
END IF
IF Flag = 1 THEN
  IF VForce > ForceES THEN
    ForceES = VForce                   ' Max force after elec stim
    fForceES = k
  END IF
END IF
54
LOOP                                  ' Loop for File #1 (1st)
```

```
CLOSE 1
NoF = k
k = 0
PRINT "# of Frames", USING "########"; NoF
PRINT "Fr_ES_Begin", USING "########"; fBeginES
'PRINT "Fr_ES_End", USING "########"; fEndES
PRINT "Fr_MVIC_Begin", USING "########"; fBeginMVIC
PRINT "Fr_MVIC_End", USING "########"; fEndMVIC
PRINT "Pre_ES_Force", USING "#####.##"; ForceMVIC
PRINT "Post_ES_Force", USING "#####.##"; ForceES
OPEN Dir + "\" + Subj + "\" + File + ".dat" FOR INPUT AS #1   ' 2nd time
OPEN Dir + "\" + Subj + "\" + File + "x.txt" FOR OUTPUT AS #4
DO UNTIL EOF(1)
k = k + 1
  INPUT #1, Ves, VForce
  VForce = ABS(VForce)
'  PRINT USING "#########"; j; k;
'  PRINT USING "###.###"; Ves; VForce
IF k > fBeginES - FrameR / 2 AND k < fBeginES THEN
  SumForce1 = SumForce1 + VForce                    ' SumForce-half second
END IF
IF VForce > ForceMVIC * .9 AND k < fBeginES THEN
  SumForce2 = SumForce2 + VForce                    ' SumForce-90% pre_max
  Count = Count + 1
END IF
NoF15 = FrameR * .015                               ' # of frames in 15 ms
IF k > fBeginES - NoF15 AND k < fBeginES THEN
  SumForce15 = SumForce15 + VForce                  ' SumForce-15 ms
END IF
'** Extract data from .5 s before to .5 s after stim
IF k > fBeginES - FrameR / 2 AND k < fBeginES + FrameR / 2 THEN
PRINT #4, USING "###.#####"; Ves; VForce
END IF
LOOP                                    ' Loop for File #1 (2nd)
CLOSE 1, 4
ForceMVIC2 = SumForce1 / FrameR * 2         ' MVIC-half second
ForceMVIC90 = SumForce2 / Count             ' MVIC-average > 90% MVIC
ForceMVIC15 = SumForce15 / NoF15            ' MVIC-15 ms
PRINT "MVIC-.5s", USING "###.##"; ForceMVIC2
PRINT "MVIC90", USING "###.##"; ForceMVIC90
CAR = ForceMVIC / ForceES
CAR2 = ForceMVIC2 / ForceES
CAR90 = ForceMVIC90 / ForceES
CAR15 = ForceMVIC15 / ForceES
PkBurst = ForceES - ForceMVIC15
PRINT
```

```
PRINT "CAR", USING "###.##"; CAR
PRINT "CAR(.5s)", USING "###.##"; CAR2
PRINT "CAR(90%)", USING "###.##"; CAR90
PRINT "CAR(15ms)", USING "###.##"; CAR15
WHILE INKEY$ = "": WEND
' ** OUTPUT Data ***
PRINT "Output computed parameters ... "
PRINT #2, CHR$(34) + File + CHR$(34)
PRINT #2, USING "####.##"; ForceMVIC; ForceMVIC2; ForceMVIC90;
ForceMVIC15; ForceES; PkBurst
PRINT #2, USING "####.##"; CAR; CAR2; CAR90; CAR15
PRINT #2,
'WHILE INKEY$ = "": WEND
PRINT
'100
' Reset values
k = 0
Flag = 0
Switch = 0
ForceMVIC = 0: ForceMVIC2 = 0: ForceMVIC90 = 0: ForceMVIC15 = 0
ForceES = 0
SumForce1 = 0: SumForce2 = 0: SumForce15 = 0
Count = 0
100
NEXT                          ' Loop for r
NEXT                           ' Loop for q
CLOSE 2
LOOP                           ' Loop for File #3
END
```

APPENDIX B
QUICK BASICPROGRAM FOR ISOKINETIC STRENGTH

```
        * Filename: Isok1.bas                    10/4/04'
'** This programs seaches for peak isokinetic torques (Dana)
DIM Dir AS STRING
DIM Subj AS STRING          ' Subject ID
DIM Speed AS STRING          ' Isokinetic speed (180 or 60 deg/s)
DIM Side AS STRING          ' left (L) or right (R) leg
DIM File AS STRING
DIM Trial AS STRING         ' trial # 1 or 2
DIM Dummy AS STRING
DIM MxForce, MnForce, MxForAng, MnForAng, MxForAngVel
DIM MnForAngVel, Time(2000), Ang(2000), AngVel(2000), Force(2000)
DIM MxForce(35), MnForce(35), MxForAng(35), MnForAng(35), MxForAngVel(35)
DIM MnForAngVel(35)
Dir = "c:\ESS_Res\BiStreng\Data\"
'OPEN Dir + "subjlist.txt" FOR INPUT AS #3
OPEN Dir + "onesubj.txt" FOR INPUT AS #3
DO UNTIL EOF(3)
CLS
INPUT #3, Subj, ArmL, ArmR
PRINT Subj, ArmL, ArmR
OPEN Dir + "\" + Subj + "\" + Subj + ".dat" FOR OUTPUT AS #2
FOR p = 1 TO 2
IF p = 1 THEN Speed = "180"
IF p = 2 THEN Speed = "60"
  FOR q = 1 TO 2
   IF q = 1 THEN Side = "L"
   IF q = 2 THEN Side = "R"
    FOR r = 1 TO 2
     IF r = 1 THEN Trial = RTRIM$(LTRIM$(STR$(1)))
     IF r = 2 THEN Trial = RTRIM$(LTRIM$(STR$(2)))
IF r = 1 THEN
  j = 1
' ELSE
'  j = 4
END IF
File = Subj + Side + Speed + Trial
PRINT File
PRINT #2, File
```

```
OPEN Dir + "\" + Subj + "\" + File + ".txt" FOR INPUT AS #1
'CLS
FOR i = 1 TO 35
  LINE INPUT #1, Dummy
'PRINT Dummy
NEXT i
'INPUT #1, DummyNum
k = 0
DO UNTIL EOF(1)
k = k + 1
INPUT #1, Time(k), Ang(k), AngVel(k), Force(k)
'IF k < 5 THEN
'PRINT Time(k), Ang(k), AngVel(k), Force(k)
'WHILE INKEY$ = "": WEND
'END IF
IF Force(k) > MxForce(j) THEN
  MxForce(j) = Force(k)
  MxForAng(j) = Ang(k)
  MxForAngVel(j) = AngVel(k)
END IF
IF Force(k) < MnForce(j) THEN
  MnForce(j) = Force(k)
  MnForAng(j) = Ang(k)
  MnForAngVel(j) = AngVel(k)
END IF
IF Force(k - 1) < 0 AND Force(k) >= 0 THEN
  j = j + 1
  'PRINT File, r, k, j
'WHILE INKEY$ = "": WEND
END IF
LOOP
CLOSE 1
NEXT                                    ' Loop for r
IF q = 1 THEN
  Arm = ArmL / 100                            ' Moment arm (KinCom)
ELSE
  Arm = ArmR / 100
END IF
FOR j = 1 TO 35
  PRINT MxForce(j);                   ' Peak extension force
  PRINT MxForAng(j);
  PRINT MxForAngVel(j);
  PRINT MxForce(j) * Arm
  PRINT #2, MxForce(j);                   ' Peak extension force
  PRINT #2, MxForAng(j);
  PRINT #2, MxForAngVel(j);
```

```
  PRINT #2, MxForce(j) * Arm
NEXT
  PRINT : PRINT #2,
FOR j = 1 TO 35
  PRINT MnForce(j);                ' Peak flexion force
  PRINT MnForAng(j);
  PRINT MnForAngVel(j);
  PRINT MnForce(j) * Arm
  PRINT #2, MnForce(j);              ' Peak flexion force
  PRINT #2, MnForAng(j);
  PRINT #2, MnForAngVel(j);
  PRINT #2, MnForce(j) * Arm
NEXT
PRINT : PRINT #2,
'WHILE INKEY$ = "": WEND
FOR i = 1 TO 35
  MxForce(i) = 0
  MnForce(i) = 0
  MxForAng(i) = 0
  MnForAng(i) = 0
  MxForAngVel(i) = 0
  MnForAngVel(i) = 0
NEXT i
NEXT                               ' Loop for q
NEXT                               ' Loop for p
CLOSE 2
BEEP
LOOP                               ' Loop for file #3
CLOSE
'WHILE INKEY$ = "": WEND
END
```

APPENDIX C
INSTRUMENTATION

Landice Treadmill
Model L8: 110 V, 60 Hz, 15 A
Landice, Inc.
111 Canfield Avenue
Randolph, New Jersey 07869
USA

KINCOM AP125
Chattanooga Group
4717 Adams Road
Hixson, TN 37343
USA

GRASS S48 Stimulator
Model S48: 115 V, 50-60 Hz
Grass Medical Instruments Since 1935
Quincy, Mass
USA

GRASS stimulus isolation unit
Model SIU8T
Grass Medical Instruments Since 1935
Quincy, Mass
USA

Electrodes
VERSA-STIM REF 650-3050
3 inch X 5 inch self-adhesive reusable neuromuscular stimulation electrodes.
CONMED Corporation
310 Broad St.
Utica, NY 13501
USA

LabVIEW
© National Instruments Corporation
11500 North Mopac Expressway
Austin, Texas 78759
USA

Microsoft ® QuickBasic 4.5
© 2004, Microsoft Corporation
Microsoft Corporation
One Microsoft Way
Redmond, WA 98052-6399
USA

SPSS for Windows Version 11.0.1
Copyright © 2003, SPSS Inc.
SPSS Inc. Headquarters
233 S. Wacker Drive
Chicago, Illinois 60606
USA

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BIOGRAPHICAL SKETCH

I was born in Ocala, Florida, on June 4th 1980, to Mr. David A. Otzel and Mrs. Donna B. Otzel. I attended Belleview High School (BHS) in which I lettered in four varsity sports (basketball, volleyball, softball, and golf) and was a member and treasurer of the National Honor Society. I also was the recipient of the All American Scholar Award; the Citizen/Scholar/Athlete of the Year in 1995, 1996, and 1997; US Marines Corps Scholastic Excellence Award; US Army Academic and Athletic Excellence Award; and the BHS Nominee for the Silver Garland in Athletics. After graduating from high school in 1998, I attended Stetson University in DeLand, Florida.

I decided to major in athletic training after assisting Jim Simmons, the athletic trainer at BHS, who continues to be a great mentor and friend. The curriculum coordinator for the athletic training program at Stetson was Sue Guyer. Her dedication to the program was remarkable. She provided me with tremendous much support, guidance and friendship. The program was demanding and rewarding. An undergraduate biomechanics course taught by Dr. Tillman, convinced me of the appeal for biomechanics.

I started the graduate biomechanics program at the University of Florida in 2002. As a first-year graduate assistant, I taught sport and fitness classes. During the second year, I was given the opportunity to continue my experience in the athletic training field by having the head athletic trainer position at Lofton High school, as well as assisting in teaching lower assessment labs and supervising undergraduate athletic-training students.

In my third year, I worked in the Athletic Training Sports Medicine Clinic and intramural and club sports at UF. The past 7 years in college have been the best years of my life and I am looking forward to starting the doctoral program at the University of Florida.