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The Strength of the Quadriceps Femoris Muscle and the Kinetics of Cycling After Reconstruction of the Anterior Cruciate Ligament

by

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Abstract

This study examined quadriceps femoris (QF) muscle strength, the level of QF muscle inhibition (MI) and the kinetics of cycling in 11 subjects post-anterior cruciate ligament (ACL) reconstruction and 7 controls. Isokinetic knee extension (90°/s and 240°/s), isometric knee extension, MI (twitch interpolation) and anterior knee joint laxity (KT 2000) were measured. For the cycling test, pedal forces were measured in the sagittal plane, and 3-dimensional leg kinematics were filmed while the subjects pedaled at 60 RPM against a load of 150W. Compared to the controls, the ACL subjects had significant operated-thigh QF muscle weakness, less MI of the QF muscle, and the peak pedal z force occurred significantly later in the pedal revolution. Although operated-thigh QF muscle weakness was present, there was evidence that the muscle was not inhibited. Weakness of the QF muscle post-ACL reconstruction is likely multi-factorial, and thus presents a complicated rehabilitation problem.

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List of Abbreviations and Symbols

ACL	anterior cruciate ligament
ANOVA	one-way analysis of variance
avrMI	average muscle inhibition
BDC	bottom-dead-centre
BPTB	bone-patellar tendon-bone ACL reconstruction procedure
CKC	closed-kinetic-chain exercises
CI	confidence interval
cm	centimetres
CT	computed tomography
EMG	electromyogram
$\mathbf{F_{(lab)}z}$, $\mathbf{F_{(lab)}x}$	force of the foot on the pedal (lab coordinates)
$\mathbf{F_{(pedal)}z}$, $\mathbf{F_{(pedal)}x}$	force of the foot on the pedal (pedal coordinates)
$\mathbf{F_{(shank)}z}$, $\mathbf{F_{(shank)}x}$	tibiofemoral joint forces (shank coordinates)
Hz	Hertz
ICC	interclass correlation coefficient
ITT	interpolated twitch torque
JASA	Johnson Anti-Shear Accessory
LE	lower extremity
MI	muscle inhibition
mm	millimetres
MRI	magnetic resonance imaging

ms	milliseconds
MVC	maximal voluntary contraction
N	newtons
n	number of subjects
Nm/kg	torque per kilogram
Nm	torque
PD	percent deficit
kPa	kilopascals
QF	quadriceps femoris
ROM	range of motion
RPM	revolutions per minute
RTT	resting twitch torque
SD	standard deviation
TDC	top-dead-centre
V	Volts
W	Watts
°	degrees
°/s	degrees per second

1. INTRODUCTION

ACL injury in humans has been associated with decreased quadriceps femoris (QF) muscle strength, alterations in proprioception, and changes in gait patterns. At approximately six months after anterior cruciate ligament (ACL) reconstructive surgery, most patients have weakness of the operated thigh QF muscle despite the completion of a post-operative rehabilitation program (Synder-Mackler et al., 1994). At the present time, our knowledge of rehabilitation following ACL reconstruction has not been developed to the point where full strength of the QF muscle can be restored to the operated thigh (Lephart et al., 1993). To help elucidate this complicated rehabilitation problem, the following thesis tested some of the proposed theories of QF muscle weakness after ACL injury/reconstruction.

Numerous authors have attempted to quantify the incidence of ACL injury, both in normal and athletic populations. According to Jackson (1993), the ACL was stretched or torn in approximately 70% of serious knee injuries. Nielsen and Yde (1991) reviewed acute emergency room injuries within a city, and estimated the rate of ACL injury to be 0.3 per 1000 city inhabitants per year. Recent epidemiological studies show a trend towards female susceptibility to ACL injury. Arendt and Dick (1995) used the American National College Athletic Association Injury Surveillance System to determine knee injury patterns between males and females (per 1000 athlete exposures). The ACL injury rate in women's soccer (0.31) was more than double that of the men's game (0.13). For basketball, the women's rate of ACL injury (per 1000 athlete exposures) was 0.29 compared to 0.07 for the men.

It has been questioned whether reconstruction of the ACL is the treatment of choice for the ACL-deficient patient wishing to return to sport. Daniel et al. (1994) followed 292 patients who had sustained an acute, traumatic knee hemarthrosis for approximately 5 years post-injury. The authors determined that the ACL-deficient patients who continued to participate in sports with an unstable knee (defined as an injured minus normal knee difference in anterior tibial translation greater than 3mm with KT-1000 measurement) had a moderate to high risk of requiring surgical reconstruction. The above result may be an example of diagnostic suspicion bias; individuals who presented to the clinic with KT unstable knees may have been selected for surgery, despite their ability to continue in sport post-injury. Roos et al. (1995) compared questionnaire results from 778 elite Swedish soccer players (male and female) who had sustained a knee injury 7 years prior, to 180 non-elite players. The ACL-injured players were compared, and there was no difference in the rate of return to competitive soccer (which was approximately 20%) between the ACL-deficient and ACL-reconstructed players. Thus, reconstruction of the ACL did not appear to increase the longevity of sport participation for the study population post-ACL injury.

Once the decision to surgically reconstruct the ACL has been made, the choice of reconstructive procedure depends on the advantages and disadvantages of the particular technique, and the functional demands of the patient. Options for ACL graft material include autogenous tissue, allogenic tissue and synthetic materials. Presently, the most common surgical procedure is the bone-patellar tendon-bone (BPTB) autograft technique where the central third of the patellar tendon is excised from the ACL-deficient knee and

used to replace the torn ACL (Jackson, 1993). Advantages of the BPTB graft include the graft's bone plug insertions and high tensile strength (Lephart et al., 1993).

Rehabilitation following reconstruction of the ACL has been a much published and often controversial subject. Prior to 1980, the knee joint was typically immobilized for 6 weeks post-surgery to protect the graft and the graft insertions from excessive strain during the initial healing stages. Rehabilitation exercises for knee range of motion and strengthening followed; and patients returned to full activity about 1 year post-surgery, at a time when graft healing was thought to be complete. Surgical advances, including the use of patellar tendon grafts with bone-plug insertions, have shortened the post-operative time required for graft fixation (Noyes et al., 1983). On the basis of subjective and objective patient information, Shelbourne and Nitz (1990) introduced an accelerated rehabilitation program, which allowed patients to return to full activity in as little as 4 to 6 months post-surgery. It has been stated that use of an accelerated ACL rehabilitation program may allow early return of knee range of motion, prevention or reversal of severe muscle atrophy, and fewer patellofemoral joint symptoms (Shelbourne and Nitz, 1990). Accelerated rehabilitation has been used extensively despite the fact that the method evolved from clinical impressions, not from experimental evidence. At this time, there is no published evidence, in the form of a randomized controlled trial, to support accelerated versus traditional rehabilitation programs.

One of the primary goals of rehabilitation following ACL reconstruction is to return to functional activities (squat, climb stairs or run, for example) as early as possible without

comprising healing of the graft (DeMaio et al., 1992). A large part of the success of rehabilitation after ACL reconstruction may be associated with strength of the QF muscle. The literature, however, continues to document weakness of the QF muscle following ACL reconstruction (Seto et al., 1988, LoPresti et al., 1988; Yasuda et al., 1992; Maitland et al., 1993; Snyder-Mackler et al., 1994; Snyder-Mackler et al., 1995; Arangio et al., 1997; Pfeifer and Banzer, 1999). Long-term follow-up studies after ACL reconstruction report decreased QF muscle strength up to 7 years post-surgery. For example, Seto et al. (1988) examined strength (isokinetic QF muscle strength), knee stability, functional activity (survey), and sport participation levels in 25 individuals (15 with extraarticular and 10 with intraarticular procedures) 5 years after ACL reconstruction. The authors found a significant correlation between increased QF muscle strength and return to functional activities in the intraarticular group. Yasuda et al. (1992) assessed the post-operative isometric QF muscle strength of 65 patients 3 to 7 years following ACL reconstruction (patellar tendon autograft), and found the QF strength of the operated thigh was significantly less than that of the contralateral thigh. Considering the studies reported, bias may be introduced by patient attrition. For example, from the study of Yasuda et al. (1992), 65 patients were available for follow-up examination from an original sample population of 87 patients. Non-random sampling is also common in studies such as these to recruit an adequate sample size.

Weakness of the QF muscle may be accounted for by three mechanisms: decreased muscle cross-sectional area; decreased muscle activation; and/or altered knee joint mechanics. In the literature reviewed, several theories were proposed to explain QF

muscle weakness after ACL injury and reconstruction. For the purpose of this thesis, the theories have been categorized in the following manner:

1) **Atrophy of the QF muscle**

   a) Disuse atrophy
   b) Ineffective strengthening exercises
   c) Peri-operative tourniquet use
   d) Subconscious change in day-to-day behavior

2) **Inhibition of the QF muscle**

   a) Knee joint injury (including ACL injury)
   b) Anterior knee pain
   c) Knee effusion
   d) The effect of surgical technique on QF muscle strength
   e) Neural factors
   f) Loss of ACL mechanoreceptor input

3) **Altered knee joint mechanics**

   a) Knee laxity

The supporting literature for each theory will be summarized in the next chapter (Review of Literature). Theories 1(d) and 2 were tested in the thesis, and will be discussed at length in chapter two; the others have been included for continuity.

Some authors have suggested that an altered motor coordination strategy (or a subconscious change in day-to-day behavior) may be responsible for the reduced strength of the QF muscle post-ACL injury, especially during activities where use of the muscle may cause an anterior shear force of the tibia relative to the femur (Hogervorst and Brand, 1998; Solomonow et al., 1987; Lorentzon et al., 1989). After ACL injury and

reconstruction, changes to the kinetics and kinematics of gait have been documented. The predominant conclusion was that the extensor moment at the knee was reduced during stance (from 0 to 30° of knee flexion), and absent for some individuals (Andriacchi, 1990; Berchuk et al., 1990; Timoney et al., 1993; DeVita et al., 1997; De Vita et al., 1998; Wexler et al., 1998).

Presently, ACL rehabilitation programs include exercises for range of motion, strength, agility, endurance and balance. Stationary biking is included in these programs if the individual does not have significant patellofemoral irritation from pedaling. Also, the knee joint range of motion can be controlled through adjusting the seat height, increasing the bicycle's versatility as a rehabilitative tool (McLeod and Blackburn, 1980). Analytical knee models have shown that the tibiofemoral joint shear forces (anterior/posterior) were relatively small during cycling, suggesting that the ACL strain values were low (McLeod and Blackburn, 1980; Ericson and Nisell, 1986).

The biomechanics of cycling have been studied extensively. The use of an instrumented pedal has allowed the measurement of the pedal force on the cyclist's foot (Soden and Adeyela, 1979; Hull and Davis, 1981). The measured pedal forces and kinematic information from the motion of the lower extremities have been used to calculate the resultant moments at the ankle, knee and hip (Caldwell et al., 1999; Redfield and Hull, 1986). Electromyographical (EMG) analysis has also been used to examine the muscle activity patterns of the lower extremities during cycling (Hull and Jorge, 1985; Gregor et al., 1985).

In the cycling literature, the bicycle-rider system has been modeled as a closed five-bar linkage. The model assumes that the cycling motion occurs in the sagittal plane, that the hip is fixed, and that the knee joint cannot extend past 0°. Models such as these may be divided into two categories, those that consider the leg motion to occur predominantly in the sagittal plane, and those that include motion in the frontal plane. Sagittal plane models include those by Gregor et al. (1985), and Hull and Jorge (1985). Frontal plane models include those by Ericson et al. (1984) and Ruby et al. (1992).

To date, the majority of biomechanical research in cycling has focused on the optimization of elite cycling performance (bike geometry, foot-pedal interface, joint moment patterns, and muscle activation/coordination). Limited research has been conducted using patient populations. Seated ergometer pedaling has been investigated in hemiplegic (post-stroke) populations (Brown and Kautz, 1998). Pedaling a bicycle is task requiring both intra and interlimb muscular coordination to propel the crank through a constrained motion. Depending on the experimental outcome, movement speed may be controlled by the pedaling rate, and resistance may be incrementally applied. Since subjects are in a seated position while cycling, balance is not a factor in accomplishing the task, as in walking. Thus, bicycle pedaling is a motor task which is ideal for investigating the basic mechanisms of bipedal coordination, both in healthy and patient populations (Fregly and Zajac, 1996). If the muscular coordination of the lower extremities changes post-ACL reconstruction, investigation of the kinematics and kinetics of cycling may contribute both to the understanding of these changes, and to the post-operative prescription of cycling exercise.

The primary purpose of this research study was to examine theories of QF muscle weakness post-ACL reconstruction in two groups of subjects, a group of controls and a group of ACL-reconstructed individuals approximately 6 months post-surgery. Thus, to accomplish the primary purpose, the objectives of this case-controlled study were as follows:

1) Measure the isometric (65° knee angle) and isokinetic (at 90°/s and 240°/s) strength of the QF muscle.
2) Measure the muscle inhibition (MI) of the QF muscle (twitch interpolation test).
3) Measure the knee laxity (KT 2000 knee joint arthrometer).
4) Measure the bilateral kinematics and kinetics of cycling (pedal dynamometers and video motion analysis).

Following the introduction, chapter 2 will review the pertinent literature. Chapter 3 presents the testing methodology used in the study. The study results are presented in chapter 4 followed by a discussion of the results, sources of error and implications for further research in chapter 5.

2. REVIEW OF LITERATURE

2.1 Anatomy and Function

2.1.1 Anterior cruciate ligament

The cruciate ligaments (anterior and posterior) are named cruciate because they cross, and anterior/posterior from their tibial attachments. In humans, the ACL attaches medially to the anterior tibial intercondylar area, blending with the anterior cornu of the lateral meniscus. From its tibial attachment, the ligament ascends posterior-laterally, twisting and widening to attach to the posterior-medial aspect of the lateral femoral condyle (Gray, 1989). Butler et al. (1980) conducted anterior drawer tests in human cadaver specimens (at 30° and 90° of knee flexion). Restraining forces of the knee structures were measured before each ligament was cut. The ACL was the primary restraint to the anterior drawer motion of the tibia relative to the femur, and provided an average of 86% of the total resisting force (at 90° of knee flexion). All other ligaments and capsular structures provided the remaining, secondary restraints to anterior motion.

The posterior articular nerve is the major nerve to the ACL (Kennedy et al., 1982). The human ACL is thought to contain a small number of mechanoreceptors, which are defined as receptors that respond to mechanical pressure or distortion (Gray, 1989). Madey et al. (1997) stained the ACLs of five cats, and identified a range of between 5 and 17 ovoid nerve endings resembling Golgi tendon organs. The sensory endings described above were located throughout the ACL, in the subsynovial layers and between the collagen fibres. Schutte et al. (1987) examined the ACLs from 6 cadavers (obtained at autopsy). The ligaments were stained with gold-chloride and sectioned. Three

morphologically distinct mechanoreceptors were identified as Ruffini end-organs, Pacinian corpuscles and free nerve-endings. The free nerve-endings were theorized to function primarily as nociceptors. Some researchers have concluded that the mechanoreceptors found in the ACL may influence motor coordination and/or proprioception of the lower extremity, and that receptor loss may lead to dysfunction (Hogervorst and Brand, 1998).

2.1.2 Quadriceps femoris muscle group

The primary function of the quadriceps femoris muscle group is to extend the leg at the knee. The muscle group, which is located in the anterior compartment of the thigh, consists of four distinct parts: the rectus femoris muscle, located in the middle of the anterior thigh (also acts as a hip flexor); lateral is the vastus lateralis muscle; medial is the vastus medialis muscle; and between the two vastii is the vastus intermedius muscle. The QF muscle group is innervated by branches of the femoral nerve (Gray, 1989).

2.2 Theories of Quadriceps Femoris Weakness After ACL Injury/Reconstruction

2.2.1 Atrophy of the QF muscle

With injury to the ACL, a chronic pattern of atrophy and weakness of the QF muscle develops in most individuals. Studies investigating the amount of atrophy of the QF muscle following ACL injury have used either computed tomography (CT) or magnetic resonance imaging (MRI) to quantify the cross-sectional area of the muscle. Lorentzon et al. (1989) measured the thigh muscle cross-sectional area and strength of the QF muscle in 18 ACL-deficient males (range 7 months to 12 years post-injury). CT results showed a

mean 5% atrophy of the QF muscle of the ACL-deficient thigh compared to the non-injured side. Isokinetic testing of the QF muscle at 90°/s revealed a mean 25% strength difference between the injured and contralateral thighs. The authors found no correlation between QF muscle isokinetic performance and cross-sectional area, and concluded that non-optimal activation of the QF muscle was the most likely mechanism of the decreased strength measured in the study population. Lack of control of time elapsed between injury and testing may have confounded the results reported in the above study.

Gerber et al. (1985) also measured the cross-sectional area of the QF muscle in 41 ACL-deficient individuals (range 6 weeks to 10 years post-injury). CT scan results showed a mean 10% reduction in QF muscle cross-sectional area between the injured and non-injured thighs. Examination of biopsy specimens from the vastus lateralis of both lower extremities revealed a decrease in fibre size (not preferential to Type I or Type II fibres), and an increase in the intracellular fat content of the injured thigh QF muscle. No strength measures of the QF muscle were included in the above study.

To investigate the amount of QF atrophy following ACL reconstruction, Arangio et al. (1997) measured the cross-sectional area and strength of the thigh musculature in 33 patients (majority had iliotibial band autograft techniques, and were tested at a mean of 49 +/- 7 months post-surgery). The operated thigh QF muscle cross-sectional area (measured using MRI) was significantly decreased (8.6%) compared to the contralateral side. There was also a 10% deficit in strength of the operated thigh QF muscle with isokinetic knee extensor testing compared to the contralateral side. The authors found a

significant correlation between QF muscle cross-sectional area and strength of the QF muscle (in contrast to the previous study reported by Lorentzon et al., 1989).

Harder et al. (1990) measured the isokinetic QF muscle strength (120 °/s) in subjects pre and post-ACL reconstruction (n=46, BPTB and hamstring tendon autograft procedures), and found no significant reduction in the operated thigh QF muscle strength deficit (mean 14%) at 24 months post-surgery. The studies reported by Arangio et al. (1997) and Harder et al. (1990) support the hypothesis that reconstruction of the ACL does not significantly increase either the cross-sectional area of the QF muscle or the strength of the QF muscle. To the best of this author's knowledge, there are no prospective studies (pre and post-ACL reconstruction) quantifying QF muscle strength and atrophy in the literature reviewed. Prospective studies are essential for the understanding of the development of QF muscle atrophy and/or weakness post-ACL reconstruction.

2.2.1.1 Disuse atrophy

Prior to recent surgical and rehabilitation advances, disuse atrophy of the QF muscle (a reduction in muscle fibre size) occurred with the cast immobilization and limited weight bearing on the ACL-reconstructed limb. Häggmark and Eriksson (1979) randomized 16 subjects post-ACL reconstruction (BPTB autograft) into either cylinder cast or cast-brace (20° to 60° range of motion) immobilization for a 4 week period (both subject groups were permitted to fully bear weight on the operated lower extremity). Muscle biopsies were taken from the vastus lateralis muscle the day before surgery, and

after removal of the cast. The subjects in the cast-brace group had a significantly smaller reduction in Type I (slow twitch) muscle fibre size compared to the subjects with cylinder casts. Although the subjects in the cast-brace group returned to sporting activities earlier than the subjects in the cylinder cast group, there were no significant differences in the clinical assessment of knee joint stability between the two groups at 1 year post-surgery. Information regarding the strength of the QF muscle was not collected at the 1 year follow-up.

At the University of Calgary Sport Medicine Centre, ACL-reconstructed patients (post-BPTB autograft surgery) use crutches and a removable splint (to protect the operated lower extremity) for a brief period of time post-operatively (2-5 weeks). The patients are allowed to apply their bodyweight to the operated lower extremity as tolerable, with the aid of crutches. This period of relative immobilization may result in some disuse atrophy of the involved limb's musculature, but the atrophy should be minimized if it is mitigated solely by decreased use.

Studies using needle biopsy of the QF muscle have determined that the muscle wasting accompanying knee injury was predominantly due to atrophy of the muscle fibres, not due to a reduction in muscle fibre number (Stokes and Young, 1984). However, investigations have been inconclusive whether or not the atrophy of muscle fibres was preferential to Type I (slow twitch) or Type II (fast twitch) muscle fibres. Baugher et al. (1984) examined muscle biopsies from the vastus medialis muscle in 14 male subjects. The subjects were subdivided into 2 groups, group A with an acute ACL

injury (minimum interval from injury to surgery was 1 year) and group B with a chronic ACL injury (over 1 year post-injury). The authors found a statistically significant difference in the ratio of Type II to Type I muscle fibre area, and concluded that QF muscle atrophy in the chronic ACL-injured subject group was correlated with a relative decrease in Type II muscle fibre size. Lindboe and Patou (1982) examined biopsies from the vastus medialis muscle of the QF muscle group in 10 subjects following knee surgery (8 with menisectomies and 2 with incarceration of the subpatellar adipose tissue). Although the muscle fibres were atrophied, there was no statistically significant difference in fibre size between the Type I and Type II muscle fibre groups.

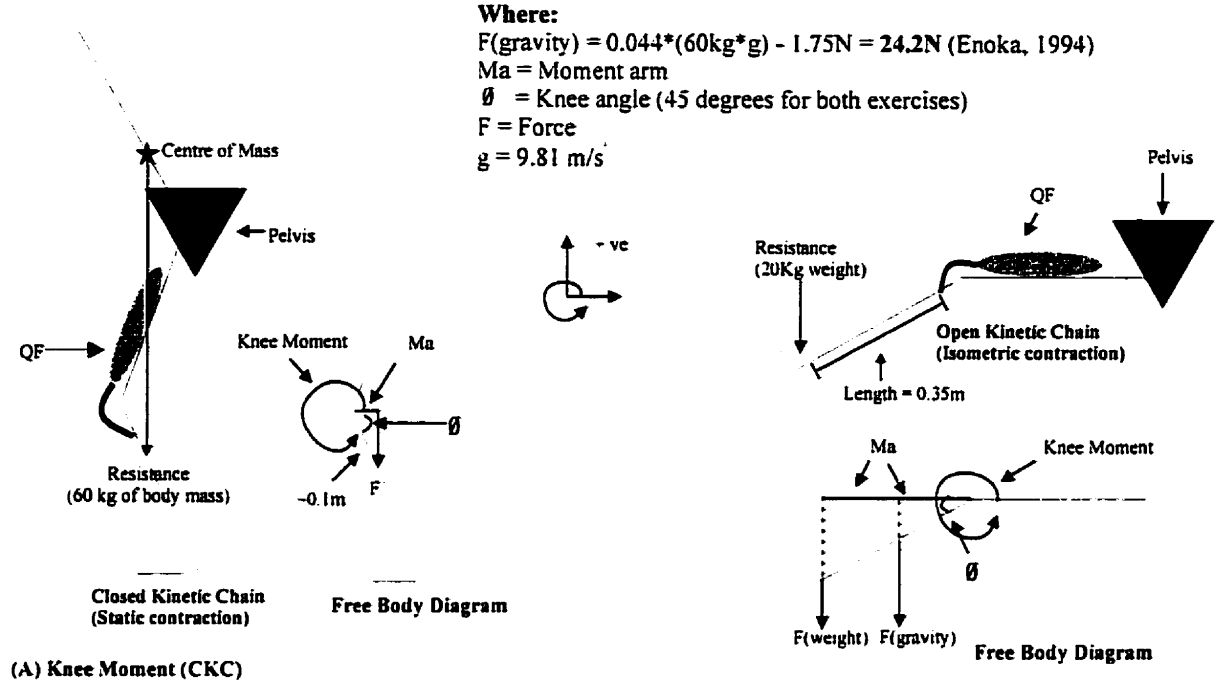
2.2.1.2 Ineffective strengthening exercises

Steindler (1955) was among the first to describe the mechanical behavior of the extremities as a kinetic chain. The following quote is from his original description:

> "A kinetic chain is a combination of several successively arranged joints constituting a complex motor unit. We designate as open kinetic chain a combination in which the terminal joint is free. A closed kinetic chain, on the other hand, is one in which the terminal joint meets with some considerable external resistance which prohibits or restrains its free motion."

Post-ACL reconstruction, the above terminology is used clinically to describe two subgroups of exercises (see Figure 2.1). For open kinetic chain (OKC) exercises, the muscles of the lower extremity are used to flex and extend the leg while the foot is free (resistance may be applied to the leg or foot). Closed kinetic chain (CKC) exercises require the foot to be fixed, and the lower extremity to move relative to the foot. However, post-ACL reconstruction, patients are typically instructed not to perform OKC

exercises to strengthen the QF muscle as the anterior drawer force of the tibia shearing forward relative to the femur may stretch the graft (Arms et al., 1984).



(A) Knee Moment (CKC)

= Force x Ma
= (60Kg x g) x (cos45 x 0.1m)
= 44.8 Nm

(B) Knee Moment (OKC)

Torque(knee) = Torque(gravity) + Torque(weight))
Torque(gravity) = [cos45*(0.433*.35m)] * (24.2N)
Torque(weight) = [cos45*(.35m)] * (20kg * g)

Torque(knee) = 2.6N + 48.6N
= 51.2 Nm

Figure 2.1: Calculation of resultant knee moment for of CKC (A) versus OKC (B) exercises using a simplified model.

Some authors have questioned whether CKC exercises are of sufficient intensity to provide a strengthening stimulus following ACL reconstruction. Snyder-Mackler et al. (1994) studied strength and inhibition of the QF muscle following ACL reconstruction. The authors questioned the efficacy of the CKC QF muscle strengthening exercises used in rehabilitation. Typically, only partial squats (0° to 45° of knee flexion) are prescribed

for the first 6-12 weeks post-ACL reconstruction, and they may provide a limited strengthening stimulus for the QF muscle. Figure 2.1 compared the resultant knee moments for a static CKC exercise (single leg squat at 45° knee flexion) and an OKC exercise (isometric knee extension at 45° of knee flexion with 20kg weight on the distal tibia). For the CKC exercise, the resultant extensor knee moment would be smaller at knee angles less than 45° of flexion, and if double leg support was used. For muscle fibre hypertrophy and strength increases, the muscle force developed must be above approximately 70% of the force of a maximal voluntary contraction (MVC) (Jones et al., 1989). In healthy muscle tissue, if the force threshold for muscle hypertrophy can be reached, strength of the involved QF muscle will increase. The converse is also true; if the force threshold for muscle hypertrophy cannot be reached, QF muscle strength may stay the same or decrease over time despite regular loading of the muscle through exercise. For example, if a subject's isometric knee extensor torque during a MVC (at 45° of knee flexion) was 100Nm, neither the CKC nor the OKC exercise modeled in Figure 2.1 would be adequate for muscle hypertrophy. Given the above argument, it seems likely that the present QF muscle strengthening regime (post-ACL reconstruction) is not adequate for increasing muscle strength and hypertrophy, especially during the initial stages of rehabilitation.

As mentioned above, OKC exercises for strengthening the QF muscle are usually not prescribed post-ACL reconstruction. To investigate the rationale for this clinical practice, the in-vivo strain of the ligament during rehabilitation exercises (CKC and OKC) has been measured. To estimate the in-vivo strain of an intact, healthy ACL,

Beynnon et al. (1997) inserted a strain-measuring device arthroscopically (using a local anesthetic) into the anteromedial bundle of the ACL in 8 subjects. The instrument was first calibrated by applying anterior/posterior loads of 100N to the knee in 20° of flexion. The resultant ACL strain was measured (2.6%). Next, the subjects performed OKC and CKC exercises of varying resistance. The maximum ACL strain values measured from CKC exercises (double leg squat, 3.6%) did not differ significantly from those measured with OKC exercises (knee extension with a 45 N boot, 3.8%). The authors suggested a posterior retrotilt of the tibial surface may cause the femur to slide posteriorly on the tibia with the application of a compressive force, thus increasing the strain of the ACL. The strain device only measured the anteromedial ACL bundle strain behavior; no information was collected from other regions of the ligament where strain may also have occurred with the exercises used in the study.

To investigate the strain behavior of the BPTB autograft in cadaver knees, Arms et al. (1984) used a strain transducer with a voltage output proportional to the strength of the magnetic field between the two ends of the transducer. The accuracy of this device was stated to be a 0.2% strain. After performing a BPTB ACL reconstruction on cadaver knees (n=21), the strain of the reconstruction was measured (using the transducer), and it was found to exhibit a strain behavior similar to the anteromedial bundle of the ACL.

2.2.1.3 Peri-operative tourniquet use

Use of the pneumatic tourniquet and the "bloodless" field for lower extremity surgery has been cited as a possible source of QF muscle weakness post-ACL reconstruction. Ischaemic damage to the muscle fibres with tourniquet use has been reported in the literature post-ACL reconstruction. Appell et al. (1993), using electron microscopy, examined muscle biopsies from the vastus lateralis muscle of 14 subjects who underwent ACL reconstruction (hamstring autograft procedure). A tourniquet was applied to the upper thigh at 400 mmHg, and muscle biopsies were sampled at 15, 30, 60, and 90 minutes after tourniquet application. Signs of fibre necrosis were found after 90 minutes of ischaemia, and the authors concluded that the tourniquet-induced QF muscle damage may represent an initial step towards atrophy of the QF muscle following ACL reconstruction.

2.2.1.4 Subconscious change in day-to-day behavior

It was established earlier in this chapter that some ACL-reconstructed subjects have residual weakness and atrophy of the QF muscle (up to 7 years post-surgery). If some ACL-reconstructed subjects consistently alter the kinematics and kinetics at the knee during gait and other day-to-day activities (climbing stairs for example), a relative disuse atrophy of the QF muscle may occur secondary to this subconscious change in day-to-day behavior.

A) Gait changes: in subjects with an ACL-deficient knee

Andriacchi (1990) examined the kinematic and kinetic patterns of gait in persons with ACL-deficient knees. During level walking, the subjects had a reduced knee extensor

moment in the stance phase when the knee was near full extension. The following hypothesis was generated: as the knee moves into extension during the stance phase of the gait cycle, contraction of the QF muscle will cause repetitive stimulation of the anterior knee joint capsule nerve afferents due to excessive anterior tibial translation. Over time, a central reprogramming of locomotion may occur with rupture of the ACL to prevent anterior tibial subluxation. Andriacchi coined this particular gait pattern "Quadriceps Avoidance", and it has since become a common descriptor of gait in ACL-injured persons, although not all subjects display the pattern when tested (Berchuck et al., 1990). Further studies of gait in ACL-deficient individuals have suggested that "Quadriceps Avoidance" may become more evident in persons with chronic ACL deficiency (Wexler et al., 1998). The conclusion that the decreased extensor torque was mitigated by reduced activation of the QF muscle may not be entirely correct. Co-contraction between the QF and hamstring muscle groups at the knee may also create this gait pattern, as the hamstrings contract to stabilize the tibia from excessive anterior tibial translation.

Some ACL-deficient individuals are able to tolerate activities as running to a stop without any symptoms of instability. During activities such as these, the moment at the knee was measured in ACL-deficient subjects, and the net extensor moment was decreased at the beginning of stance (compared to normal controls). It was theorized that the ACL-deficient individuals were using higher than normal hamstring muscle contraction to counteract any instability at the knee that may occur with the knee flexed (Berchuck et al., 1990).

Rudolph et al. (1998) examined the 3-dimensional kinetic and kinematic gait patterns of 16 ACL-deficient subjects, 8 subjects who had symptoms of instability in activities of daily living (non-coping subjects), and 8 subjects who had returned to pre-injury activity levels without symptoms of instability (coping subjects). There was a significant difference in knee kinematics between the two groups. The non-coping subjects landed at initial contact with significantly less flexion on the involved knee. There was also a significant, inverse correlation (for all subjects) between the QF muscle isometric strength deficit and the subjects' self-report of functional ability. The study populations tested may have represented distinct subgroups of ACL-deficient individuals because the subject sample was not chosen randomly, and the mean time intervals between injury and testing were 17 months for the non-coping subject group and 66 months for the coping subject group.

Kålund et al. (1990) measured the EMG of the QF and hamstring muscle groups of ACL-deficient subjects (n=9) and controls (n=9) walking on a treadmill at two separate speeds and inclines. On a level treadmill, there were no significant differences in muscle activity between the subjects and controls. However, when the incline was increased, the authors recorded a significantly earlier activation in the hamstring muscles of the ACL-deficient subjects. The authors concluded that the earlier activation of the hamstrings may help stabilize the knee for the increased load of uphill walking.

B) Gait changes: in subjects after ACL reconstruction

There is evidence to support the hypothesis that the kinematics and kinetics of gait are also not normal following ACL reconstruction (Timoney et al., 1993; DeVita et al., 1998; Snyder-Mackler et al., 1995). Since acute surgical reconstructions are rarely performed, most patients will have an ACL-deficient knee for a period of time before surgery. The consensus is that post-surgical gait alterations which persist may represent a learned muscular coordination strategy secondary to the primary ACL disruption, although this hypothesis has not been directly tested (Devita et al., 1997; Berchuck et al., 1990).

Patient gait adaptations following reconstruction of the ACL are less clear compared to individuals with ACL-deficient knees. The large differences in surgical and rehabilitation procedures may increase the variability of the results (DeVita et al., 1998). Post-operative symptoms such as muscle weakness, knee joint pain and/or knee joint instability may also affect an individual's gait pattern following ACL reconstruction. Timoney et al. (1993) analyzed the kinetics and kinematics of gait in ten subjects 8-12 months following ACL reconstruction. The subjects had a significant reduction in their midstance knee extensor moments compared to 10 controls. This gait pattern was slightly different from the "Quadriceps Avoidance" pattern described earlier by Andriacchi (1990) as there was a net extensor moment present at the knee. Unfortunately, no EMG information was collected to support this conclusion. Any changes in the timing of QF and/or hamstring muscle activation between the ACL-reconstructed and control group might help support or refute the authors' conclusions.

To study the development of gait changes post-ACL injury and reconstruction, Devita et al. (1997) examined the kinetics and kinematics of gait in 22 control subjects and 9 ACL-reconstructed subjects (BPTB autografts), 2 weeks post-injury (pre-surgery), and 3 and 5 weeks after surgery. The peak extensor moment at the knee was significantly less for the ACL-group at all times tested, and did not significantly change between the time intervals. The same group of ACL subjects were re-tested at 6 months post-surgery, and the results were similar (Devita et al., 1998). Restoring the stability of the knee through reconstruction of the ACL did not restore the gait patterns of the ACL-injured subjects to that of the controls. Given the study results, the authors suggested that loss of the ACL, and loss of its mechanoreceptor input, may precipitate some of the gait changes observed in individuals with ACL-injured and reconstructed knees.

2.2.2 Inhibition of the quadriceps femoris muscle

A portion of QF muscle weakness following ACL reconstruction may be related to the body's inability to fully activate the available motor units. This phenomenon is known as muscle inhibition (MI) (Hurley et al., 1994). In the literature, there are numerous studies which have associated QF MI with factors such as joint injury, knee pain (including patellofemoral pain), knee joint effusion, and/or neural factors (Spencer et al., 1984; Elmqvist et al., 1988; Fahrer et al., 1988; Newham et al., 1989; Hurley et al., 1992; Hurley et al., 1994; Snyder-Mackler et al., 1994; Leroux et al., 1995; Suter et al., 1998a; Suter et al., 1998b; Pfeifer and Banzer, 1999). According to Hurley (1997), MI prevents maximal force generation, and if prolonged, may result in muscle fibre atrophy. The neurological mechanism of MI has been described as reflexive in nature (Stokes and

Young, 1984). Although the actual neurological pathway remains unknown, it has been hypothesized that abnormal afferent information (from pain, joint effusion, and/or joint pathology, for example) may (through inhibitory pathways) modulate the efferent alpha motor neuron signal, and reduce the activation of the motor units used for muscle contraction (Hurley, 1997). With regard to ACL injury and/or reconstruction, potential mechanisms precipitating inhibition of the QF muscle have been investigated and are as follows: knee joint injury; anterior knee pain; knee effusion; BPTB autograft surgical technique; neural factors; and loss of the ACL mechanoreceptor input.

2.2.2.1 Knee joint injury (including ACL injury)

Following rupture of the ACL, individuals may have a large reduction in QF muscle activation, and will likely respond poorly to rehabilitation if they are unable to fully recruit and strengthen the muscle fibres of the QF to help stabilize the injured knee (Snyder-Mackler et al., 1994). Hurley et al. (1994) measured the inhibition and strength of the QF muscle in 8 male ACL-deficient patients (with concurrent, extensive knee trauma at injury) before and after a period of intensive rehabilitation (5 hours per day, 5 days per week for one month). The mean isometric strength deficit (non-injured – injured thigh) was significantly increased (40.5% to 45.5%) for the operated thigh group. The authors found no statistically significant change in the amount of MI even though the mean percent MI of the QF muscle in the operated thigh group decreased from 45.6% to 28.5%. Although clinically it appears that MI decreased with the rehabilitation, the relatively small sample size (n=8) and increased sample variability may have reduced the power of the study to detect a significant difference. Suter et al. (1998a) investigated the

level of inhibition of the QF muscle in persons with ACL deficiency (n=12), and found MI to be present in both the affected leg (38%) and the contralateral leg (37%). Contralateral leg inhibition may be a result of bilateral convergence of the afferent information at the spinal cord level; thus, if one of the causes of MI is abnormal afferent information, incoming signals may be reflexively interpreted as bilateral leading to inhibition of the QF muscle in both legs (Hurley, 1997).

2.2.2.2 Anterior knee pain

Suter et al. (1998b) included such disorders as patellofemoral dysfunction, chondral and osteochondral lesions, tendinitis, bursitis, synovitis and/or meniscal tears in the definition of anterior knee pain. Post-ACL reconstruction, anterior knee pain may also be a complaint (Marder et al., 1991). Marder et al. (1991) conducted a prospective study of BPTB autograft versus hamstring autograft ACL reconstruction patients (n=80). Two years after ACL reconstruction, 24% of the patients experienced anterior knee pain, and there were no differences between the two operative techniques within this subgroup. To determine whether MI of the QF muscle was a component of anterior knee pain, Suter et al. (1998b) measured the amount of MI in 25 patients with anterior knee pain pre and post-arthroscopy (at 6 weeks and 6 months post-surgery). Using a twitch interpolation technique, the authors found a persistent MI in both the affected and contralateral QF muscle.

2.2.2.3 Knee effusion

Knee effusion or swelling has been associated with atrophy of the QF muscle (Spencer et al. 1984; Fahrer et al. 1988). It was theorized that a knee effusion inhibits the QF muscle through a reflexive pathway, resulting in decreased muscle activation and/or disuse atrophy. Spencer et al. (1984) injected saline (up to 60 ml) into the healthy knee joints of ten subjects, and measured the inhibition of the QF muscle motorneuron pool by recording the Hoffmann (H) reflexes. All subjects displayed a significant reduction in the H-reflex amplitude following the introduction of the saline. The authors concluded that introduction of an experimental, painless knee joint effusion lead to inhibition of the QF muscle. Fahrer et al. (1988) investigated QF isometric muscle strength and activation (surface integrated EMG) in 13 patients with chronic knee joint effusions. After QF strength and activation were measured, 25 to 110 ml of fluid was aspirated from the knee joint, and a second set of measurements were taken. The authors reported a significant increase in QF muscle strength and activation post-aspiration.

2.2.2.4 The effect of surgical technique on QF muscle strength and inhibition

Harvesting the central third of the patellar tendon disrupts the knee extensor mechanism; this may be associated with QF muscle weakness and inhibition following ACL reconstruction. However, the literature is inconclusive as to whether the strength of the QF muscle varies with the surgical procedure used. Rosenberg et al. (1992) selected 10 individuals 12 to 24 months post-reconstruction (BPTB autografts). Isokinetic knee extensor testing at 60°/s showed an average QF muscle operated thigh deficit of 18% compared to the contralateral thigh. CT revealed a significant decrease in the operated

thigh QF muscle cross-sectional area (13%) compared to the contralateral thigh, and MRI confirmed persistent defects at the ACL graft harvest site.

Other authors have compared the post-operative QF muscle strength of patients following ACL reconstruction (BPTB autograft) to patients with other surgical graft techniques. Lephart et al. (1993) compared the isokinetic QF muscle strength of 33 active males at 12 to 24 months post-reconstruction. The subjects were separated (non-randomly) into two groups; one with BPTB autografts (n=15) and the other with BPTB allografts (n=18). Post-operatively, all subjects completed a rehabilitation program at the same institution. No significant differences in QF muscle strength were found between the two groups. Bias may have been introduced to the study with the non-random assignment of surgical technique. Sachs et al. (1989) reviewed follow-up (one year post-op) information from 126 patients who underwent reconstruction of the ACL (BPTB, hamstring or iliotibial band autografts). The subjects completed a "traditional" rehabilitation program post-operatively including 6-8 weeks of crutch use. When the subjects were stratified for type of operation, the mean QF muscle strength deficit (between-thigh) was significantly greater for the BPTB autograft subjects compared to the hamstring autograft subjects.

It has been theorized that the BPTB autograft surgical technique may lead to an increase in patellofemoral joint symptoms such as pain, effusion and/or crepitus post-surgery (Sachs et al., 1989). Patellar tendon shortening has been reported post-BPTB ACL reconstruction (Breitfuss et al., 1996). Breitfuss et al. (1996) performed a

retrospective clinical and radiographic examination of the patellofemoral joint in 41 patients approximately 2 years after ACL reconstruction. In 73% of the patients, radiographs showed patellar tendon shortening, despite the fact that 80% of the patients self-reported good to very good results. Flexion contracture after reconstruction of the ACL may also lead to patellofemoral joint pain. Sachs et al. (1989) reassessed 126 ACL-reconstructed subjects one year post-surgery. Knee flexion contractures of 5° or greater were present in 24% of the patients. Also, flexion contractures correlated positively with patellofemoral joint pain and QF muscle weakness (defined as a between-thigh strength deficit of 20% or more with isokinetic knee extension at 60°/s).

2.2.2.5 Neural factors

For ACL-deficient individuals, some authors have postulated that exercises used to strengthen the QF muscle subsequently inhibit the involved muscle (Snyder-Mackler et al., 1994; Lorentzon et al., 1989). In the ACL-deficient knee or ACL-reconstructed knee with anterior knee joint laxity, contraction of the QF muscle from 60° flexion to full extension may create an anterior drawer force on the tibia (relative to the femur), and may stretch the anterior knee joint capsule (since the torn ACL was the primary restraint to anterior drawer motion) (Butler et al., 1980). This is thought to inhibit the QF muscle through a reflexive pathway; the abnormal afferent information from excessive stretch of the anterior knee joint capsule may decrease the efferent motor input to the QF muscle (Snyder-Mackler et al., 1994). There is evidence against this theory. Animal studies have shown a lack of convincing support for the existence of a direct stretch reflex loop from the mechanoreceptors of the joint capsule to the alpha motor neurons of the thigh

muscles (Johansson et al., 1990). Also, investigations measuring MI in the QF muscle post-ACL reconstruction have either shown no significant MI (Snyder-Mackler et al., 1994; Pfeifer and Banzer, 1999) or a small amount of MI (Suter et al., 1999).

A primary goal of reconstruction of the ACL is to improve the anterior/posterior stability of the knee. By reducing the anterior drawer of the tibia relative to the femur, this in turn may decrease the level of MI of the QF muscle and improve the patient's ability to strengthen the weakened muscle. To investigate the relation of QF muscle strength and MI, Pfeifer and Banzer (1999) measured the isometric knee extensor strength, knee laxity (KT-1000 joint arthrometry), and MI of the QF muscle (twitch interpolation technique) in 20 healthy controls and 39 subjects (arthroscopic BPTB autograft technique, 10-16 months post-surgery). Although the ACL-reconstructed subjects had a 21% mean deficit in isometric strength of the QF muscle, there were no significant MI of the QF muscle or knee laxity for the reconstructed group. The authors concluded that insufficient rehabilitation strengthening exercises may be responsible for the inability of the ACL-reconstructed subjects to regain the QF muscle mass lost during post-surgical immobilization. The current study, by investigating similar variables to Pfeifer and Banzer (1999), should further elucidate the relation between QF muscle strength, QF MI and knee laxity post-ACL reconstruction.

2.2.2.6 Loss of ACL mechanoreceptor input

Changes in muscle coordination and/or muscle recruitment strategies following loss of the ACL, resulting in decreased activation of the QF muscle, have been reported in the literature (Wexler et al., 1998; Berchuck et al., 1990). The source of these changes is unknown, but some authors question whether the changes are precipitated by loss of the mechanoreceptor input from the torn ACL, and/or altered stimulation of the remaining knee articular sensory afferents (Hogervorst and Brand, 1998).

Some authors have theorized that thigh muscle activation patterns may be influenced by mechanoreceptor feedback from the ACL. Solomonow et al. (1987) inserted a steel wire around the ACLs of six cat hindlimbs (externally stabilized with pins) and, by creating a strain force on the ACL, found a significant increase in the indwelling EMG recorded in the hamstring muscle group. From the results of the experiment, Solomonow concluded there was evidence for a direct reflex arc between the ACL and the hamstring muscles. The reflexive effect produced in this study has been questioned due to the high ligament loads required (130 to 150 N) for a hamstring muscle response (Hogervorst and Brand, 1998).

In a similar cat model, Johansson et al. (1990) attached electromagnetic pullers to the hindlimb knee flexor muscles to stimulate fusimotor neuron activity with sinusoidal stretching. While monitoring the response of the muscle spindle afferents of the knee flexor muscles, traction forces (5 to 70 N) were applied to the intact ACL of the cats. With a concurrent ACL strain, a significant change was noted in the sensitivity of the

muscle spindle afferents (which signal the change in length of the muscle spindles) to the sinusoidal stretching. The authors concluded that an increased ACL load may influence the muscles surrounding the knee joint by altering the muscle stiffness. Control experiments showed a disappearance of the muscle spindle afferent effect with the application of load to the posterior cruciate ligament following transection of the posterior articular nerve (Sojka et al., 1989). Activation of the fusimotor system through such a reflex may have a modulating or indirect effect on the alpha motor nerves to the knee flexor muscles. Thus, a fusimotor system response likely negates a protective, reflex contraction of the hamstring muscle group with sudden anterior shear of the tibia relative to the femur.

It has not been established whether or not there is a gain in the mechanoreceptor feedback in other, intact periarticular structures, such as the joint capsule, to compensate for the loss of the ACL (Hogervorst and Brand, 1998). It appears that acute transection of the feline ACL does not change the response of the articular knee joint afferents to mechanical stimuli. Khalsa and Grigg (1996) measured the responsiveness (activation threshold and position sensitivity) of single, knee joint capsule afferents from the knees of 9 cats. In each cat, the knee joint was rotated using angular displacements before and after the ACL was transected. The responsiveness of the capsule afferents was not significantly changed after cutting the ligament.

2.2.3 Altered knee joint mechanics

In the literature reviewed, it has been suggested that differences in the measured knee extensor torque between ACL-intact and ACL-deficient knees may be partially due to a change in the moment arm of the QF muscle, not due to a reduction in the strength of the QF muscle. Lorentzon et al. (1989), after studying the size, morphology, and strength of the QF in ACL-deficient males, concluded that anterior displacement of the tibia during knee extension exercise may decrease the moment arm of the QF muscle, and thus decrease the torque measured from the QF muscle. To study the ligament and extensor mechanism function in the ACL-deficient knee, Pandy and Shelburne (1998) developed a 2-dimensional model of the knee to simulate anterior drawer of the tibia relative to the femur during isometric knee extension exercise. The authors found that the moment arm of the extensor mechanism and the extensor torque at the knee were equal between the ACL-intact and ACL-deficient models, and concluded that differences in measured knee extensor torque between ACL-intact and ACL-deficient subjects were likely due to a deficit in QF muscle strength.

2.3 Measurement Techniques

2.3.1 Assessment of QF muscle strength

Two advantages to using isokinetic dynamometry for the assessment of muscle strength are that it permits isolation of the muscle group tested and that strength can be measured through the muscle group's ROM. With isokinetic knee extensor testing or exercise, resultant forces on the ACL graft and the potential for graft injury are difficult to predict. Kaufman et al. (1991) calculated dynamic knee joint forces using experimental data from concentric, isokinetic knee extension. A triaxial goniometer was

attached to the right knee of 5 male subjects, and was used to collect 3-dimensional knee angular displacement with isokinetic knee extension exercise (at 60°/s and 180°/s). The maximal anterior shear force of the tibia relative to the femur, after being normalized to each subject's body mass, was compared to other rehabilitation exercises. The authors concluded that the measured force was approximately 5 to 6 times greater than the measured anterior shear force of the tibia at the knee during cycling.

The Johnson Anti-Shear Accessory® (JASA), is a Cybex® (Cybex, Lumex Inc., Ronkonkoma, NY) attachment with a proximal resistance pad for the shank. The device was designed (from calculations) to decrease the anterior shear force of the tibia during isokinetic testing (Johnson, 1982). Nisell et al. (1989) used a sagittal plane mathematical model to calculate the magnitude of tibiofemoral joint compressive and shear forces during isokinetic knee extension exercise. Two different speeds were used (30°/s and 180°/s), and the resistance pad was tested in both a proximal and distal position. The authors concluded the anterior shear force of the tibia was reduced significantly when the resistant pad was placed on the proximal tibia. The JASA accessory has also been investigated for validity as compared to the regular Cybex arm (Timm, 1986). A correlation coefficient was calculated for the resultant QF muscle torque between the JASA and the regular Cybex arm, and the r value was 0.97 (Timm, 1986). Pincivero et al. (1997) examined the intratester reliability of isokinetic dynamometry, and concluded that with adequate system calibration, gravity correction and standardization of patient position, the reliability of isokinetic peak torque measures was relatively high (test-retest

coefficient of r=0.88 to r=0.97). Table 2.1 summarizes some of the isokinetic and isometric torque values recorded for subjects following ACL reconstruction.

Table 2.1: Documented values of isokinetic quadriceps femoris muscle strength in subjects following ACL reconstruction

Authors	Speed	Sample Size	Months Post-Surgery	Mean Strength Deficit (Nonop. – operated leg)
Sachs et al., (1989)	60°/s	126	12	34%
Shelbourne and Nitz (1990)	180 °/s	247	7-10	17%
Yasuda et al., (1992)	Isometric	29	12	22%
Rosenberg et al., (1992)	60 °/s	10	12-24	18%
Maitland et al., (1993)	60°/s	24	5-10	31%
	150 °/s	24	5-10	23%
	240 °/s	24	5-10	16%
Pfeifer and Banzer (1999)	Isometric	39	10-16	21%

2.3.2 The twitch interpolation technique

The twitch interpolation technique involves applying a brief, percutaneous stimulation to the peripheral motor nerve (or muscle) during a MVC, and comparing any additional torque produced to the torque produced during a resting twitch. The technique has been used in the literature to estimate the level of MI of the QF muscle (Rutherford et al.,

1986; Allen et al., 1995; Behm et al., 1996; Suter et al., 1996; Suter et al., 1998a; Suter et al., 1998b; Pfeifer and Banzer, 1999). The measured torque above the MVC is assumed to represent motor units not previously activated by volition or an increase in the firing frequency of any submaximally activated motor units. The percentage of MI is calculated as the interpolated twitch torque (ITT) divided by the resting twitch torque (RTT), and multiplied by 100% (Hurley et al., 1992). Other techniques have been reported in the literature to measure the amount of MI of the QF muscle including the superimpostition burst technique (Snyder-Mackler et al., 1994), the H-reflex amplitude (Spencer et al., 1984), and the integrated EMG (Fahrer et al., 1988).

The relatively small amplitude of the ITT is difficult to measure when the signal is superimposed on the waveform of torque created with a MVC. Thus, the sensitivity of the measurement may be diminished (Behm et al., 1996). It has been estimated that use of an amplifier to enlarge and isolate the ITT allows measurements of less than 0.5% of the total torque. By improving the resolution of the measurement, small increments of torque may be detected. Using this method of signal processing, it has been shown that even highly trained subjects often fail to achieve the maximal level of voluntary activation (Allen et al., 1995).

Behm et al. (1996) investigated the sensitivity of the twitch interpolation technique for the plantarflexor and QF muscle groups. The authors compared a variety of conditions including submaximal voluntary contractions, potentiated resting twitches and multiple twitches (up to quintuplets). Methods which improved the sensitivity of the twitch

interpolation technique included the use of a doublet twitch to summate the imposed torque (improved the signal-to-noise ratio) and the use of a potentiated resting twitch (since all interpolated twitches are potentiated).

To investigate the reliability of the twitch interpolation technique, Allen et al. (1995) measured the maximal voluntary activation of the biceps brachii muscle in 5 subjects on 5 different days, and calculated an interclass correlation coefficient (ICC) of 0.97 (representative of intratester reliability).

The validity of twitch interpolation has also been investigated in the literature. Suter et al. (1996a) used a twitch interpolation technique to measure MI of the QF muscle in 20 healthy subjects. The subjects performed 20 QF muscle contractions ranging from about 5-10% of MVC to MVC while measurements of ITT were taken for each contraction. The authors found that a negative relation existed between the strength of contraction and the ITT. A second-order polynomial regression line fitted through all data points gave a coefficient of determination (representative of validity in the study) of r^2=0.777. Refer to Table 2.2 for a summary of results from interpolated twitch testing of the QF muscle reported in the literature.

Table 2.2: Reported values of quadriceps femoris muscle inhibition measured by the interpolated twitch test

Authors	Knee Angle	Twitch Type	# of Subjects & Condition	Percent Inhibition (mean)
Newham et al. (1989)	~90°	singlet	**11** ACL-deficient	**25.3%** (injured) **5.2%** (contralateral)
Hurley et al. (1992)	~90°	singlet	**10** ACL-deficient	**9.5%** (injured) **8.7%** (contralateral)
Suter et al. (1996a)	90°	singlet	**20** controls	**4.3%**
Suter et al. (1996b)	60°	doublet (potentiated)	**10** controls	**12.8%**
Suter et al. (1998b)	60°	singlet	**30** anterior knee pain	**37.5%** (pre-op) **32%** (6 months post-op)
Huber et al. (1998)	60°	singlet	**13** some with knee injury	**21.5%** (no injury) **9%** (prior injury)
Suter et al. (1998a)	30° (JASA)	doublet (potentiated)	**12** ACL-deficient	**38%** (injured) **37%** (contralateral)
Suter et al. (1999)	30° (JASA)	doublet (potentiated)	**22** ACL-reconstructed	**19.5%** (reconstructed) **18%** (contralateral)

2.3.3 Knee laxity measurement

Traditionally, the anterior laxity at the knee has been assessed by either the anterior drawer and/or Lachman tests. The KT 1000 and KT 2000 (MEDmetric, San Diego, CA) are commercial devices designed to quantify anterior knee joint laxity. Stratford et al. (1991) studied the sensitivity of the KT 1000 joint arthrometer, and concluded the responsiveness (sensitivity to change) of the arthrometer test was related to the magnitude

of the displacement force. Thus, the total displacement in mm was measured with the application of 135N of force for all subjects tested in the present study. Brosky et al. (1999) measured the intratester reliability of the KT 1000 following ACL reconstruction, and found an ICC of 0.91 to 0.93 for the reconstructed limbs tested. The ICC's reported for the contralateral limbs tested were considerably lower (0.69 to 0.72). Myrer et al. (1996) assessed the intertester reliability of the KT 2000 joint arthrometer in 30 healthy controls, and reported an ICC of 0.58 for side-to-side differences in anterior knee joint laxity. Subject-to-subject variability needs to be accounted for when interpreting ICC values; there is usually less between-subject variability in the side-to-side difference of anterior laxity when compared to anterior laxity (mm) measurements.

2.4 The Biomechanics of Cycling

2.4.1 Cycling kinematics

To examine the kinematics of the lower extremities during cycling, the leg-bicycle system has been modeled as a closed, planar five-bar linkage with the frame as the fixed link (Hull and Jorge, 1985; Redfield and Hull, 1986; Ruby et al., 1992). To develop the model, three kinematic inputs and one geometric constraint were needed. The kinematic inputs were the crank and pedal angles (recorded with continuous rotation potentiometers), and the relative angular velocity of the pedal angle (approximated with a sine function). For the geometric constraint, the knee was not allowed to extend past 0° (Hull and Jorge, 1985).

The kinematics of cycling are principally affected by cadence, rider-bicycle geometry, hip motion and ankling pattern (pattern of range of motion at the ankle joint to improve

the efficiency of the torque produced at the crank) (Gregor et al., 1991). Nordeen and Cavanagh (1975) examined the kinematics of the lower extremities during cycling at different seat heights. The authors found that within a reasonable range, the seat height had a minimal effect on the pattern of foot movement. For the knee and thigh angles, however, a 4.4 cm seat height increased the hip and knee range of motion (ROM) by approximately 15°, and increased the maximum knee and hip flexion angles by approximately 5°. Nordeen-Synder (1977) studied the effect of bicycle seat height variation (95, 100, and 105% of trochanteric height) upon lower limb kinematics. The author found that the majority of adaptations to seat height increases occur with the knee and ankle angles.

2.4.2 Measurement of pedal forces

A detailed knowledge of the pedal loading is crucial to complete a kinetic examination of the knee during cycling. Previous investigators have developed instrumentation to measure the forces applied to the pedals. Prior to 1980, dynamometer measurements were limited to the sagittal plane pedal forces ($F_{(pedal)}x$ and $F_{(pedal)}z$ as per Figure 2.3) (Soden and Adeyefa, 1979). Davis and Hull (1981) constructed a six-load component pedal dynamometer that allowed measurement of the $F_{(pedal)}z$, $F_{(pedal)}x$, and $F_{(pedal)}y$ loads. Using the dynamometer, the authors used 3 pedaling conditions to investigate the foot-pedal connection: a soft-sole shoe; a soft-sole shoe with toeclip; and a rigid shoe with cleat and toeclip. Use of the cleated shoe improved the pedaling efficiency (defined as how well the cyclist used muscular exertion to power the crank), and also created larger negative ($F_{(pedal)}x$) loads during the backstroke. The authors concluded that addition of cleats to the foot-pedal connection may allow enhanced activity of the flexor muscle groups, although specific muscle group contributions were not estimated. In the current study, all participants wore running shoes and used a toeclip to secure the foot on the pedal (no cleated foot-pedal connection).

Davis and Hull (1981) also investigated the relative load and moment contributions between $F_{(pedal)}z$, $F_{(pedal)}x$ and $F_{(pedal)}y$. The authors concluded that the $F_{(pedal)}y$ force and resultant moment were in phase with the $F_{(pedal)}z$ force. For example, when the pedal moves from the top-dead-centre to the bottom-dead-centre position, the $F_{(pedal)}z$ force reaches a maximum level. Concurrently, the $F_{(pedal)}y$ force peaks during this portion of the pedal cycle as the foot pushes outward on the pedal. The measured $F_{(pedal)}y$ force component was of comparable magnitude to the $F_{(pedal)}x$ component; thus, the authors concluded that out of plane forces are significant, and measures to decrease such forces may be beneficial to prevent overuse injury at the knee. Unfortunately, the six-load dynamometer was difficult to calibrate and was thought to introduce error through cross-sensitivity between the loads. For the purpose of the current study, pedal dynamometers measuring the $F_{(pedal)}z$ and $F_{(pedal)}x$ loads were used for a sagittal plane analysis of the pedal forces. Motion at the knee in the frontal plane was also examined to help quantify the amount of out-of-plane motion occurring. Refer to Figure 2.2 for a pictorial description of the pedal cycle.

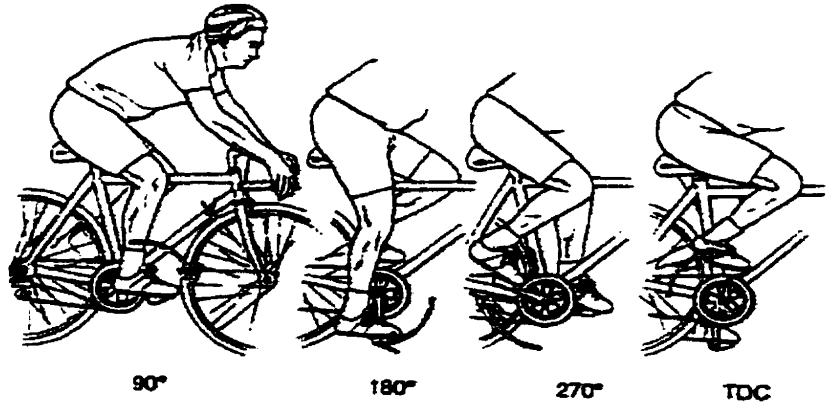


Figure 2.2: Terminology used to describe a pedal revolution. TDC refers to top-dead-centre, and 180° is equivalent to bottom-dead-centre (BDC) (Adapted from Hamill and Knutzen, 1995).

Ericson and Nisell (1986) investigated the tibiofemoral joint forces during pedaling in 6 subjects at 2 different pedal-foot positions. The first position was defined as anterior and described as the centre of the pedal aligned with the head of the second metatarsal. The second position (defined as posterior) was 10 cm posterior from the first position (pedal spindle aligned with the medial arch of the foot). The positive tibiofemoral joint force in the x direction (equated with anterior shear force of the tibia on the femur in the study) was significantly increased with the use of the posterior foot position. The authors did not discuss the foot-pedal connection for either of the 2 positions. Whether cleats and/or toe clips were used may alter the results and conclusions drawn from the study. In the current study, only one size of toeclip was used for all the study participants. Depending on differences in the size of the subjects' feet, the position of the foot on the pedal may have varied among the subjects.

For the purpose of this study, the pedal used was designed in the following manner. The pedal spindle was instrumented with eight stain gauges (offset by 90° intervals), connected with two Wheatstone bridge circuits. The location and interconnection of the strain gauges renders the dynamometer insensitive to the location of the applied pedal forces. The strain gauge design also minimizes the sensitivity of the pedal to the moments about the axis of the pedal forces, and the third force component, $F_{(pedal)y}$ (Rowe et al., 1998). Figure 2.3 is a diagram of the pedal including the pedal and lab coordinate systems used in this study.

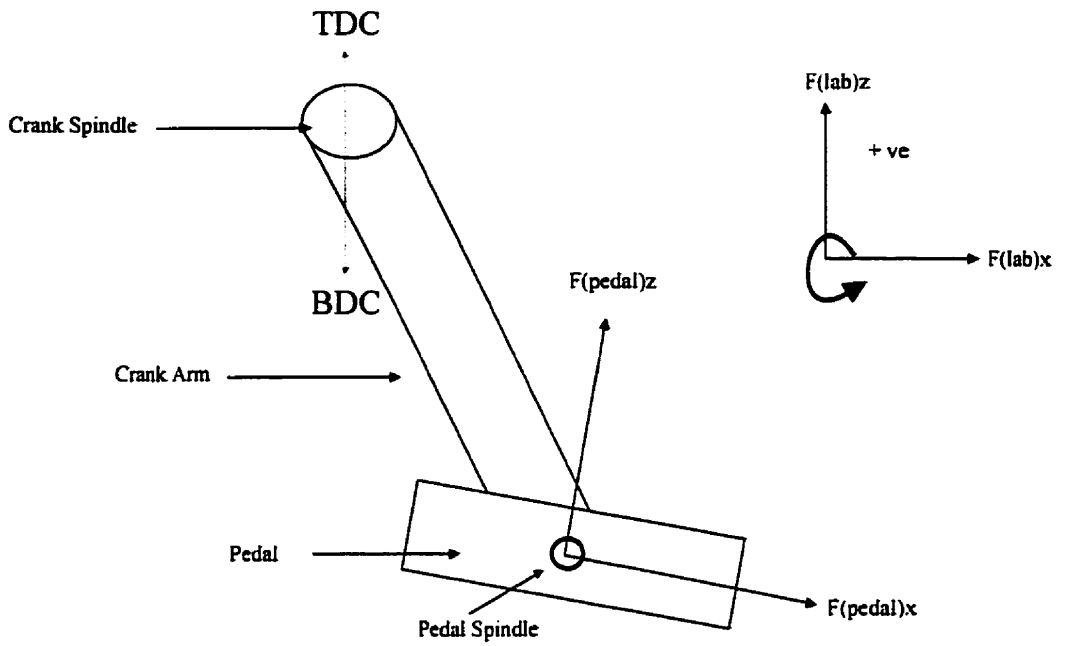


Figure 2.3: Diagram of pedal including the sagittal plane pedal coordinate system used to collect the pedal force data and the lab coordinate system (upper right-hand corner) used for the inverse dynamic calculations .

2.4.3 The moment about the knee during cycling

Gregor et al. (1985) provided a detailed description of the kinematics, kinetics and EMG activity about the knee during cycling in 5 male subjects (elite cyclists). The authors concluded that the net knee moment during the pedal cycle was sinusoidal in shape and the amount of extensor torque was greater than the flexor torque. From TDC to approximately 150°, the net moment at the knee was extensor with high QF muscle activity (measured with surface EMG) from TDC to 90°. The net knee moment changed to flexor at about 150° from the TDC position.

Hull and Jorge (1986) recorded sagittal plane pedal forces, crank and pedal angles (measured with a potentiometer), and surface EMG (from 8 leg muscles) during pedaling

in 3 subjects. The total joint moments were separated into the moments due to motion only (kinematic), and the moments due to the pedal forces (static). At high pedaling rates (greater than 130 RPM), the kinematic moment contributed more to the total joint moment whereas at low pedaling rates (less than 70 RPM), the static moment contributed more to the total joint moment.

The motion occurring at the knee from 90° to 180° of a crank cycle - knee extension during an internal knee flexion moment - has been compared to that described by Lombard (1903), and has been discussed in the literature as an example of Lombard's Paradox (Andrews, 1987; Gregor et al., 1985). The activity of a two-joint muscle when the required moment or motion at one of the joints is in the opposite direction to action of the muscle has been called Lombard's Paradox (Lombard, 1903). In Gregor et al. (1985), the mean joint torque patterns of the knee and hip were compared to the integrated average EMG pattern of the knee flexor and extensor muscles. The lack of knee extensor muscle activity while the knee was extending (from 90° to BDC) lead the authors to conclude that the knee flexors were eccentrically controlling extension at the knee, thus providing a creative solution to Lombard's Paradox.

2.4.4 Strain of the ACL during pedaling

Fleming et al. (1998) used a device (differential variable reluctance transducer) to measure the in-vivo strain behavior of the ACL during bicycle pedaling. During cycling, the amount of ligament strain was relatively low (~1.7%) compared to other rehabilitation activities such as squatting (3.6%) or isometric QF contraction at 15° of knee flexion (4.4%). The peak strain force was recorded in the latter part of the power stroke (~160°), and did not differ significantly with changes in power level and/or cadence. Because the peak strain occurred at the same time the hamstrings were

contracting, the authors concluded that strain of the ACL may be a result of the gastrocnemius muscle producing an anterior-directed force on the tibia.

2.4.5 Pedaling in hemiparetic populations

Muscular weakness and increased muscle spasticity contribute to movement dysfunction in post-stroke hemiparesis. Pedaling a seated bicycle ergometer has been examined in such populations. Brown and Kautz (1998) measured the pedal reaction forces and EMG of 7 muscles in 15 hemiplegic subjects and 12 controls during randomly ordered workload and cadence combinations. Although the net mechanical work was significantly asymmetrical between hemiplegic subjects and controls, the net mechanical work of the paretic lower extremity increased as the workload increased without inappropriate muscle activity. The authors concluded that exertional pedaling exercise was beneficial in the studied population for achieving gains in muscular force output. In the present study, it was hypothesized that the ACL-reconstructed subjects would pedal more asymmetrically when compared to the controls.

2.5 Summary

Weakness of the QF muscle following ACL injury and ACL reconstruction presents a complicated rehabilitation problem. Following ACL injury, some individuals are able to return to prior activity levels (including sport) with an ACL-deficient knee but others may require a reconstruction of the ACL for instability. In the literature reviewed, there was evidence that operated thigh QF muscle weakness and atrophy persists after ACL reconstruction. This unilateral decrease in QF muscle strength and thigh cross-sectional area does not seem to be compounded by inhibition of the QF muscle, as no operated thigh increases in QF MI were reported in the studies reviewed.

If operated thigh QF muscle weakness is not mediated by MI, perhaps the CKC strengthening exercises used post-ACL reconstruction are not adequate for muscle hypertrophy. Also, due to evidence of kinematic and kinetic changes in the lower extremities post-ACL reconstruction, there may be a relative disuse atrophy of the QF muscle if the muscle is not being resisted in day-to-day activities. It has yet to be determined if loss of the mechanoreceptor input from the torn ACL mediates these gait changes, either through alterations in muscle activation, timing of muscle contraction and/or muscle stiffness.

To the best of this author's knowledge, the kinetics and kinematics of bicycling have not been studied in an ACL-reconstructed population. The use of a lower extremity cycle test should provide information regarding the joint angles, pedal forces, and resultant joint moments for an activity with low stress on the ACL graft and greater knee flexion angles than walking. In the literature reviewed, no studies were found which included measures of QF muscle strength, QF MI, knee laxity and the kinetics of motion following ACL reconstruction. The results of the bicycle test, combined with an examination of the strength and activation levels of the QF muscle, should contribute to our knowledge of lower extremity use post-ACL reconstruction.

2.6 Purpose and Hypotheses

Three *a priori* hypotheses were tested in this study:

1) Inhibition of the QF muscle may be a cause of weakness of the QF muscle post-ACL reconstruction. The first *a priori* hypothesis tested was that the ACL-reconstructed subjects would have significantly greater amounts of inhibition of the QF muscle when compared to the controls

2) Knee laxity has been cited as a possible cause of MI of the QF muscle. The second *a priori* hypothesis tested was that knee laxity would be significantly correlated to inhibition of the QF muscle.

3) Subconscious changes to day-to-day activities, altering the kinematics and kinetics of the lower extremities, may occur post-ACL reconstruction. These changes may in turn lead to a relative disuse atrophy of the QF muscle. The third *a priori* hypothesis tested was that the difference (between-leg) in peak $F_{(pedal)}z$ force would be significantly greater for the ACL-reconstructed subjects when compared to the controls.

3. METHODS AND MATERIALS

The study protocol and informed consent protocol were approved by the University of Calgary Conjoint Medical Ethics Committee. All subjects were volunteers and gave informed, written consent to participate in this study.

3.1 Subjects

Retrospectively, it was estimated that approximately 180 individuals had ACL reconstructive surgery performed by the surgeons from the University of Calgary Sport Medicine Centre during 1998. From this population, potential ACL-reconstructed subjects were identified through the database (as willing to participate in research), and were contacted by phone from January to March of 1999. Table 3.1 details the University of Calgary Sport Medicine Centre ACL rehabilitation protocol. The control sample was a non-random group of students and employees from the University of Calgary, and were chosen relative to the ACL-reconstructed sample for gender, approximate age (+/- 5 years), and activity level (hours per week of athletic participation).

Exclusion criteria for the ACL-reconstructed population included: pregnancy; reinjury to the reconstructed knee; history of knee injury to the non-operated limb; and/or inability to perform the test procedure. Exclusion criteria for the control population included: pregnancy; history of knee injury or chronic knee pain; and/or a between-knee difference (measured with the KT 2000) in anterior tibial laxity greater than 3 mm.

Table 3.1: Details of the University of Calgary Sport Medicine Centre ACL rehabilitation exercise protocol.

Stage One (0-6 weeks post surgery)
Protection: extension splint to be worn for 2-5 weeks post-op.
Weight-bearing: weight-bearing as tolerated (using crutches) for 3-5 weeks post-op.
Range of motion: progress to full knee ROM by 6 weeks post-op.
Strength: ***QF*** – double leg squats, progress to shallow single leg squats, leg press.
Hamstrings – leg curls with elastic tubing resistance.
Hip Musculature – abduction, adduction, flexion and extension.
Lower leg – calf raises, resisted ankle dorsiflexion.
Endurance: stationary cycling as tolerated.

Stage Two (6-12 weeks post-surgery)
Strength: ***QF*** – single leg squats (no greater than 45° of knee flexion), electrical muscle stimulation, single leg press (no greater than 90° of knee flexion).
Hamstrings – leg curls on weight machine, hamstring eccentrics.
Hip Musculature – resisted abduction, adduction, flexion and extension.
Endurance: stationary cycling (progress to intervals and standing), stair machine, treadmill (walking first, progress to jogging).
Balance: single leg balance, wobble board.

Stage Three (12 to 24 weeks post surgery)
Strength: partial squats with hand weights, leg press, hamstring curls emphasized.
Endurance: stationary cycling, stair climber, treadmill (running)
Muscular power/balance/endurance: forward/backward/diagonal lunges against elastic tubing, side-to-side shuttles, single leg balance.

At the University of Calgary Sport Medicine Centre, ACL-reconstructed patients are assessed at six months post-surgery. The physical therapy assessment portion includes isokinetic Cybex (Cybex 340® used) testing and knee laxity measurement using a KT 2000® joint arthrometer. As certain ACL-reconstructed subjects were recruited after their six-month assessment, some of the isokinetic and KT 2000 tests were conducted by experienced Physical Therapists at the clinic; the rest of the tests were performed by the author. One ACL-reconstructed subject had completed isokinetic testing at another facility; this information was not included in the data analysis, and the subject did not

consent to have further testing done. For continuity, the above tests were also performed on the control subjects. The KT 2000 information was used to rule out undiagnosed instability.

3.2 Strength Testing

The isokinetic Cybex testing protocol used in this study was that used by the University of Calgary Sport Medicine Centre, and consisted of 4 practice repetitions and 5 maximal repetitions at an angular velocity of 90°/s, and 4 practice repetitions and 25 maximal repetitions at an angular velocity of 240°/s. The order of Cybex tests was held constant for all study participants. For the ACL-reconstructed subjects, the non-operated thigh was tested first followed by the operated thigh, and no systematic leg order was used for the control subject tests. A JASA attachment was used for all control and ACL-reconstructed subject knee extensor strength tests. The peak extensor torque was recorded for each set of isokinetic measurements. Prior to testing, each subject's leg was weighed as part of the dynamometer protocol. The leg weight at the angle corresponding with the recorded peak torque was used for the gravity correction factor. The isometric knee extension strength test protocol will be described in the twitch interpolation test section.

3.3 Measurement of Anterior Tibial Translation

Anterior tibial translation was measured using the KT 2000 joint arthrometer. The device was strapped to the anterior leg with the pads in contact with the tibia and patella, and anterior/posterior displacement of the tibia was measured relative to the patella

(Jackson, 1993). The load-displacement curves were recorded with the KT 2000 joint arthrometer, and the results were sent to a computer software program (Maitland et al., 1995). Both knees of each subject were tested separately, with the arthrometer strapped to the anterior leg and the knee flexed to approximately 25°. For the ACL-reconstructed subjects, the non-operated knee was tested first, followed by the operated knee. No systematic knee order was used for the controls. For the ACL-reconstructed subjects, knee laxity (mm) was displayed as the difference between the operated and non-operated knees; for the controls, the variable was presented as the difference between the left and right knees.

3.4 Twitch Interpolation Technique

In preparation for the testing procedure and before the electrodes were applied, each subject completed a warm-up consisting of 10 minutes of stationary biking. All subjects (ACL-reconstructed and controls) performed the isometric knee extensions on a Cybex (NORM) dynamometer. To standardize the subject position in the Cybex chair, the following procedures were followed. The lateral epicondyle of the femur was aligned with the axis of rotation of the dynamometer arm, at a distance of approximately 5 to 10cm. Straps were used to secure the leg to the JASA and the thigh to the chair. A seatbelt was worn over the pelvis and chest. The subjects were asked to hold onto side handles near the chair seat for all contractions, and were encouraged to watch the torque curve output on the Cybex during the isometric contractions (Cybex Norm User's Guide). The knee angle was held constant at 65° of flexion for all tests; this value was normalized by calibrating the Cybex to a known angle (90° of flexion from the horizontal) before

each testing period, and by reassessing the knee angle during testing (using a goniometer). Watkins et al. (1991) investigated the intratester reliability of knee flexion and extension goniometer measurements, and found an intraclass correlation coefficient of 0.99 for flexion and 0.98 for extension. Each subject performed a warm-up consisting of a minimum of two maximal isometric contractions on the Cybex prior to testing.

To deliver the muscle stimulation, a Grass S88 (Quincy, MA) muscle stimulator was used in series with an isolation unit. The skin was first shaved and cleaned with alcohol. Next, two electrodes (4.5cm x 10cm) were covered thinly with conductive gel and taped on the anterior thigh. One electrode was taped on the skin over the femoral nerve at its most superficial, proximal location (at the base of the femoral triangle, just distal to the inguinal ligament), and the other electrode was taped over the patellar tendon, just proximal to the patella (Suter et al., 1998b). Doublet pulses of 0.8ms duration, separated by an 8ms interval, were applied to the muscle up to a maximal intensity of 240V. All values were also corrected for a small offset voltage present in the amplified tracing. Torque signals were sampled at 200 Hz per channel and displayed online for immediate feedback for the tester and subject (Suter et al., 1998b).

The testing protocol was as follows. Resting muscle twitches at a lower intensity than the test stimulus were completed first to familiarize the subjects with the testing technique. Next, the subject was asked to perform 3 maximal, isometric contractions. When the torque plateau was reached (after approximately 2-3s), a doublet of stimulation pulses was delivered to the QF muscle and the interpolated twitch torque (ITT) was

measured. Two-minute rest intervals were used between all contractions. Following the 3 ITT measurements, 3 resting muscle twitches were completed at the same voltage. Again, 2 minute intervals were used between the resting twitches, and the resting twitch torque (RTT) was recorded for each of the 3 twitches. To calculate the percentage of MI, the average RTT was calculated for the 3 separate twitches, and the ITT with the highest torque at MVC were used. The non-operated thigh was tested first followed by the operated thigh for the ACL-reconstructed subjects. No systematic thigh order was used for the controls. Immediately following the interpolated twitch testing, all participants were asked to complete a 100mm visual analogue pain scale for each knee; and they were asked to comment only on their level of knee pain during the isometric QF contractions, not the discomfort of the muscle twitches (Carlsson, 1983).

3.5 The Cycling Test

3.5.1 Pedal dynamometers

The pedal dynamometers used for the cycling test were designed by Moyer and Hull (1996), and were generously loaned to the University of Calgary Sport Medicine Centre from the Department of Mechanical Engineering, University of California at Davis, CA. Developed from a pedal used in track cycling, the pedals measured the driving forces, superior/inferior ($F_{(pedal)}z$) and anterior/posterior ($F_{(pedal)}x$), of cycling.

3.5.2 Pedal calibration

To calibrate the pedals, the cranks were mounted on a rigid, metal stand tall enough to hang weights underneath. Two small grooves were drilled on the surface of each pedal

(in the superior/inferior and anterior/posterior directions) to ensure the weighing apparatus was placed in exactly the same position with each weight change. Both the sensitivity and cross-sensitivity were calibrated for force in the $F_{(pedal)}z$ and $F_{(pedal)}x$ directions respectively. For each direction, linear regressions were calculated from the measured voltage versus the mass applied in newtons, and the calibration information was used to convert the force from volts to newtons. Figure 3.1 depicts the calibration procedure for force in the $F_{(pedal)}z$ direction. The cranks (with the pedals attached) were mounted directly to the calibration stand.

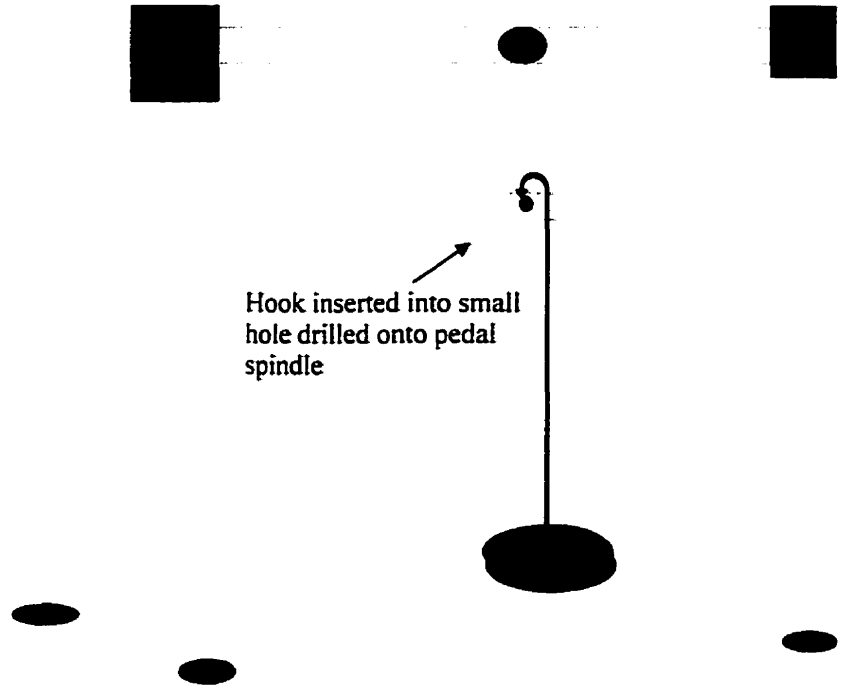


Figure 3.1: Calibration stand with the pedal mounted. The illustration depicts the calibration procedure for force in the $F_{(pedal)}z$ direction. The pedal was rotated 90° to calibrate force in the $F_{(pedal)}x$ direction.

3.5.3 Bicycle instrumentation

For the bike test, a standard road bicycle (54cm frame size) was mounted on a CompuTrainer® (RacerMate Inc., Seattle, Wash.). This trainer was designed to stabilize

the frame and add load to the rear wheel. The cranks were removed from the bicycle and were replaced with the pedal dynamometers complete with toe clips and straps to hold the foot on the pedal.

3.5.4 Subject preparation

Prior to testing, subjects were asked to change into shorts and running shoes. All subjects performed a 10 minute warm-up at a low resistance on a separate stationary bike before the cycling test. One-inch spherical reflective markers were placed on both lower extremities over the following bony landmarks: greater trochanter, lateral femoral epicondyle, lateral malleolus, tibial tuberosity, anterior tibia (just proximal to ankle joint), and over the shoe on the head of the fifth metatarsal. Stiff fabric backing was glued to each marker to allow the markers to be secured on the subject with double-sided tape. Additional tape was used when necessary to secure the markers. Each study participant was assisted onto the bike; the toe straps were adjusted and tightened by the investigator to ensure good shoe/pedal contact. To standardize the bicycle seat height between subjects, the seat was adjusted to a right knee angle (measured with a goniometer) of approximately 10°. This angle was measured with the pedal at BDC with the right ankle in approximately 90° of dorsiflexion. During the knee angle measurement, subjects were instructed to sit upright on the bicycle seat with arms at their sides.

3.5.5 System organization and calibration

Video motion analysis (Motion Analysis Corporation, Santa Rosa, CA) was used to collect kinematic information from both lower extremities. Four Falcon Hi-Res® (Motion Analysis Corporation, Santa Rosa, CA) cameras were used in the following manner. The 4 cameras were placed in an umbrella fashion around the front and sides of the bike, approximately 60° apart (see Figure 3.2). The camera positions and heights were adjusted to capture the volume of the bike and rider. Following the camera

adjustment, the bike was removed and replaced with an 8-point calibration frame. A goodness-of-fit value of 0.025 pixels (or less) was chosen for each camera calibration, and no more than two control points were eliminated per camera to obtain this value. EVa HiRES was used for the collection of 3-dimensional lower extremity kinematics and the analog (pedal force) data. EVa HiRES was also used to track the video motion data relative to the results of the cube calibration.

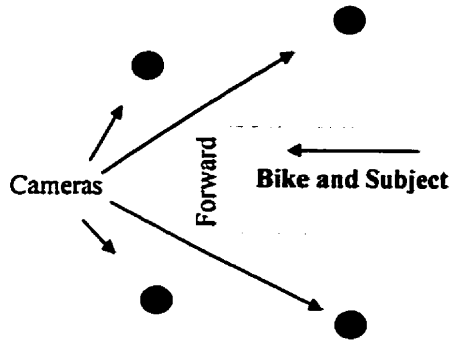


Figure 3.2: Diagram of the camera arrangement for collection of the kinematic data

The video information was recorded at a frequency of 60Hz for all subjects. The analog data were recorded at a frequency of 240Hz for the controls, and 2400Hz for the ACL-reconstructed subjects (frequency increased for the collection of EMG). Since no potentiometers were used to measure the crank and pedal angle, a rigid, wooden surface was used to mount three additional markers onto the side of the pedal. One marker was placed in the middle (aligned with the pedal spindle), and was used to calculate the crank angle. The two other markers were mounted on either end of the surface and were used to calculate the pedal angle (see Figure 3.3).

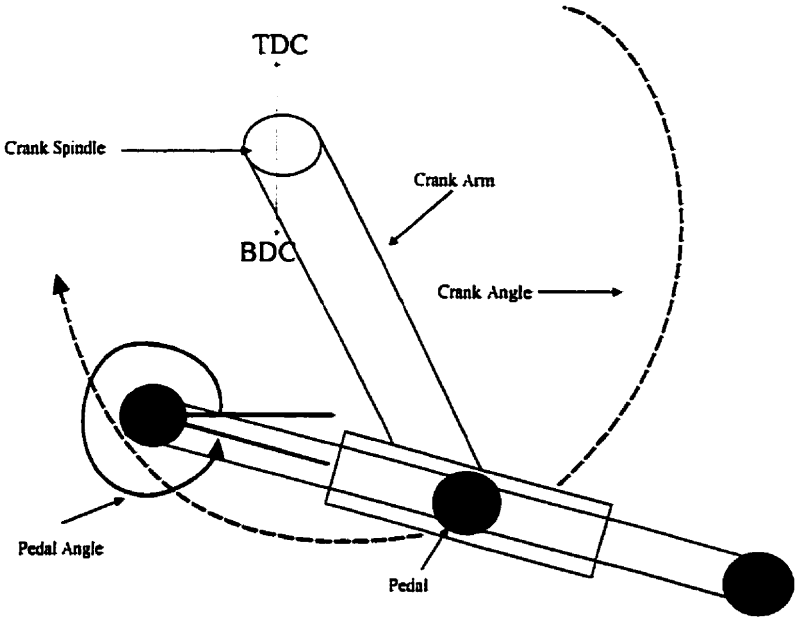


Figure 3.3: Pedal with additional reflective markers used to calculate the crank and pedal angles.

3.5.6 Cycling test procedure

To standardize the applied load, the rolling resistance of the rear tire was calibrated prior to each testing session, and the tires were inflated to 590 kPa. Each subject was assisted onto the bike, and the seat was adjusted according to the parameters outlined in a previous section. After approximately 5 minutes of pedaling at 60 RPM and a load of 100W, the resistance was increased to 150W, and the cadence held at 60 RPM. Two trials of 25 seconds each were collected from each subject to provide a provisional set of data. Each subject had approximately 1-2 minutes of rest between the trials where the load was decreased to 100W. During the brief rest period, the previously recorded trial results were inspected.

3.6 Data Analysis

3.6.1 Data analysis for the strength, muscle inhibition, and knee laxity tests

The QF muscle strength, QF muscle inhibition, and knee laxity data were summarized in the following manner. Using the formula in Table 3.2, the percent deficit (PD) in QF muscle strength was first calculated for all three strength tests (isometric, and isokinetic at 90°/s and 240°/s). Next, the means and 95% confidence intervals (CI) were calculated for the percent deficit in QF muscle strength and the muscle inhibition data. The knee laxity data were summarized by calculating the between-knee difference in laxity (non-dominant minus dominant knee for the controls, and operated minus non-operated knee for the ACL-reconstructed subjects).

With regard to statistical analysis, the first *a priori* hypothesis regarding QF muscle inhibition was addressed. To include both the group and leg effects, a two-way ANOVA was used to analyze the muscle inhibition data. Because no significant leg effect was present, each thigh measurement of MI was combined within a subject, and the average MI was calculated for each subject. A two-sample t-test was used to compare the average MI between the control and ACL-reconstructed groups. The second *a priori* hypothesis (whether knee laxity was significantly correlated to inhibition of the QF muscle) was addressed next. A Pearson Correlation Coefficient was determined between the values measured for knee laxity (anterior laxity in mm) and QF muscle inhibition (%). A significance level of $p<0.05$ was used for the *a priori* hypothesis tests.

An additional statistical comparison was performed on the strength data, and the results from this test were considered secondary to the *a priori* tests. A two-sample t-test was used to test the following hypothesis: the ACL-reconstructed subjects would have a greater percent deficit in isometric strength of the QF muscle than the controls (dominant – nondominant). A significance level of $p<0.05$ was also used for this secondary hypothesis test. No correction for multiple comparisons was used given the distinction between the *a priori* and secondary hypothesis tests.

Table 3.2: The calculation of the percent deficit of QF muscle strength within a subject.

ACL subjects: Percent Deficit = $\frac{\text{Torque (non-operated)} - \text{Torque (operated)}}{\text{Torque (non-operated)}} \times 100\%$

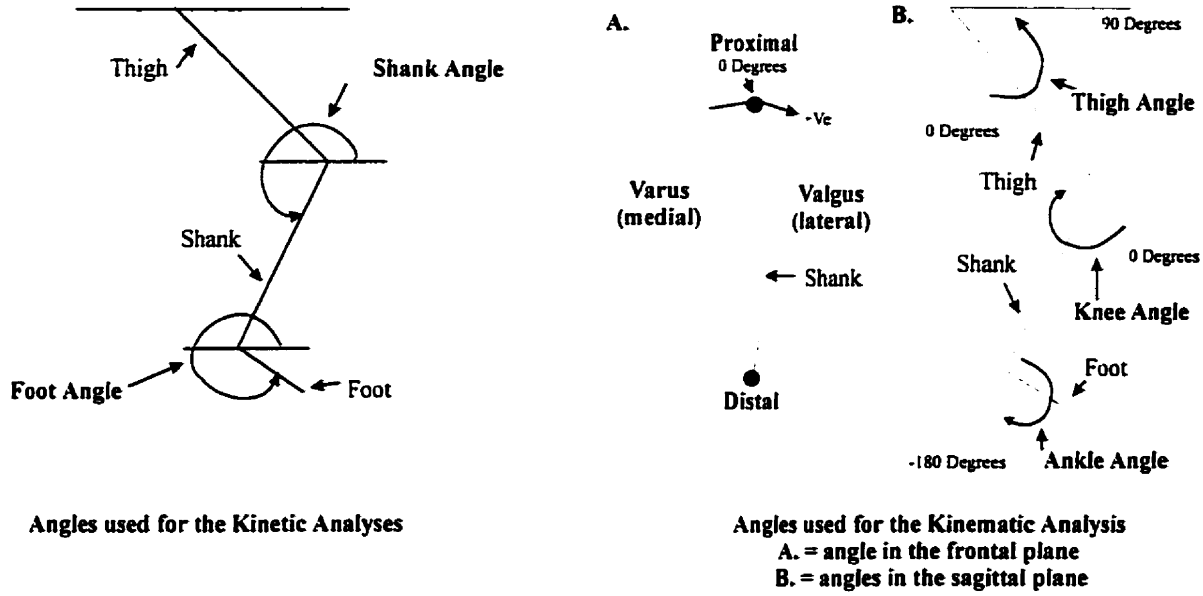
Controls: Percent Deficit = $\frac{\text{Torque (dominant)} - \text{Torque (non-dominant)}}{\text{Torque (dominant)}} \times 100\%$

Controls: Percent Deficit = $\frac{\text{Torque (stronger)} - \text{Torque (weaker)}}{\text{Torque (stronger)}} \times 100\%$

3.6.2 Data Analysis for cycling test

Although the kinematic data were filmed in 3D, the y-lab coordinate positions were dropped after the video information was tracked (except for those of the anterior tibial tubercle and anterior tibia markers), leaving the x and z lab coordinate positions for further kinematic analysis. The kinematic data were filtered as outlined in Kautz et al. (1994): a fourth order, zero phase shift, Butterworth filter of 9Hz was used to smooth the kinematic data, while a fourth order, zero phase shift Butterworth filter of 20Hz was used to smooth the kinetic data. The crank spindle was chosen as the coordinate system

origin, and was subtracted from all the filtered marker data. To calculate the segment angles, the lower extremity was modeled as 3-linked (by hinges) segments (see Figure 3.4) (Caldwell et al., 1999).



Angles used for the Kinetic Analyses

Angles used for the Kinematic Analysis
A. = angle in the frontal plane
B. = angles in the sagittal plane

Figure 3.4: Diagram of lower extremity modeled as 3-linked segments. The angles used for the kinetic analyses are on the left hand side, and the angles used for the kinematic analyses are on the right hand side.

During the analysis of the collected pedal force data, an error was noted. The range of voltage input set in the data acquisition box for the $F_{(pedal)}x$ was not adequate for the data collected. For some of the subjects tested, the right $F_{(pedal)}x$ force was clipped during the 0° to 90° phase of the pedal revolution. No left $F_{(pedal)}x$ forces were complete. For the right pedal results, the subject files with complete $F_{(pedal)}x$ forces were used for further calculations such as pedal-to-lab coordinate conversion, crank torque, knee forces, and the knee moment. The results for this data were compared descriptively between the ACL-reconstructed and control groups with the respective number of subject files

included for clarity. Individual results (foot angle, shank angle, pedal forces, knee forces, crank torque, and knee moments) for the controls and ACL-reconstructed subjects are provided in the Appendix, Part B.

All cycling data displayed were averaged over approximately 25 pedal revolutions per leg per subject. A program written in Matlab© (The Math Works, New Jersey, USA) was used to process the data (see Appendix, part A, pages 112-117). To determine the kinetics of the model, free body diagrams of each segment of interest (the foot and shank) were constructed, and the equations of motion were defined using Newton's third law. Pedal forces were converted from bits to newtons, and from a pedal to a lab coordinate system with the following equations:

Where: pedal_x = force of the foot on the pedal in the x direction (bits)

pedal_z = force of the foot on the pedal in the z direction (bits)

$$Fpx\ (volts) = Eva\ gain\ (volts) / 2047\ (bits) * pedal\_x \quad (3.1)$$

$$Fpz\ (volts) = Eva\ gain\ (volts) / 2047\ (bits) * pedal\_z \quad (3.2)$$

$$F_{Foot\ on\ Pedal X_{(Pedal)}},\ F_{Foot\ on\ Pedal Z_{(Pedal)}} = [Calibration\ Matrix] * [Fpx;\ Fpz] \quad (3.3)$$

$$F_{Foot\ on\ Pedal X_{(Lab)}} = [\cos(foot\ angle) * F_{Foot\ on\ Pedal X_{(Pedal)}}] - [\sin(foot\ angle) * F_{Foot\ on\ Pedal Z_{(Pedal)}}] \quad (3.5)$$

$$F_{Foot\ on\ Pedal Z_{(Lab)}} = [\sin(foot\ angle) * F_{Foot\ on\ Pedal\ X_{(Pedal)}}] + [\cos(foot\ angle) * F_{Foot\ on\ Pedal Z_{(Pedal)}}] \quad (3.6)$$

The crank torque was calculated with the following equation:

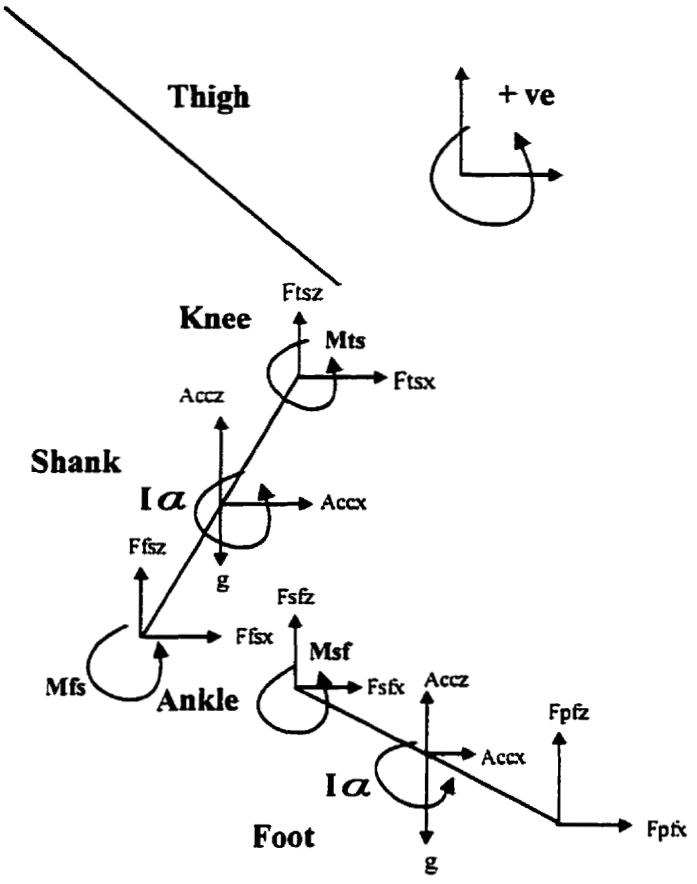
$$Torque_{Crank} = [F_{Foot\ on\ Pedal X_{(Lab)}} * \cos(crankangle) * cranklength] - [F_{Foot\ on\ Pedal Z_{(Lab)}} * \sin(crank\ angle) * cranklength] \quad (3.7)$$

Knee forces were converted from a lab to a shank coordinate system with the following equations:

$$F_{\text{Thigh on Shank}X_{(\text{Shank})}} = [\cos(\text{shank angle}) * F_{\text{Thigh on Shank}X_{(\text{Lab})}}] + [\sin(\text{shank angle}) * F_{\text{Thigh on Shank}Z_{(\text{Lab})}}] \quad 3.8$$

$$F_{\text{Thigh on Shank}Z_{(\text{Shank})}} = -[\sin(\text{shank angle}) * F_{\text{Thigh on Shank}X_{(\text{Lab})}}] + [\cos(\text{shank angle}) * F_{\text{Thigh on Shank}Z_{(\text{Lab})}}] \quad 3.9$$

From the equations of motion, the force and moment equations for the ankle and knee were derived. The segment masses, moments of inertia, and centre of gravity locations were estimated from Enoka (1994). Figure 3.5 consists of the schematic diagram used in this study to define the forces and moments of the lower extremity during cycling.



- Schematic representation of the forces and moments of the lower extremities during cycling.

Where:
Ffpx, Ffpz = $F_{\text{Foot on Pedal}X_{(\text{Lab})}}$, $F_{\text{Foot on Pedal}Z_{(\text{Lab})}}$ (see Calculations 3.7 and 3.8)
Fpfx, Fpfz = Force of the pedal on the foot in either the x or z direction
Fpfx = -(Ffpx)
Fpfz = -(Ffpz)
Accx, Accz = Acceleration of the centre of mass of the segment in either the x or z direction.
g = Acceleration due to gravity (9.81m/s^2)
Iα = Moment of inertia * angular acceleration of segment.
Fsfx, Fsfz = Force of the shank on the foot in either the x or z direction.
Ffsx, Ffsz = Force of the foot on the shank in either the x or z direction.
Msf = Moment of the shank on the foot.
Mfs = Moment of the foot on the shank.
Ftsx, Ftsz = Force of the thigh on the shank in either the x or z direction.
Mts = Moment of the thigh on the shank.
Ffsx, Ffsz = **-(Fsfx), -(Fsfz)** respectively
Mfs = -(Msf)

Figure 3.5: Moments and forces of the lower extremity during cycling.

1) Forces at the proximal foot
Where:
Fpfx = -1 * (Ffpx)
Fpfz = -1 * (Ffpz)

$\Sigma F = mass_{(Foot)} * Acc_{(at\ CG\ Foot)}$

$Fsfx = mass_{(Foot)} * Acc_{x(at\ CG\ Foot)} - Fpfx$ 3.10

$Fsfz = mass_{(Foot)} * Acc_{z(at\ CG\ Foot)} - Fpfz + mass_{(Foot)} * (g)$ 3.11

(2) Forces at the proximal shank
Where:
Ffsx = -1 * (Fsfx)
Ffsz = -1 * (Fsfz)

$\Sigma F = mass_{(Shank)} * Acc_{(at\ CG\ Shank)}$

$Ftsx = mass_{(Shank)} * Accx_{(at\ CG\ Shank)} - Ffsx$ 3.12

$Ftsz = mass_{(Shank)} * Accz_{(at\ CG\ Shank)} - Ffsz + mass_{(Shank)} * (g)$ 3.13

(3) Moment of the Shank on the Foot (β = foot angle)

$\Sigma Moment_{CG(Foot)} = I_{CG(Foot)} * \alpha_{(Foot)}$

$Moment_{(Shank\ on\ Foot)} = [I_{CG(Foot)} * \alpha_{(Foot)}] - [-(\sin\beta) * (foot\ length * 0.5) * Fpfx] - [\cos\beta * (foot\ length * 0.5) * Fpfz] - [\cos(\beta + 180°) * (foot\ length * 0.5) * Fsfz] - [-(\sin(\beta + 180°)) * (foot\ length * 0.5) * Fsfx]$ 3.14

(4) Moment of the Thigh on the Shank (Ø = shank angle)

Where: Ffsx = -1 * (Fsfx)
Ffsz = -1 * (Fsfz)
$Moment_{(Foot\ on\ Shank)} = -1 * (Moment_{(Shank\ on\ Foot)})$

$\Sigma Moment_{CG(Shank)} = I_{CG(Shank)} * \alpha_{(Shank)}$

$Moment_{(Thigh\ on\ Shank)} = [I_{CG(Shank)} * \alpha_{(Shank)}] - Moment_{(Foot\ on\ Shank)} - [-(\sin Ø) * (shank\ length * 0.567) * Ffsx] - [\cos Ø * (shank\ length * 0.567) * Ffsz] - [\cos(Ø + 180°) * (shank\ length * 0.433) * Ftsz] - [-(\sin(Ø + 180°)) * (shank\ length * 0.433) * Ftsx]$ 3.15

With regard to the *a priori* hypothesis test for the cycling data, the peak $F_{(pedal)}z$ force (averaged over approximately 25 pedal revolutions) was recorded for each subject within each group (control and ACL-reconstructed). To compare the pedaling symmetry between the two groups, the mean result from the non-dominant limb was subtracted from the mean result for the dominant limb. For example, for the controls, the left leg peak $F_{(pedal)}z$ force was subtracted from that of the right leg. For the ACL-reconstructed subjects, the operated leg peak $F_{(pedal)}z$ force was subtracted from that of the non-operated leg. The means and 95% confidence intervals for the between-leg difference were first calculated for both groups, then a two-sample t-test was used to compare the means between the two groups.

A secondary hypothesis was also tested using the cycling data. A two-sample t-test was used to test the following hypothesis: the peak $F_{(pedal)}z$ force for the ACL-reconstructed subjects would occur significantly later in a pedal revolution compared to the controls. A 0.05 significance level was used for this hypothesis test. With regard to the complete pedal force trials, four control files (right leg only), and 8 ACL-reconstructed subject files (3 operated and 5 non-operated legs) were complete and used for further kinetic analysis. The results of these data were presented graphically.

4. RESULTS

4.1 Subjects

Eleven persons with a reconstructed ACL (six men and five women; mean age, 28.4 years; age range, 23 to 46 years) volunteered for the study. The mean height of the ACL-reconstructed subjects was 174.3cm (range (156 to 188cm), and the mean weight was 75.5kg (range 52 to 109.3kg). The mean time from surgery to testing was 224 days (range 184 to 278 days). All ACL-reconstructed subjects had arthroscopic BPTB autograft procedures. Table 4.2 outlines the mean time from injury to surgery, concurrent arthroscopic procedures, and post-operative complications for the ACL-reconstructed subjects. All ACL-reconstructed subjects completed at least six weeks of a modified accelerated ACL rehabilitation program (see Table 3.1, page 46); two subjects did not complete their rehabilitation at the University of Calgary Sport Medicine Centre.

One control subject was excluded from the data analysis for a between-knee difference in anterior tibial translation (as measured with the KT 2000) greater than 3mm. Thus, the control sample consisted four men and three women: mean age, 30.8 years; age range, 23 to 42 years. The mean height of the controls was 172.7cm (range 169 to 180cm), and the mean control weight was 70.5kg (range 54 to 84kg).

To determine the activity level of the subjects during the test period, all subjects completed a questionnaire regarding participation in athletic activities. This information is displayed in Table 4.1.

Table 4.1: Hours per week of athletic participation

	Under 2 hours	2 to 6 hours	Over 6 hours
Controls	n=0	n=4	n=3
ACL Subjects	n=3	n=5	n=3

Table 4.2: ACL-reconstructed subject data

Subject number (Operated-leg)	Months from injury to surgery	Menisectomy?	Side to Side strength difference*	Complications?
1 (left)	10	no	25%	arthrofibrosis (manipulation)
2 (left)	11	no	16%	no
3(left)**	5	partial lateral	22%	no
4(left)**	2	partial lateral	47%	no
5 (left)	10	no	-1.0%	no
6 (right)	3	no	52%	no
7 (right)	72	partial medial	32%	no
8 (left)	30	partial medial and lateral	31%	lateral menisectomy (98/11/20)
9 (right)	61	medial repair	1%	no
10 (left)	336	partial medial	17%	no
11 (right)	124	partial lateral and medial	29%	no

* **Strength Difference** is defined as **percent deficit** (see Table 3.2) for the isometric torque at 65° of knee flexion.

** **Subject did not complete post-operative rehabilitation at the University of Calgary Sport Medicine Centre.**

4.2 Muscle Inhibition

Figure 4.1 displayed the measured muscle inhibition (+ 1 standard error of the mean) for each group tested. There was no significant group-by-leg interaction ($F(1,16)=0.46$, $p=0.508$), no significant leg effect ($F(1,16)=0.59$, $p=0.454$), and a slight group effect ($F(1,16)=3.7$, $p=0.073$). Given the results of the 2-way ANOVA (no significant leg effect), the right and left leg results for each subject (in both the control and ACL-reconstructed groups) were averaged, and the average MI (AvrMI) was calculated. The controls demonstrated a mean AvrMI (95% CI) of 36.04% (22.71%, 49.89%), and the cases had a mean AvrMI of 21.08% (12.24%, 29.92%). A two-sample t-test was used to test the first *a priori* hypothesis, and the ACL-reconstructed subjects did not have significantly greater amounts of AvrMI when compared to the controls ($p=0.97$). The power of the above statistical test (to detect a significant difference) was calculated at 0.57.

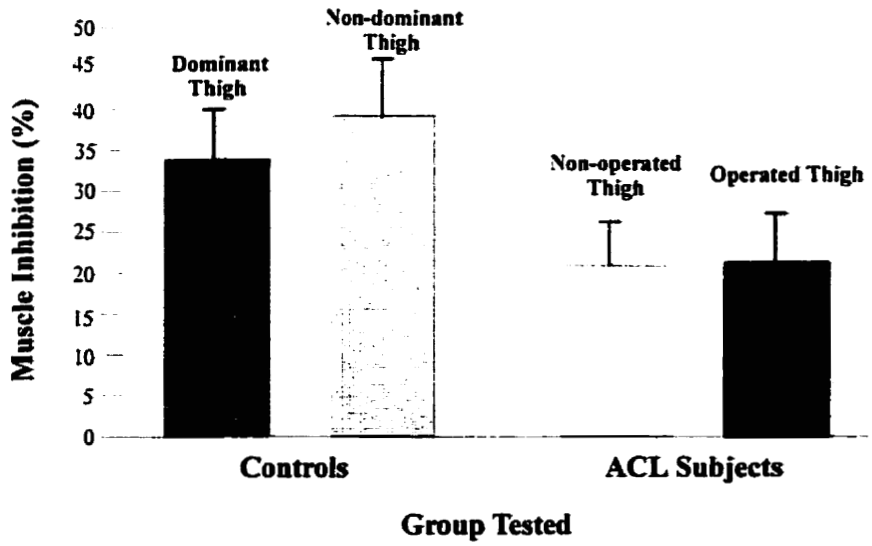


Figure 4.1: The mean percentage of MI of the QF (+ one standard error) for the control and ACL-reconstructed subject groups.

4.4.1 Muscle inhibition, knee laxity, and QF muscle strength

With regard to the second *a priori* hypothesis, there was no significant correlation between muscle inhibition and knee laxity (refer to Figure 4.2, correlation coefficient=0.2057; p=0.23). To examine the relation of QF muscle strength, MI and knee laxity, the isometric between-thigh QF muscle strength (Nm/kg) was plotted against between-thigh MI (Figure 4.3) and against between-knee anterior laxity (Figure 4.4). The power of the above statistical test was calculated at 0.067.

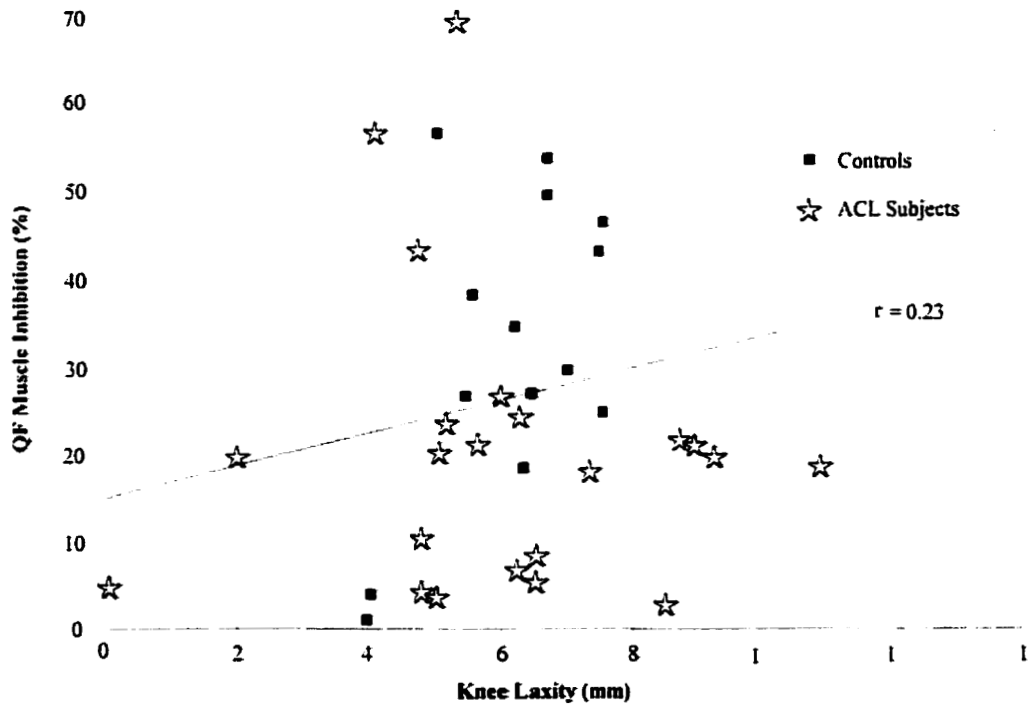


Figure 4.2: Scatterplot of the QF muscle inhibition (%) versus knee laxity (mm) for the ACL-reconstructed subjects and controls.

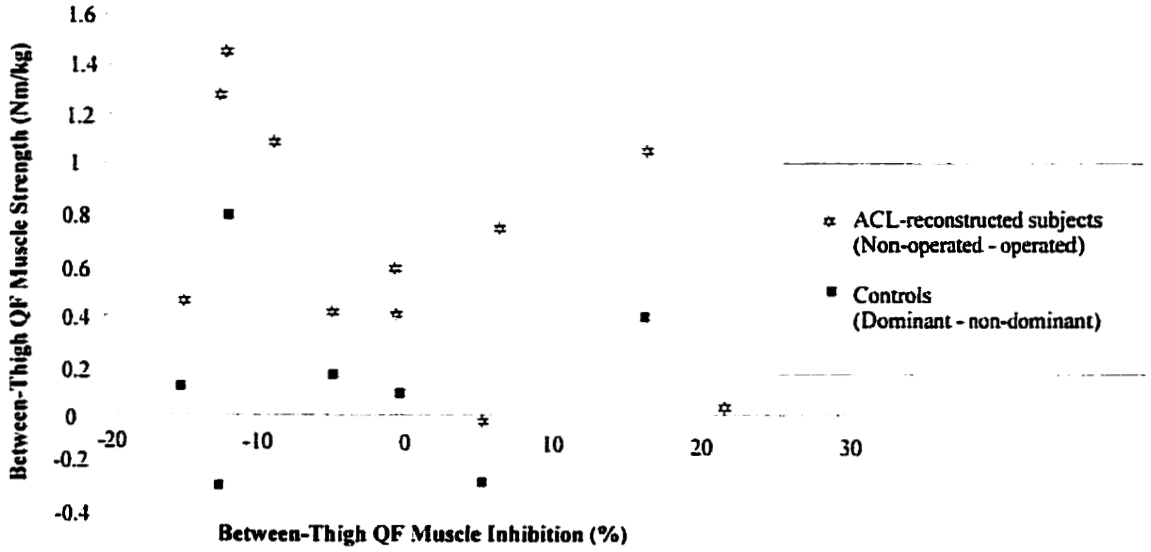


Figure 4.3: Scatterplot of between-thigh QF muscle strength versus between-thigh QF muscle inhibition for all subjects.

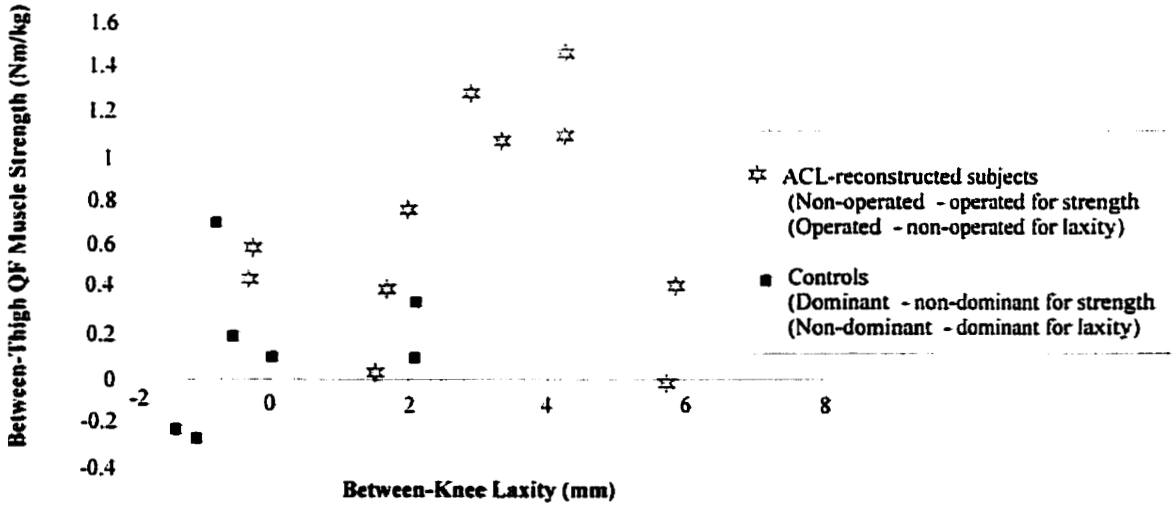


Figure 4.4: Scatterplot of between-thigh QF muscle strength versus between-knee laxity for all subjects.

4.4.2 Pain during muscle inhibition testing

One control subject reported knee pain during the interpolated twitch testing. Seven ACL-reconstructed subjects reported knee pain; of the ACL-reconstructed subjects who reported pain during testing, the pain was bilateral in 4 subjects. If the pain was bilateral, the pain scale measurements were averaged between the right and left legs of the subjects (ACL-reconstructed) who reported pain. The average control subject pain rating was 2.7/100 mm and the average ACL-reconstructed subject pain rating was 8.3/100 mm. One ACL-reconstructed subject reported operated leg anterior knee pain that lasted approximately 48 hours after twitch interpolation testing. There did not appear to be a relation between knee pain and MI of the QF muscle.

4.3 Anterior Tibial Translation

The mean between-knee difference (95% CI) in anterior tibial translation for the ACL-reconstructed subjects (operated knee – non-operated knee) was 2.81mm (1.39, 4.24). The control subjects had a mean difference (non-dominant knee – dominant knee) of 0.18 mm (-1.12, 1.48).

4.4 Strength Measurements

The strength measurements, recorded as percent deficit (PD) (for the calculation used, refer to Table 3.2, page 57), are summarized in Table 4.3. Figure 4.5 contains the mean torque measurement for both groups tested. The ACL-reconstructed subjects had a significantly greater PD of QF muscle strength compared to the controls for isometric knee extension at 65° of knee flexion ($p=0.04$).

Table 4.3: Comparison of the means and range for the PD of isokinetic and isometric knee extensor muscle strength

Percent Deficit Measurement:	90 °/s	240 °/s	Isometric (65°)
ACL-reconstructed:			
mean:	14.91%	16.35%	23.81%
range:	-9.24 to 36.51	2.51 to 28.06	-0.69 to 52.08
Controls:	(dominant – non-dominant)		
mean:	2.53%	3.12%	4.04%
range:	-17.95 to 14.09	-11.69 to 27.84	-18.51 to 34.32
Controls:	(stronger thigh – weaker thigh)		
mean:	9.02%	8.30%	12.43%
range:	0.66 to 15.99	0.95 to 27.84	3.45 to 34.32

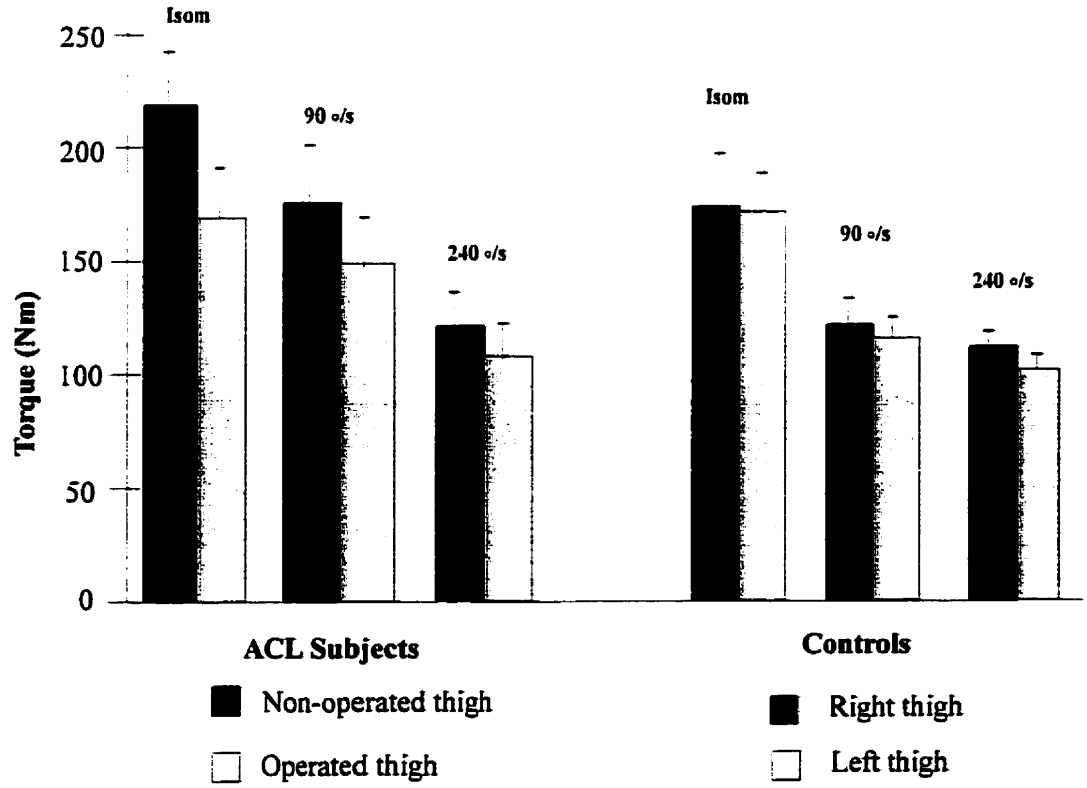


Figure 4.5: Mean peak extensor torque (Nm + one standard error) measured for the knee extensor strength tests.

4.5 The Cycling Test

4.5.1 Calibration results

Both the sensitivity and cross-sensitivity were measured for the force in the x and z directions. Linear regression was calculated for the voltage versus force (newtons), and the correlation coefficient value results are presented in Table 4.4. The slope from each linear regression was combined to form a calibration matrix for each pedal; and this information was used to convert the force from volts to newtons.

Table 4.4: Linear regression results (correlation coefficient value) for the pedal calibration

Right Pedal	**Sensitivity**	**Cross-sensitivity**
X-Direction	0.9992	0.9647
Z-Direction	0.9968	0.9630
Left Pedal		
X-Direction	0.9996	0.9498
Z-Direction	0.9997	0.9728

4.5.2 Lower extremity kinetics during cycling

For all study participants, $F_{(pedal)}z$ forces (pedal coordinate system) were collected. The results are presented graphically in Figure 4.6 (mean $F_{(pedal)}z$ forces over a pedal revolution). The dotted lines in Figure 4.6 refer to the difference in timing of the peak $F_{(pedal)}z$ force between the control and ACL-reconstructed groups. The peak $F_{(pedal)}z$ forces were recorded for each subject (control and ACL-reconstructed). The means (95% CI) for the difference in peak $F_{(pedal)}z$ force are presented below in Table 4.6. There was

no significant difference between the two values (p=0.22). For the above statistical test, the power to detect a significant difference was calculated as 0.32.

Table 4.5: Means and 95% confidence intervals for the between-leg difference in peak $F_{(pedal)}z$ force (measured in newtons)

Controls	23.1 N (-20.0, 66.2)
ACL-reconstructed subjects	0.1 N (-20.5, 20.6)

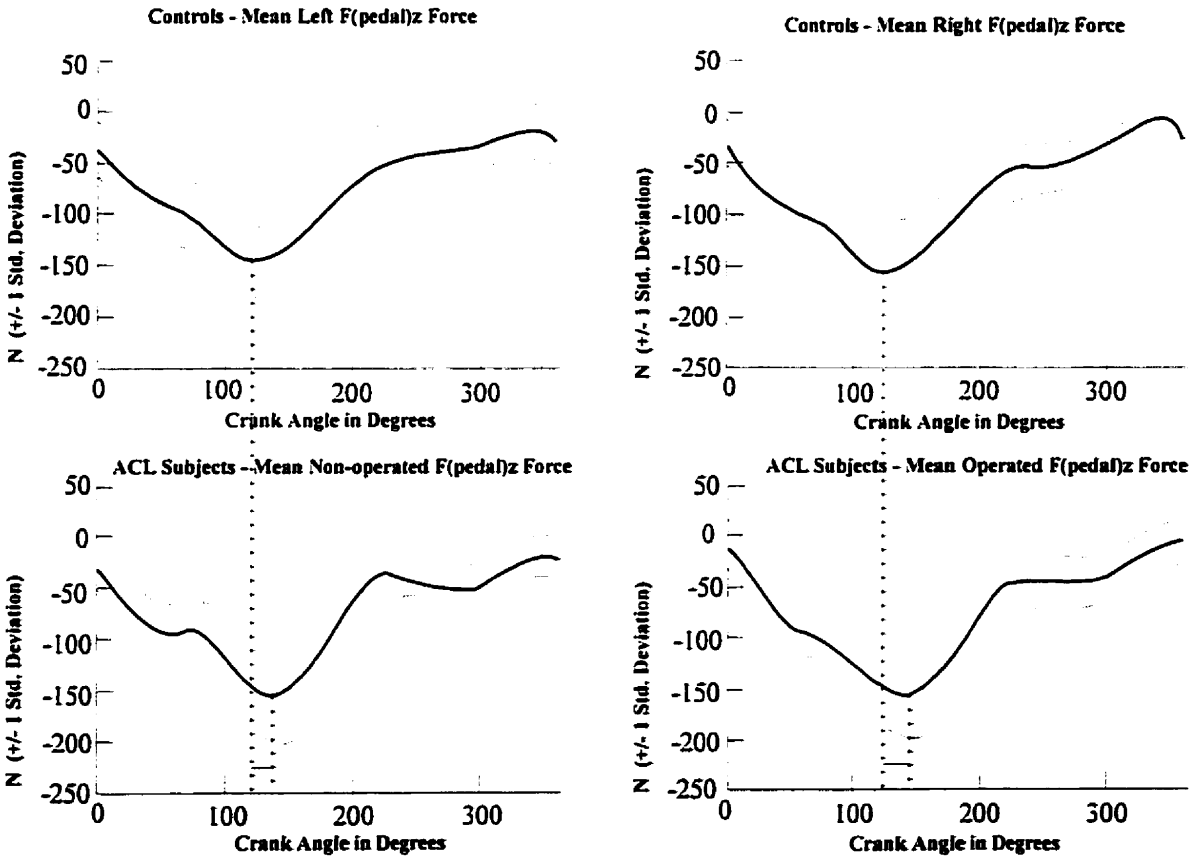


Figure 4.6: Mean normal pedal force (in newtons) for all subject groups (+/- 1 standard deviation). The dotted line depicts the differences in timing of the peak $F_{(pedal)}z$ force between the controls and ACL-reconstructed subjects.

To calculate the difference in timing of peak $F_{(pedal)}z$ force, the results (in degrees of the crank angle) from each leg were combined to calculate a between-leg average result. For the control subjects, the mean between-leg average (95% CI) was 120.9° (105.1°, 136.8°), and for the ACL-reconstructed subjects, the mean between-leg average was 138.8° (132.2°, 145.5°). A two-sample t-test for independent data was performed, and the peak $F_{(pedal)}z$ force occurred significantly later in a pedal revolution ($p=0.01$) for the ACL-reconstructed subjects.

For the complete pedal force trials, the forces of the foot on the pedal are displayed in Figure 4.7, and the sagittal tibiofemoral joint forces (in a shank coordinate system) are displayed in Figure 4.8. The crank torque (Figure 4.9) and knee moments (Figure 4.10) are also displayed for the controls and the ACL-reconstructed subjects. Refer to the appendix (Part B) for individual subject (control and ACL-reconstructed) data summaries.

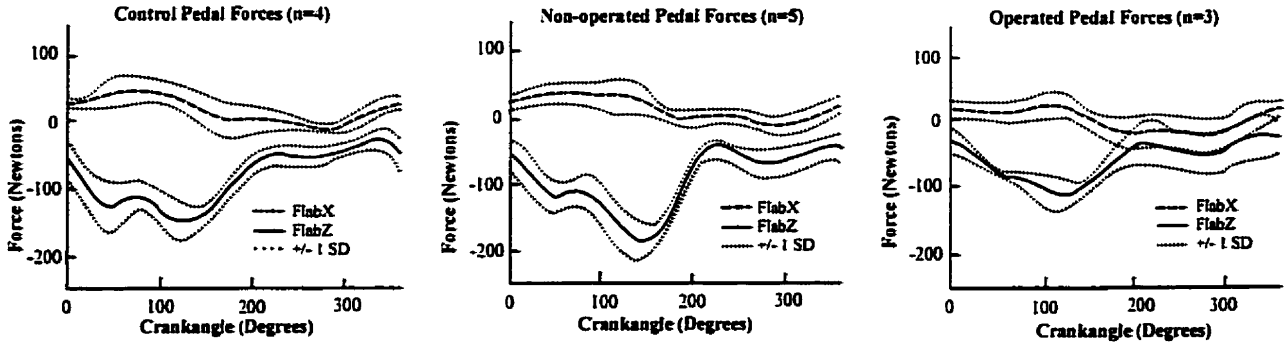


Figure 4.7: Force of the foot on the pedal (+/- 1 standard deviation) for the controls (n=4, right lower extremity only) and the ACL-reconstructed subjects (non-operated, n=5, and operated, n=3, lower extremities).

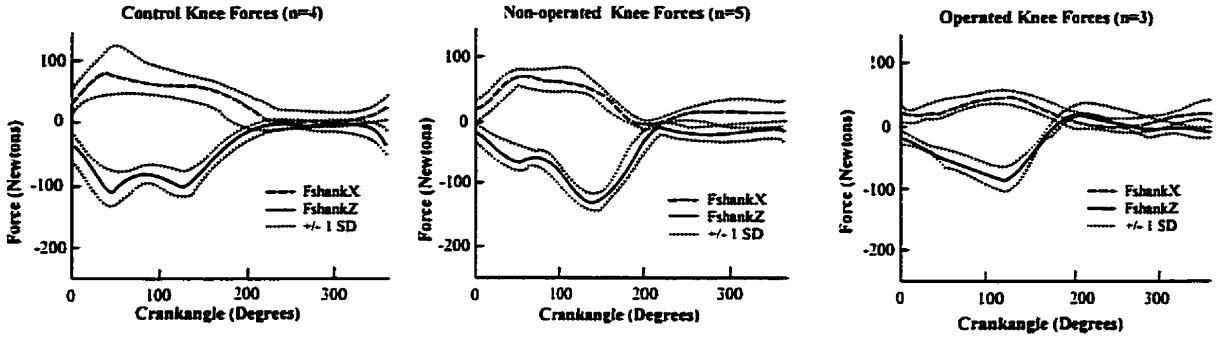


Figure 4.8: Knee forces (force of the thigh on the shank) (+/- 1 standard deviation) for the controls (n=4, right lower extremity only) and the ACL-reconstructed subjects (non-operated, n=5, and operated, n=3, lower extremities).

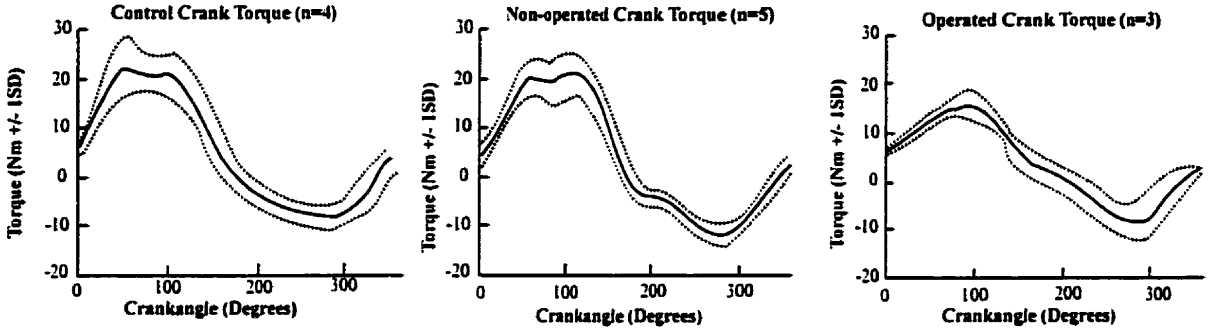


Figure 4.9: Crank torque (+/- 1 standard deviation) for the controls (n=4, right lower extremity only) and the ACL-reconstructed subjects (non-operated, n=5, and operated, n=3, lower extremities).

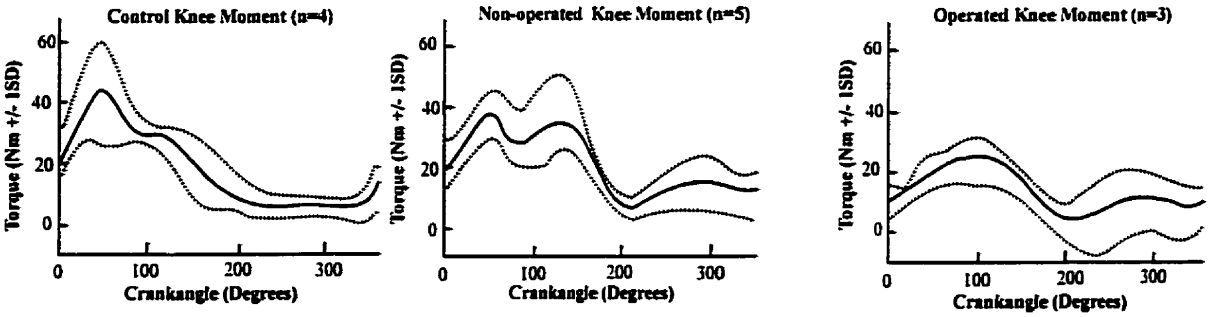


Figure 4.10: Knee moment (+/- 1 standard deviation) for the controls (n=4, right lower extremity only) and the ACL-reconstructed subjects (non-operated, n=5, and operated, n=3, lower extremities).

4.5.3 Lower extremity kinematics during cycling

The kinematic data collected from the cycling portion of the study were inspected. The knee angles for the respective control and ACL-reconstructed subject groups are presented in Figure 4.11. For the controls, the mean knee extension angle (95% CI) at BDC was 29.55° (21.79°, 37.32°) for the right lower extremities tested and 28.22° (20.45°, 35.99°) for the left lower extremities tested. For the ACL-reconstructed subjects tested, the mean knee extension angle (95% CI) at BDC was 34.41° (32.22, 36.61) for the non-operated lower extremities tested and 35.67° (31.85°, 39.50°) for the operated lower extremities tested.

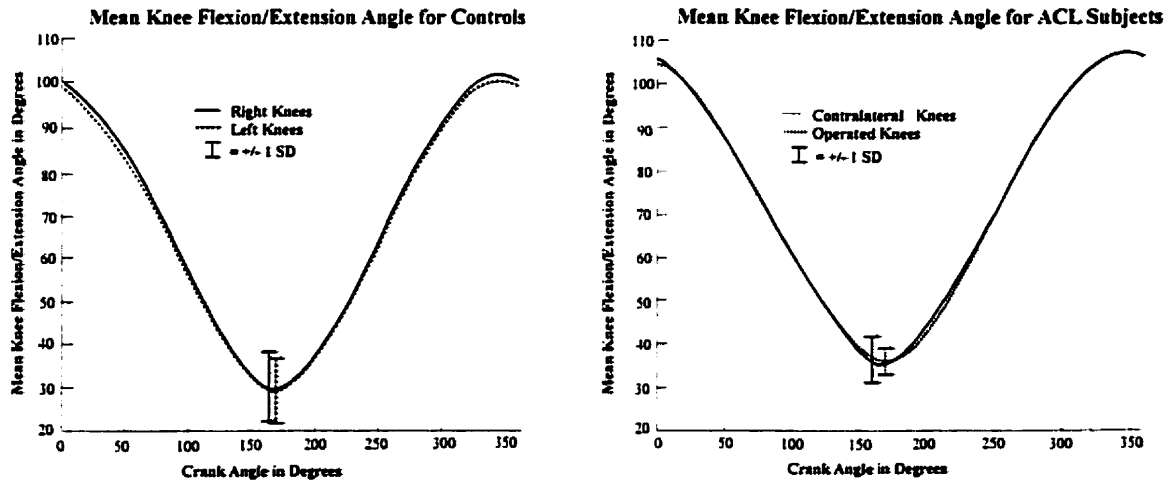


Figure 4.11: Mean knee flexion/extension angles for the control and ACL-reconstructed groups.

Given the difference in knee extension at BDC, the kinematics of the joints proximal and distal to the knee joint were examined next. The thigh angles for the control and ACL-reconstructed groups are presented in Figure 4.12.

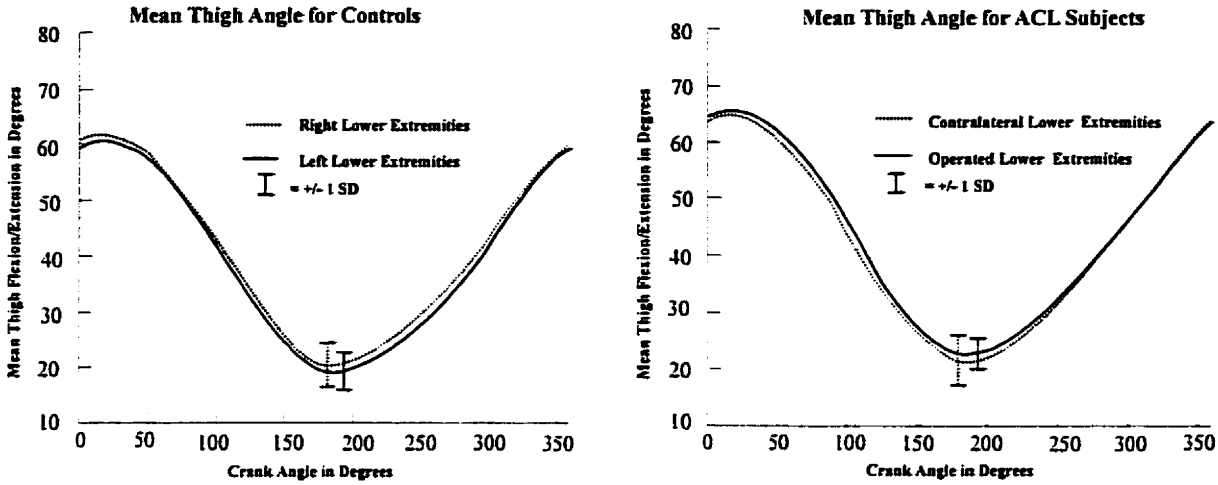


Figure 4.12: Mean thigh flexion/extension angles (relative to the horizontal) for the control and ACL-reconstructed groups

Rather than the hip flexion angle, the thigh flexion angle was used for comparison with the rest of the lower extremity kinematics (no pelvic marker data were collected to determine the hip flexion angle). The mean thigh flexion angle was approximately 5° greater for the ACL-reconstructed group, and the mean thigh extension angle was approximately 2° less for the ACL-reconstructed group (although there was overlap of the standard deviations between the two groups). Figure 4.13 displays the mean knee flexion angles versus the thigh flexion angles for the ACL-reconstructed subjects and controls. At TDC, there appeared to be an increase in both knee flexion and hip flexion for the ACL-reconstructed subjects.

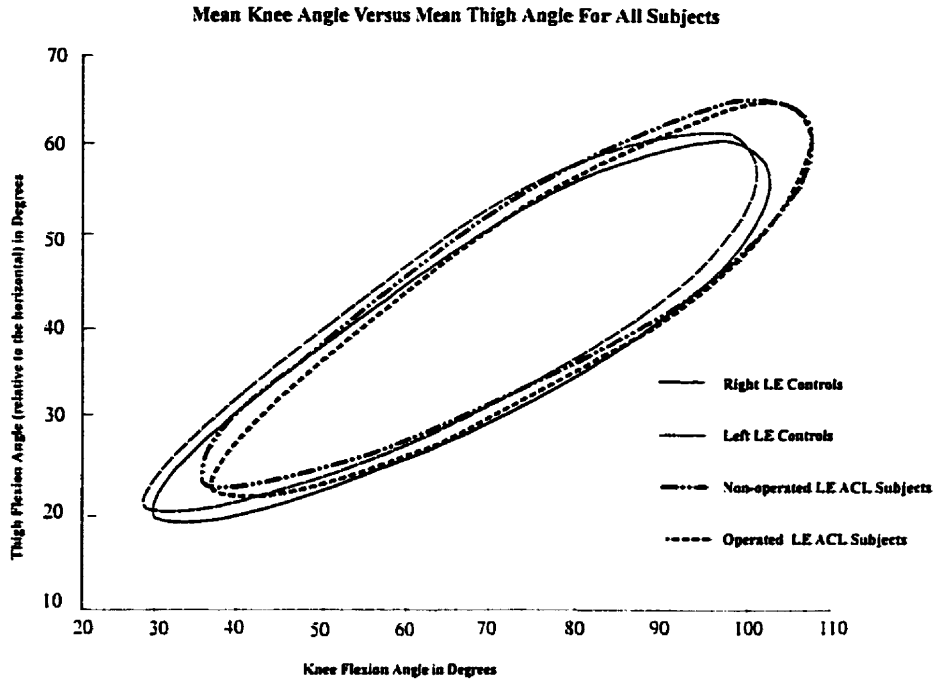


Figure 4.13: Angle-angle plot of mean knee flexion angle versus mean thigh flexion angle (relative to the horizontal) for the ACL-reconstructed subjects and the controls.

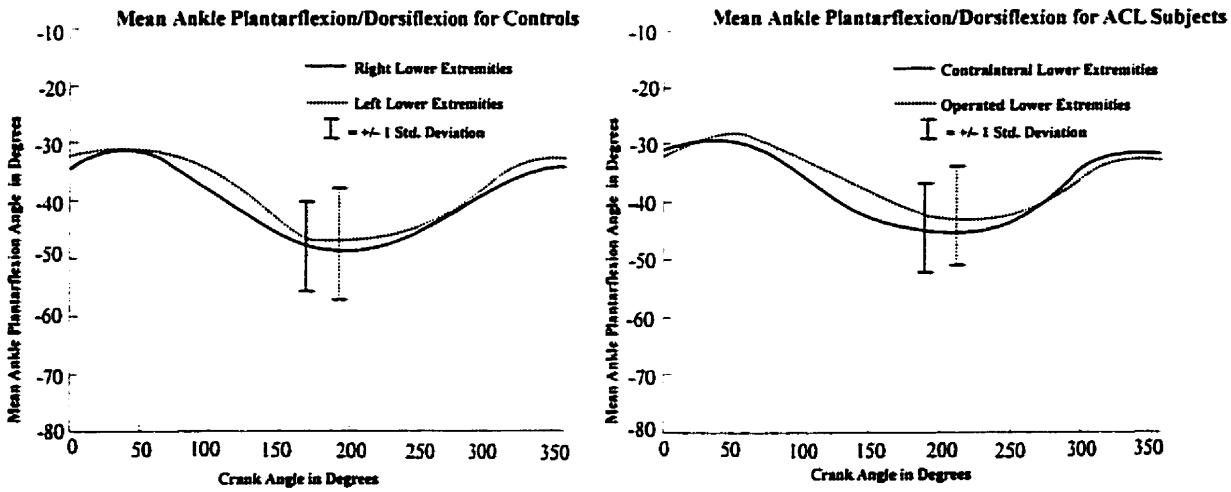


Figure 4.14: Mean ankle plantarflexion angles for the control and ACL-reconstructed groups.

Figure 4.14 displays the mean ankle plantarflexion angles for the subject groups. There was higher intersubject variability for the ankle angle results. The ankle

plantarflexion angle was plotted against the knee flexion angle and displayed in Figure 4.15. From Figure 4.15, the ACL-reconstructed subjects had more ankle dorsiflexion and knee flexion, and less ankle plantarflexion and knee extension than the control subjects.

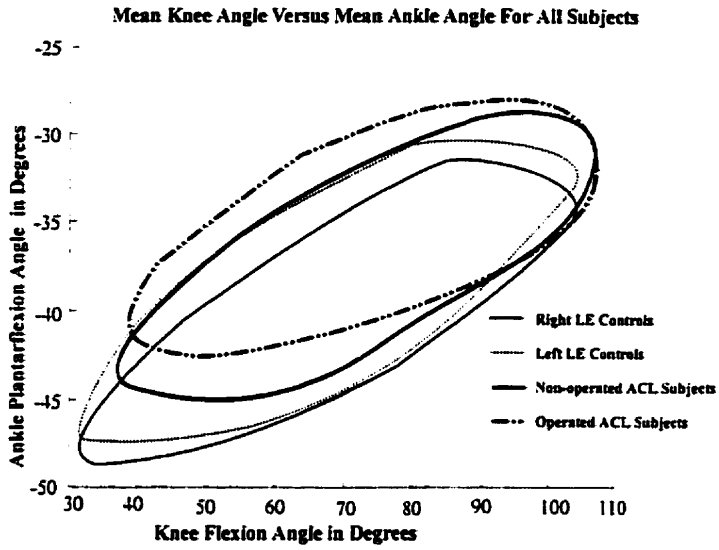


Figure 4.15: Angle-angle plot for the ankle versus the knee angle.

Table 4.6 displays the means (95% CI) for the motion in the frontal plane during the cycle test. A negative value indicates the knee was in a varus position, and a positive value indicates the knee was in a valgus position.

Table 4.6: Mean peak valgus angle (95% CI) at the knee during cycling

Controls	**Right Knee**	**Left Knee**
	-0.2° (-4.8°, 4.8°)	3.3° (-5.3°, 12.0°)
ACL-reconstructed	**Contralateral Knee**	**Operated Knee**
	-0.3° (-3.3°, 2.7°)	2.0° (-0.3°, 4.3°)

5. DISCUSSION

The current study confirms previous documentation of persistent weakness of the QF muscle post-ACL reconstruction. Thus, restoring the stability of the knee by surgical reconstruction of the ACL may not result in a full recovery of strength of the QF muscle. The objectives of this research study were to test hypotheses relating to theories of QF muscle weakness after reconstruction of the ACL in healthy controls and ACL-reconstructed subjects (approximately six months post-surgery): specifically, QF muscle inhibition (using a twitch interpolation technique) and a subconscious change in day-to-day behavior (the cycling test). This is the first study in which the kinematics and kinetics of cycling have been measured in an ACL-reconstructed population.

The three *a priori* hypotheses tested showed no increase in MI of the QF muscle for the ACL-reconstructed subjects (when compared to the controls), no correlation between MI of the QF and knee joint laxity, and no significant difference in the between peak $F_{(pedal)}z$ force between the controls and ACL-reconstructed subjects. When the secondary hypothesis was explored for the cycling test, the peak $F_{(pedal)}z$ force occurred significantly later in the pedal revolution for the ACL-reconstructed subjects. This secondary result suggests that the ACL-reconstructed subjects were using a different motor coordination strategy to pedal a bicycle (at a relatively low load and cadence) when compared to the control subjects.

There were limitations to this study, including a relatively small sample size (for both the controls and ACL-reconstructed subjects), a single-test experimental design,

incomplete pedal force data, and a short term follow-up. The following sections will compare the QF muscle strength, the QF MI, and the cycling test results with those reported in the literature, and to the hypotheses stated in chapter 2. Also, the study limitations are further outlined and possible sources of error are presented. Finally, the key findings of the study are summarized, and suggestions for further research are discussed.

5.1 Study Findings

5.1.1 Inhibition of the quadriceps femoris muscle

In the literature reviewed, some investigators have suggested that a reduced activation of the QF muscle may compound weakness of the QF muscle following ACL injury and/or reconstruction (Suter et al., 1998a; Hurley et al., 1992). Other investigators have reported significant weakness of the QF muscle without significant inhibition of the QF muscle following ACL reconstruction (Snyder-Mackler et al., 1994; Pfeifer and Banzer, 1999).

In this study, it was hypothesized that the ACL-reconstructed subjects would have significantly greater MI of the QF muscle when compared to a group of healthy controls. There was no significant difference in MI of the QF between the two groups. From the results of the 2-way ANOVA used on the MI data, there was no significant group-by-leg interaction ($p=0.5$). The paradigm that the QF muscle weakness and atrophy occurs secondary to MI of the QF muscle was not substantiated in the present study, although the post-surgical follow-up was limited to six months, and no measures of pre-operative

QF muscle strength or QF MI were taken. The ACL-reconstructed subjects tested in this study had a significant percent deficit in isometric QF muscle strength, yet no between-thigh differences in MI.

The ACL-reconstructed group did not have greater MI of the QF when compared to the control group. In fact, the controls had greater MI of the QF when the two means were compared ($p=0.03$). It seems counterintuitive that the ACL-reconstructed group, demonstrating weakness of the operated thigh QF muscle, should have less inhibition of the QF muscle when compared to the control group. If some of the present theories (such as pain and joint effusion) regarding the etiology of MI are true, then it seems reasonable to hypothesize that the levels of MI measured would be greater in the ACL-reconstructed subjects (who may have residual pain and effusion in the operated knee). The level of MI reported for the control group in this study, however, was higher than other control values reported in the literature (refer to Table 2.2, page 36).

The levels of MI of the QF measured in the control population in this study were high compared to the work of previous investigators who also used a twitch interpolation technique. Suter et al. (1996b) measured a mean MI of the QF muscle of 12.8% in 10 control subjects (handy sample) at 60° of knee flexion using a regular Cybex arm. Also, in Huber et al. (1998), a mean MI of the QF muscle of 21.5% was measured at 60° of knee flexion (regular Cybex arm) in 13 subjects with no history of knee injury. Suter et al. (1998a), reported MI values which increased approximately 10% when a JASA was used for twitch interpolation testing of the QF muscle in ACL-deficient subjects at 30° of

knee flexion. No measures of MI of the QF muscles in healthy controls using a JASA were reported in the literature. As stated previously, the control subjects tested in this study were a small, non-random sample (n=7) of individuals with varying activity levels. All subjects, control and ACL-reconstructed, were prepared for the testing in a standardized manner, and were encouraged to perform maximal isometric contractions during the practice and test repetitions.

One explanation for the discrepancy between the measured control MI values and those of the literature could be that some of the control subjects were performing submaximal isometric contractions; this may have caused an overestimation of the measured MI of the QF muscle in such a relatively small sample size. Examination of the variability of average MI for the control population revealed one relatively low value (3%), four values between 28% and 39%, and two relatively high values (51% and 60%). The MI values measured were comparable between legs, and only the interpolated twitch with the highest torque plateau was used for the calculation of the MI. Unfortunately, the possibility that some of the control subjects were performing submaximal contractions cannot be excluded; however, the same assumption must hold true for the ACL-reconstructed population tested in this study.

For the measurement of the resting QF muscle twitch at 65° of knee flexion, the subjects were instructed to relax their thigh. The mass of the leg on the Cybex arm may have reduced the absolute height of the resting twitch, and subsequently increased the calculated amount of MI of the QF muscle. Torque values (in Nm amplified

approximately 20 fold) were examined for both the control and ACL-reconstructed subject groups to see if there was a discrepancy in the mass of the leg on the Cybex arm between the two groups. The mean value (95% CI) for the control subjects was 3.75 (2.91, 4.60), while the mean value for the ACL-reconstructed subjects was 2.78 (2.00, 3.56). The range of resting twitch torque (RTT) values measured was 75 to 188 for the controls, and 75 to 201 for the ACL-reconstructed subjects. Compared to the RTT, the torque measured for the leg mass was small. Also, there did not appear to be a difference in leg mass between the controls and ACL-reconstructed subjects.

A third possible explanation for the higher than previously documented control MI values may be due to the order of testing between the interpolated twitch torque (ITT) and the resting twitch torque (RTT). When the ITT is measured, the muscle is potentiated from the contraction. The effects of potentiation have been examined as a function of knee angle. Suter et al. (1996b) concluded that potentiation was highest immediately following contraction, and that the potentiation effect was greatest for a knee angle of 60°. At a knee angle of 60°, the effects of potentiation were still present at 120 seconds but were considerably lower than at 5 seconds post-twitch. In this study, the first RTT was recorded for all subjects (control and ACL-reconstructed) 2 minutes following the ITT measurement, and subsequent RTT's were measured at 2-minute intervals following the first resting twitch. Too great of a time interval passed between the MVC and the resting twitch, therefore the RTT's were not potentiated. The reduced effect of potentiation may account for overestimation in the measured MI for all subjects (control and ACL-reconstructed) tested in this study.

The MI values of the QF muscle calculated for the ACL-reconstructed population tested in this study were comparable to those previously reported in the literature. Suter et al. (1999) measured MI of the QF muscle (potentiated, doublet twitches at 30° of knee flexion using a JASA) in 22 ACL-reconstructed subjects, and reported mean values of approximately 17.5% for the operated thighs and 19% for the non-operated thighs tested. The mean measured levels of MI of the QF muscle (95% CI) for the ACL-reconstructed group tested in the present study were 21.22% (11.06, 31.37) for the operated leg and 20.93% (9.84, 32.03) for the contralateral limb. Given the differences in methodology between the two studies (in particular, knee angle and potentiation), the MI of the QF muscle calculated for the ACL-reconstructed group in this study were comparable to Suter et al. (1999). In the same abstract, Suter et al. (1999) also measured MI in 24 ACL-deficient individuals, and reported mean values of approximately 28% (injured thigh) and 27% (contralateral thigh). Unfortunately the study design of Suter et al. (1999) was cross-sectional; no cause and effect relationship between the ACL-deficient and ACL-reconstructed groups can be determined given this design. Also, the study apparently did not control for time between injury and testing (ACL-deficient group), and for time between reconstruction and testing (ACL-reconstructed group). Control of subjects within testing time intervals is important for drawing conclusions from experimental results. For the present study, ACL-reconstructed subjects were tested at a mean 7.5 months post-surgery. This time period is critical following an accelerated rehabilitation program as most ACL-reconstructed individuals are preparing to return to sport.

In the literature reviewed, there was evidence to support the notion that level of MI of the QF muscle decreases following reconstruction of the ACL. Weakness of the QF muscle following this surgery may be mediated by another mechanism. Snyder-Mackler (1994) theorized (using the results from an ACL-reconstructed population) that the presence of QF muscle weakness without reduced activation may be due to a selective atrophy of the inhibited muscle fibres. Conversely, Huber et al. (1998) reasoned that the reduced amount of muscle inhibition following knee injury may be due to the additional recruitment of motor units to compensate for the QF muscle atrophy during the period of injury and resultant detraining. The ACL-reconstructed subjects tested in this study, despite having a significant between-thigh QF muscle strength deficit, had a trend towards decreased amounts of MI compared to the control group. In the present study, QF muscle atrophy was not quantified; thus, it cannot be concluded that the ACL-reconstructed subjects had significant atrophy of the QF muscle compared to the controls. In Table 5.1, the between-thigh differences in MI are displayed for the ACL-reconstructed subjects. Although there was no leg effect for the ACL-reconstructed subjects when the data were analyzed, there was considerable variability between the ACL-reconstructed subjects tested. Part of the variability of the recorded MI values may be due to the r^2 value of 0.777 (representative of validity) reported for the interpolated twitch test in the literature (Suter et al., 1997).

Table 5.1: Between-thigh differences (non-operated minus operated) in MI of the QF muscle for the ACL-reconstructed subjects

Subject:	**1**	**2**	**3**	**4**	**5**	**6**	**7**	**8**	**9**	**10**	**11**
MI:	-15%	0.1%	-5%	17%	5%	-12%	-12%	-9%	22%	-0.5%	6%

One theory of why QF MI occurs following ACL injury was that rupture of the ACL may precipitate knee joint instability through loss of the mechanical support between the tibia and femur. As proposed by Solomonow (1987), the mechanoreceptors located in the ACL function to regulate excessive anterior tibial translation, and loss of this afferent information may trigger an increased responsiveness in the remaining periarticular mechanoreceptors (those of the anterior knee joint capsule, for example). During the stance phase of gait, the anterior tibial translation that occurs with knee extension in an ACL-deficient knee may cause inhibition of the QF to prevent further tibial translation (relative to the femur) and instability (Andriacchi, 1990). Restoring the mechanical stability of the knee by reconstructing the ACL may decrease anterior/posterior knee joint laxity.

In regard to the second *a priori* hypothesis tested in this study, there was no significant correlation between anterior knee joint laxity (mm) and inhibition of the QF muscle (%) ($r=0.23$). This result does not support the theories presented in the preceding paragraph by Solomonow (1987) and Andriacchi (1990). Both groups (control and ACL-reconstructed) used a JASA during the twitch interpolation test. The JASA was designed to reduce the anterior shear of the tibia relative to the femur during knee extension

dynamometer testing (Johnson, 1982). Thus, because of the methodology used in this study, the QF muscle was likely not inhibited by excessive anterior tibial translation during the interpolated twitch test. Anterior knee joint laxity and resultant inhibition of the QF muscle is probably more complex than a relatively simple stretch reflex of the anterior knee joint mechanoreceptors. The literature reviewed suggests that MI of the QF muscle decreases after ACL reconstruction (Snyder-Mackler et al., 1994; Suter et al., 1999). A prospective study of QF muscle strength and muscle inhibition pre and post-ACL reconstruction may help clarify how surgery may decrease inhibition of the QF muscle. Further investigation, using animal models, of the development of QF muscle inhibition and/or atrophy immediately post-ACL transection may also help elucidate the inter-relation of these two variables.

In the literature reviewed, increased anterior knee joint laxity was thought to reduce the strength of the QF muscle by two mechanisms: a decrease in the moment arm of the QF muscle, and inhibition of the QF muscle through stimulation of the anterior knee joint capsule mechanoreceptors. The ACL-reconstructed subjects tested had greater amounts of knee laxity (measured with the KT 2000) compared to the control group (refer to page 69). When between-knee laxity was compared to between-thigh strength (Nm/kg) (see Figure 4.4, page 68), the ACL-reconstructed subjects with more between-knee laxity appeared to have a greater deficit in QF muscle strength. This study result may support the hypothesis that chronic knee laxity may have a cumulative effect on reducing the strength of the QF muscle, either through inhibition of the QF muscle, or through changes to the moment arm of the QF muscle. Because a JASA was used in this study for all

subjects and all knee extensor tests, any acute effects of increased anterior knee laxity (decreasing the moment arm of the QF muscle during the Cybex test, for example) should have been minimized during the knee extensor testing. When between-thigh strength was compared to between-thigh MI of the QF (see Figure 4.3, page 68), the two variables did not appear to be related.

There is a possibility that the type of athletic activity was different between the two groups. Most of the subjects in the ACL-reconstructed group were regularly performing resisted QF muscle strengthening exercises as part of a maintenance rehabilitation program. The type of athletic activity that the controls were performing was not collected. Regular resistance training increases muscle activation levels; thus, part of the discrepancy in MI values (between the controls and ACL-reconstructed subjects) may be due to differences in exercise routines (Jones et al., 1989). When the hours per week of athletic participation (refer to Table 4.1, page 65) were compared to the percent deficit of QF muscle strength, the two variables did not appear to be related. The hours per week of athletic participation only accounts for the activity levels of the study participants during the testing period. For the ACL-reconstructed subjects, an estimation of the total hours of rehabilitation exercise since surgery would also have been useful information to collect.

There is evidence in the literature to support MI of the QF muscle in ACL-deficient individuals (Suter et al., 1998a; Hurley et al., 1992; Snyder-Mackler et al., 1995). An acutely ACL-injured knee may have effusion and pain; there is evidence in the literature

that these signs and symptoms may lead to reflex inhibition of the QF (Spencer et al., 1984; Suter et al., 1998b). There was no significant difference in MI of the QF muscle between the non-operated and operated thighs of the ACL-reconstructed subjects tested in this study, even though some ACL reconstructed subjects reported increased pain during the twitch interpolation testing. This result does not support the hypothesis that reflex inhibition of QF muscle is perpetuating muscle weakness 6 months following ACL reconstruction. The ACL-reconstructed subjects had significantly decreased operated-thigh strength of the QF muscle without significant amounts of MI of the QF when compared to the control subjects. It was postulated earlier in the discussion that atrophy of the QF muscle (although not quantified in this study), not MI, may have contributed to the operated thigh QF weakness in the ACL-reconstructed subjects.

5.1.2 Strength measures

In chapter one, several theories regarding the origin of weakness of the QF muscle were introduced. Two of the theories were tested directly in this thesis, QF muscle inhibition and a subconscious change in day to day behavior (cycling test). The strength tests used quantified the amount of weakness of the QF muscle in an ACL-reconstructed population (approximately six months post-surgery) compared to a group of control subjects.

One the primary goals of rehabilitation following ACL reconstruction is the restoration of full strength of the QF muscle in the operated thigh without injuring the ACL graft. The operated thigh QF muscle strength of the ACL-reconstructed group was

significantly less than that of the control group for the isometric knee extension test at 65° of knee flexion. The literature reviewed supports this result (Maitland et al., 1993; Rosenberg et al., 1992; Sachs et al., 1989; Shelbourne and Nitz, 1990; Yasuda et al., 1992). There also appeared to be differences in the operated thigh strength of the QF muscle for the isokinetic strength tests at 90°/s and 240°/s (see Figure 4.5, page 70). For the ACL-reconstructed subjects, the mean difference in strength was greatest for the isometric test (23.8% versus 14.4% and 16.4% for the 90°/s and 240°/s tests, respectively). The literature reviewed also supports this result (Maitland et al., 1993).

Perrine and Edgerton (1978) examined the force-velocity relationship in healthy male and female subjects (n=15) using seven test velocities ranging from isometric to 288°/s. The maximal torque was measured at 30° of flexion for all test speeds. The authors compared their study results to the force-velocity relation found for isolated animal muscle, in which the force rises increasingly more sharply as velocities decrease until a maximum is attained at zero speed (Hill, 1938). The results from Perrine and Edgerton (1978) showed a sharply diminishing rate of rise of force as the test velocities decreased, especially at speeds below approximately 90°/s. The authors postulated that a neural mechanism restricting maximal muscle tension in-vivo may be responsible for the marked difference between the in-vitro and in-vivo force-velocity relations. The result from Perrine and Edgerton (1978) may support the decline in torque recorded for the operated thigh of the ACL-reconstructed subjects tested (Figure 4.5, page 70), although there was not a significant difference in between-thigh MI of the QF muscle for the ACL-reconstructed subjects (Figure 4.1, page 66).

Despite completion of rehabilitation programs post-surgery, operated thigh weakness of the QF was present in the ACL-reconstructed subjects tested. To protect the healing ACL graft from excessive stretching, the exercises used to strengthen the QF muscle were prescribed to minimize the anterior shear force of the tibia relative to the femur. These exercises are predominantly CKC, that is, the foot is fixed and the lower extremity moves relative to the foot (can be performed in a seated or standing position) (Steindler, 1955). In the literature, the efficacy of CKC exercises for strengthening the QF muscle has been questioned (Synder-Mackler et al., 1994). The ACL-reconstructed subjects had a mean percent deficit of QF muscle strength of 23.8% at 6 months post-surgery. The exercise regime used by the ACL-reconstructed subjects in this study (see Table 3.1, page 47, and Figure 2.1, page 15) was likely not adequate for increasing the strength of the QF muscle (assuming that the pre-operative percent deficit of QF muscle strength was not significantly greater than 24%). For strengthening of the hamstring muscle group, OKC exercises are prescribed post-ACL reconstruction when the individual has adequate knee ROM. Although hamstring strength results were not reported in this study, according to Yasuda et al. (1992), no significant deficits in hamstring muscle strength were reported six months following ACL reconstruction.

5.3 The Bike Test

5.3.1 Kinematics

Figure 4.11 (page 75) shows that there was approximately a 10° difference in knee extension (at BDC) between the controls (SD +/- 8°) and ACL-reconstructed subjects (SD +/- 5°). To further explore the kinematic differences between the two groups tested, the

thigh and ankle angles were examined both separately (Figures 4.12 and 4.14, pages 76 and 77 respectively), and relative to the knee angle (Figures 4.13 and 4.15, pages 77 and 78 respectively).

The following paragraph is a summary of the kinematic differences observed between the controls and ACL-reconstructed subjects during the cycling test (at BDC). When the thigh angles were plotted against the knee angles (Figure 4.13, page 77), the curves for the ACL-reconstructed subjects shifted towards decreased knee extension (at BDC) when compared to the controls. The ACL-reconstructed subjects also had approximately 5° less ankle plantarflexion at BDC (Figure 4.14. page 77) although the variability was high for both subject groups.

The ankle angle results were the most variable. Differences in pedaling technique may explain some of the variability in the angle angles reported (Faria and Cavanagh, 1978). If the differences in the knee extension angles between the 2 groups were mitigated solely by changes in the ankle angle, one would expect the ACL-reconstructed subjects to have greater amounts of ankle plantarflexion.

Two possible hypotheses were explored to explain the kinematic differences between the controls and ACL-reconstructed subjects. The first was there may have been a systematic error in the seat height between the ACL-reconstructed subjects and the controls, and the second was that the kinematic differences were secondary to changes in pedaling pattern and/or muscular coordination between the two groups. In regard to seat

height, error may have occurred with the goniometer measurement. To standardize the ankle position during the knee angle measurements, each participant was instructed to let his or her ankle drop into approximately 90° of dorsiflexion with the pedal in the BDC postion. Differences in Achilles tendon length and/or leg muscle length between the participants may have also introduced error into the measurement of seat height. Ericson et al. (1988) examined kinematic changes in the lower extremities during cycling with 3 seat height changes (102, 113, and 120% of the distance between the ischial tuberosity and the medial malleolus). To investigate the validity of the seat height measurement used in the present study, the method was compared (using a handy sample of 5 participants) to that of Ericson et al. (1988). Following leg length measurement, seat height (using the goniometer method of the present study) was adjusted. After the participant dismounted the bicycle, the seat height was measured as the greatest distance from the seat surface to the centre of the upper pedal surface in a straight line along the seat post and crank. For the 5 participants, the seat height (using the goniometer method of the present study) ranged from 116% to 120% of leg length (mean 118%).

Another possibility to refute the hypothesis of a systematic error in seat height adjustment was pelvic motion. If the ACL-reconstructed subjects displayed increased pelvic motion in the sagittal plane, this would support the existence of kinematic differences between the two groups tested. Although no pelvic marker information was collected, the z-lab coordinate positions of the greater trochanter marker were examined (maximum minus minimum value) between the controls and ACL-reconstructed subjects. For the controls, the mean between-thigh z-lab coordinate position (95% CI) was

0.0596m (0.05, 0.07). For the ACL-reconstructed subjects, the value was 0.0599m (0.05, 0.07). No further statistical tests were used, and it was concluded that the ACL-reconstructed subjects were not using increased pelvic motion while cycling when compared to the control subjects

Another explanation for the kinematic differences between the controls and ACL-reconstructed subjects was that the ACL-reconstructed subjects had increased flexor muscle use when cycling compared to the controls. In Figure 5.1, the mean ROM of the hip, knee and ankle angles for the controls and ACL-reconstructed subjects tested in this study were compared to study results reported by Ericson et al. (1988).

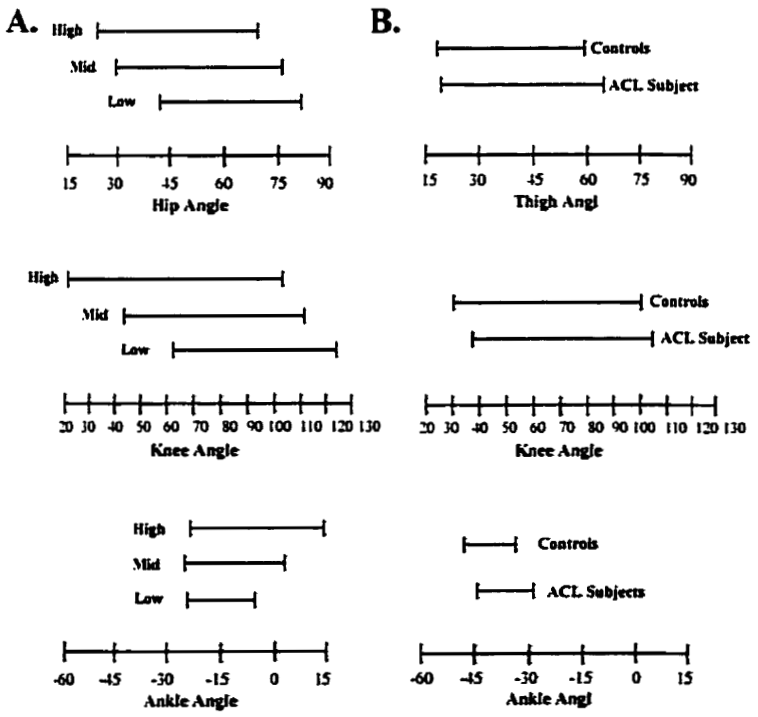


Figure 5.1: (A) Mean ROM at the hip, knee and ankle for 3 different seat heights (high, mid and low) (Ericson et al., 1988). (B) Mean ROM at the hip (thigh angle), knee and ankle from the present study.

The lower extremities during cycling may be considered as a system of rigid segments linked together, connected by hinge joints. If seat height was systematically lower for the ACL-reconstructed subjects, one would expect the pattern of changes between the ACL-reconstructed subjects and controls to resemble that of the middle and high seat positions. Although the mean knee ROM was slightly less, the mean thigh and ankle ROM were increased for the ACL-reconstructed subjects when compared to the controls. Also, for all 3 angles measured, the joint ROM for the ACL-reconstructed subject was offset from that of the controls towards increased flexion of the lower extremities. In conclusion, the fact that there may have been a systematic error in seat height between the ACL-reconstructed subjects and controls tested in this study cannot be excluded. Given the pattern of joint angle changes, one cannot also exclude the possibility that the ACL-reconstructed subjects were using a different muscular coordination strategy when cycling. This hypothesis will be explored further in the next section.

5.3.2 Kinetics of cycling

The $F_{(pedal)}z$ pedal forces were complete for all subjects. However, without the $F_{(pedal)}x$ component, conversion from a pedal to a lab coordinate system was not possible for all the study participants. Thus, the $F_{(pedal)}z$ forces were compared between the controls and ACL-reconstructed subjects in the pedal coordinate system. Unfortunately, pedal force results reported in the literature reviewed were reported in lab coordinates, making comparisons difficult. In regard to the *a priori* hypothesis for the cycling data, there was no significant between-leg difference in peak $F_{(pedal)}z$ force between the controls and

ACL-reconstructed subjects. There was a significant difference in the timing of the peak $F_{(pedal)}z$ force during a cycle revolution between the ACL-reconstructed subject group and the controls.

In the cycling literature reviewed, a result such as this (pedal force timing) has not been previously reported. Brown and Kukulka (1993) elicited cyclic lower extremity flexor responses in 10 neurologically intact males when pedaling a bicycle. When perturbed (using electrical stimulation of the tibial nerve), a reflex pattern of hip/knee/ankle flexion was simulated. The authors found the flexor response contributed most to forward progression of the crank when it was stimulated near BDC of a crank revolution (assisted the recovery phase to return the pedal to TDC). If the extensor musculature (includes quadriceps femoris, gluteus maximus, and gastrocnemius muscle groups) typically predominates during the first third of a pedal revolution, perhaps some of the ACL-reconstructed subjects were using a different muscle coordination strategy when pedaling. From EMG studies of the pedaling motion, hamstring muscle activation was greatest from approximately 90° to 200° of a pedal revolution (Faria and Cavanagh, 1978). Since the peak normal pedal force for the ACL-reconstructed subjects occurred at approximately 140°, it is possible that increased hamstring muscle use may be responsible for the change in pedaling technique. Also, in support of this hypothesis, the joint ROM for the ACL-reconstructed subjects was offset from that of the controls towards increased hip ROM and increased flexion of the lower extremities (refer to Figure 5.1, page 94).

Figures 4.7 (page 73) displayed the pedal forces for the complete subject files. From the literature reviewed, the $F_{(lab)}x$ pedal force was positive during the first half of the pedal revolution and negative for the second half of the pedal revolution if the subjects were wearing cleated shoes (Gregor et al. 1985; Davis and Hull, 1981; Caldwell et al., 1999). In this study, the $F_{(lab)}x$ pedal force remained close to zero during the latter half of the pedal revolution. The cycling literature supported this result from the present study (Fleming et al. 1998; Hull and Davis 1981). According to Davis and Hull (1981), use of a shoe with cleats was thought to enhance activity of the flexor muscle groups. For the knee forces for the complete subject trials (Figure 4.8, page 74), the $F_{(shank)}x$ knee force was positive throughout the pedal revolution. The cycling literature supported this result from the present study (Ruby et al., 1992). During this portion of a pedal revolution, due to increased activity of the QF muscle, a positive $F_{(shank)}x$ force seems plausible as contraction of the QF muscle creates an anterior shear force of the tibia relative to the femur.

According to Figure 4.9 (page 74), the ACL-reconstructed subjects (operated legs of the subjects) had less positive operated leg crank torque when compared to the controls. Decreased QF muscle use may account for the differences in crank torque between the groups. Of note for the crank torque results is the negative crank torque reported for the ACL-reconstructed subjects (non-operated legs and operated legs). Negative crank torque is representative of the torque that is retarding or slowing the forward progression of the pedal during a revolution. If the ACL-reconstructed subjects were demonstrating increased flexor muscle use in the backstroke (BDC to TDC), one would expect the crank

torque to be less negative during this portion of the pedal revolution. The pedaling efficiency may have been reduced with the exclusion of cleated shoes; thus, the differences in negative crank torque appear negligible between the controls and ACL-reconstructed subjects.

For the knee moment data (Figure 4.10, page 74), the calculated moments were comparable to those of the literature for the first half of a pedal revolution. However, for the latter half of the pedal revolution, the net moment was close to zero. In the literature, no published studies were found which calculated knee moments without using shoes with cleats. Cleats may have allowed the subjects to enhance flexor muscle activity from BDC to TDC, increasing negative $F_{(lab)}x$ pedal force during this portion of the pedal revolution. For the subjects tested, the $F_{(lab)}z$ pedal force was also negligible during this portion of the pedal revolution. Therefore, the resultant knee moment was likely correct given the kinetic contributions. For the operated thighs of the ACL-reconstructed subjects (n=3), the net extensor knee moment appeared to be less than that for the contralateral thigh of the ACL-reconstructed subjects (n=5). The mean percent deficit in QF muscle strength for the 3 subjects in the operated thigh group were 52%, 32% and 29%. Although it is a between-subject comparison of small numbers, the decrease in the net extensor moment at the knee for the operated thigh group may correspond to the weakness of the QF muscle in the ACL-reconstructed subjects tested. Interestingly, the ACL-reconstructed subject with the mean percent deficit of 52% had a net operated leg flexor knee moment and positive $F_{(shank)}z$ tibiofemoral joint force from BDC to TDC (literally, pulling up in the pedal) (refer to the Appendix, Part B, page 126, subject six).

This subject was likely using increased hamstring muscle force to aid the ipsilateral weakened QF muscle to rotate the bicycle crank.

5.2 Sources of Error

The control group was a non-random sample chosen relative to the ACL-reconstructed population for gender, approximate age (+/- 5 years), and activity level (hours per week of athletic participation). The use of the hours per week of athletic participation questionnaire may not have been the most effective tool for describing the activity levels between the control and ACL-reconstructed groups. The present study focused on strength of the QF muscle; therefore, knowledge of the type of physical exercise (ie aerobic versus strength training) the subjects were performing is important to the study results. From conversation with the participants, most of the ACL-reconstructed subjects were regularly performing lower extremity strengthening exercises; the type of regular activity that the control subjects were participating in during the study period was not recorded.

All ACL-reconstructed subjects who volunteered for the study were patients from the University of Calgary Sport Medicine Centre. Since this particular clinic specializes in "Sport Medicine", and is affiliated with a University, the ACL-reconstructed population tested may be more athletic and of a higher socioeconomic status than other ACL-injured individuals, and thus may represent a subgroup of the ACL-reconstructed population. The ACL-reconstructed subjects were contacted by phone and asked to participate in the study. Using this method, selection bias was introduced. For example, it is possible that

only the ACL-reconstructed subjects interested in a more comprehensive strength evaluation volunteered for the study. Two of the ACL-reconstructed subjects had surgical procedures after their reconstructive surgery (subject 1 – gentle manipulation (to increase knee flexion) after the ACL reconstruction for arthrofibrosis; and subject 8 - lateral menisectomy), and both demonstrated a greater than 20% (percent) deficit in strength of the QF at 65° of knee flexion. Inclusion of these two subjects into the ACL-reconstructed subject data may have increased the variability of the results but it was noted that the results reported for these subjects were comparable with that of the remainder of the ACL-reconstructed subjects tested.

The camera positions, the calibration procedure, the visibility of the markers, the size of the markers, and the fixation of the markers may affect the accuracy of kinematic data collection. The cameras were positioned at staggered heights around the bicycle-rider volume, and were calibrated with the calibration cube at the centre of the camera's field of view. Large markers (2.5cm) were used to improve marker visibility, and thus improve the tracking of the data on EVa. The markers were secured to the subject's skin with fabric backing and double-sided tape, but, due to leg motion and skin perspiration, additional tape was required for some subjects to re-attach markers.

To estimate the amount of error in the kinematic measurement of the bike testing volume, the cube calibration data were tracked as a trial using EVa. The ranges of error between the cube calibration positions entered in the project file and that calculated by

EVa were 0.004 to 0.0106 cm for the x Lab coordinate, 0.0003 to 0.327 cm for the y Lab coordinate, and 0.002 to 0.105 cm for the x Lab coordinate.

5.4 Summary and Conclusions

Weakness of the QF muscle after reconstruction of the ACL is a multi-factorial problem. The ACL subjects tested in this study had a significant decrease in operated leg QF muscle strength when compared to the control subjects. Although the ACL-reconstructed subjects demonstrated unilateral QF muscle weakness, the ACL-reconstructed subjects had no significant between-thigh MI of the QF muscle. Thus, in the ACL-reconstructed population tested in this study, inhibition of the operated thigh QF muscle did not appear to contribute to the decreased strength of the operated thigh QF muscle. Also, for the subjects tested in this study, MI of the QF was not correlated with knee laxity. At approximately six months following ACL reconstruction, the decreased strength of the QF muscle may represent muscle atrophy, although this variable was not measured in the present study. For the ACL-reconstructed subjects tested, it was also postulated that the post-operative CKC strengthening exercises for the QF muscle may not have been adequate for muscle fibre hypertrophy.

Delorme (1945) concluded that a weak, atrophied QF muscle should not be subjected to endurance-building exercises, until the muscle power has been restored to normal by power-building exercises (low repetition, high resistance). The efficacy of CKC QF muscle strengthening exercises post-ACL reconstruction should be further explored. The present emphasis on high repetition, low resistance QF muscle strengthening exercises

needs to be seriously considered in view of persistent weakness of the QF muscle post-ACL reconstruction.

There appears to be a change in some of the motor coordination strategies for gait and other automatic-type activities following ACL injury. If this change is precipitated by the loss of the mechanoreceptor input from the torn ACL, replacing the ligament with other tissue will not fully compensate for this afferent loss. In the present study, the joint ROM for the ACL-reconstructed subjects (while cycling) was offset from that of the controls towards increased flexion of the lower extremities. Also, the peak pedal z force for the ACL-reconstructed subjects occurred significantly later in a pedal revolution when compared to the control subjects. These results may indicate that the ACL-reconstructed subjects were using increased flexor muscle activity to generate power while cycling, but the lack of additional, complete kinetic data for all the subjects tested makes this hypothesis difficult to confirm.

Because cycling represents a motor task ideal for investigating bipedal coordination, a follow-up study using a larger sample size (preferably a prospective design, with pre and post-operative measurements) and different loads and cadences should be performed in an ACL-reconstructed population.

In summary, through testing some of the theories of weakness of the QF muscle, we have gained insight into the complex interactions between decreased QF muscle strength, knee laxity, QF muscle inhibition, and the kinetics and kinematics of motion following

reconstruction of the ACL. The research presented in this thesis may provide a foundation for further studies of QF muscle strength, laxity and inhibition post-knee injury, and for studies of the biomechanics of cycling.

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8. APPENDIX

PART A: Matlab Routines

```
%this file is for the processing of the video data
clear;
ncolumns = 29

%load file
load right_control1.txt, right_control1;

%convert tracked data from cm to metres
right_control = 0.01*right_control1;

%use a fourth order butterworth filter to smooth data
fidfid = [];
dt = 0.01667;
order = 4;
fcut = (9/(0.5*60));
[b,a] = butter(order,fcut);
for j=3:29;
  filfid(:,j)=filtfilt(b,a,right_control(:,j));
end

%extract variables from the matrices and remove y-coordinate
for i=1:1500;
Pedal_raw(:,[1 2]) = filfid(:,[3 5]);
Pedal_rear_raw(:,[1 2]) = filfid(:,[6 8]);
Pedal_front_raw(:,[1 2]) = filfid(:,[9 11]);
Toe_raw(:,[1 2]) = filfid(:,[12 14]);
Ankle_raw(:,[1 2]) = filfid(:,[15 17]);
Antankle_raw(:,[1 2]) = filfid(:,[19 20]);
Antknee_raw(:,[1 2]) = filfid(:,[22 23]);
knee_raw(:,[1 2]) = filfid(:,[24 26]);
TRO_raw(:,[1 2]) = filfid(:,[27 29]);
end

%Calculate crankcentre
crankcentre = mean(Pedal_raw);

%make crankcentre the coordinate system origin (except for the frontal
plane motion)
Pedal(:,1) = Pedal_raw(:,1) - crankcentre(:,1);
Pedal(:,2) = Pedal_raw(:,2) - crankcentre(:,2);
Pedal_rear(:,1) = Pedal_rear_raw(:,1) - crankcentre(:,1);
Pedal_rear(:,2) = Pedal_rear_raw(:,2) - crankcentre(:,2);
Pedal_front(:,1) = Pedal_front_raw(:,1) - crankcentre(:,1);
Pedal_front(:,2) = Pedal_front_raw(:,2) - crankcentre(:,2);
Toe(:,1) = Toe_raw(:,1) - crankcentre(:,1);
Toe(:,2) = Toe_raw(:,2) - crankcentre(:,2);
Ankle(:,1) = Ankle_raw(:,1) - crankcentre(:,1);
Ankle(:,2) = Ankle_raw(:,2) - crankcentre(:,2);
knee(:,1) = knee_raw(:,1) - crankcentre(:,1);
knee(:,2) = knee_raw(:,2) - crankcentre(:,2);
TRO(:,1) = TRO_raw(:,1) - crankcentre(:,1);
```

```
TRO(:,2) = TRO_raw(:,2) - crankcentre(:,2);

%compute the crankangle from the video data
for i=1:1500;
   crankangle(i,:) = atan2(Pedal(i,1), Pedal(i,2));
          if crankangle(i,:) < 0
            crankangle(i,:) = (crankangle(i,:))+(2*pi);
           end
end

%compute the footangle from the filtered data
A = [];
A(:,3)= atan2(Ankle(:,2) - Toe(:,2), Ankle(:,1) - Toe(:,1));
for i=1:1500;
    if A(i,3) < pi
        A(i,3) = -A(i,3) + (2*pi+pi/2);
    end
end

%compute the shankangle from the filtered data
A(:,2) = atan2(knee(:,2) - Ankle(:,2), knee(:,1) - Ankle(:,1));
for i=1:1500;
    if A(i,2) < pi/2
       A(i,2) = A(i,2) + pi;
    end
end

%compute the thighangle from the filtered data
A(:,1) = atan2(TRO(:,2) - knee(:,2), TRO(:,1) - knee(:,1));
for i=1:1500;
    if A(i,1) > pi
       A(i,1) = -A(i,1) + 2*pi;
    end
end

%compute the angle of valgus/varus at the knee
frontal_angle = [];
frontal_angle(:,1) = atan2(Antknee_raw(:,1) - Antankle_raw(:,1),
Antknee_raw(:,2) - Antankle_raw(:,2));
for i=1:1500;
    if frontal_angle(i,:) > pi
              frontal_angle (i,:) = frontal_angle(i,:) - 2*pi;
    end
end

%Convert frontalangle from radians to degrees
frontalangle = frontal_angle*(57.29578);

%compute the pedalangle from the filtered data
pedalangle= atan2(Pedal_front(:,2) - Pedal_rear(:,2), Pedal_front(:,1)
- Pedal_rear(:,1));
for i=1:1500;
    if pedalangle > pi
        pedalangle = -pedalangle + 2*pi;
    end
end
```

```
%Name variables for clarity
footangle = A(:,3);
shankangle = A(:,2);
thighangle = A(:,1);

%Calculate segment angular accelerations
Add(1,:) = [0,0,0];
for i=2:1500-1
   Add(i,:) = [A(i+1,:) - 2*A(i,:) + A(i-1,:)] / dt^2;
end
Add(1500,:) = [0,0,0];

%calculate segment Centre of Mass (CM) positions
%Thigh CM position
R(:,1:2) = (0.433 * (knee - TRO));
%Shank CM position
R(:,3:4) = (0.433 * (Ankle - knee));
%Foot CM position
R(:,5:6) = (0.5 * (Toe - Ankle));

%Calculate mean lenghts of segments
thigh_length = knee - TRO;
for i=1:1500;
    thighlength(i,:) = sqrt((thigh_length(i,1))^2 +
(thigh_length(i,2))^2);
end

shank_length = Ankle - knee;
for i=1:1500;
     shanklength(i,:) = sqrt((shank_length(i,1))^2 +
(shank_length(i,2))^2);
end

foot_length = Toe - Ankle;
for i=1:1500;
     footlength(i,:) = sqrt((foot_length(i,1))^2 +
(foot_length(i,2))^2);
end

%Calculate the ankle angle
for i=1:1500;
  Ankleangle(i,:) =
acos(dot(shank_length(i,:),foot_length(i,:))./((shanklength(i,:)) *
(footlength(i,:))));
end
for i=1:1500;
  Ankleangle(i,:) = (Ankleangle(i,:)*(57.29578)) - 90;
end

%Calculate the knee angle
for i=1:1500;
  Kneeangle(i,:) =
acos(dot(thigh_length(i,:),shank_length(i,:))./((thighlength(i,:)) *
(shanklength(i,:))));
end

Kneeangle = Kneeangle*(57.29578);
```

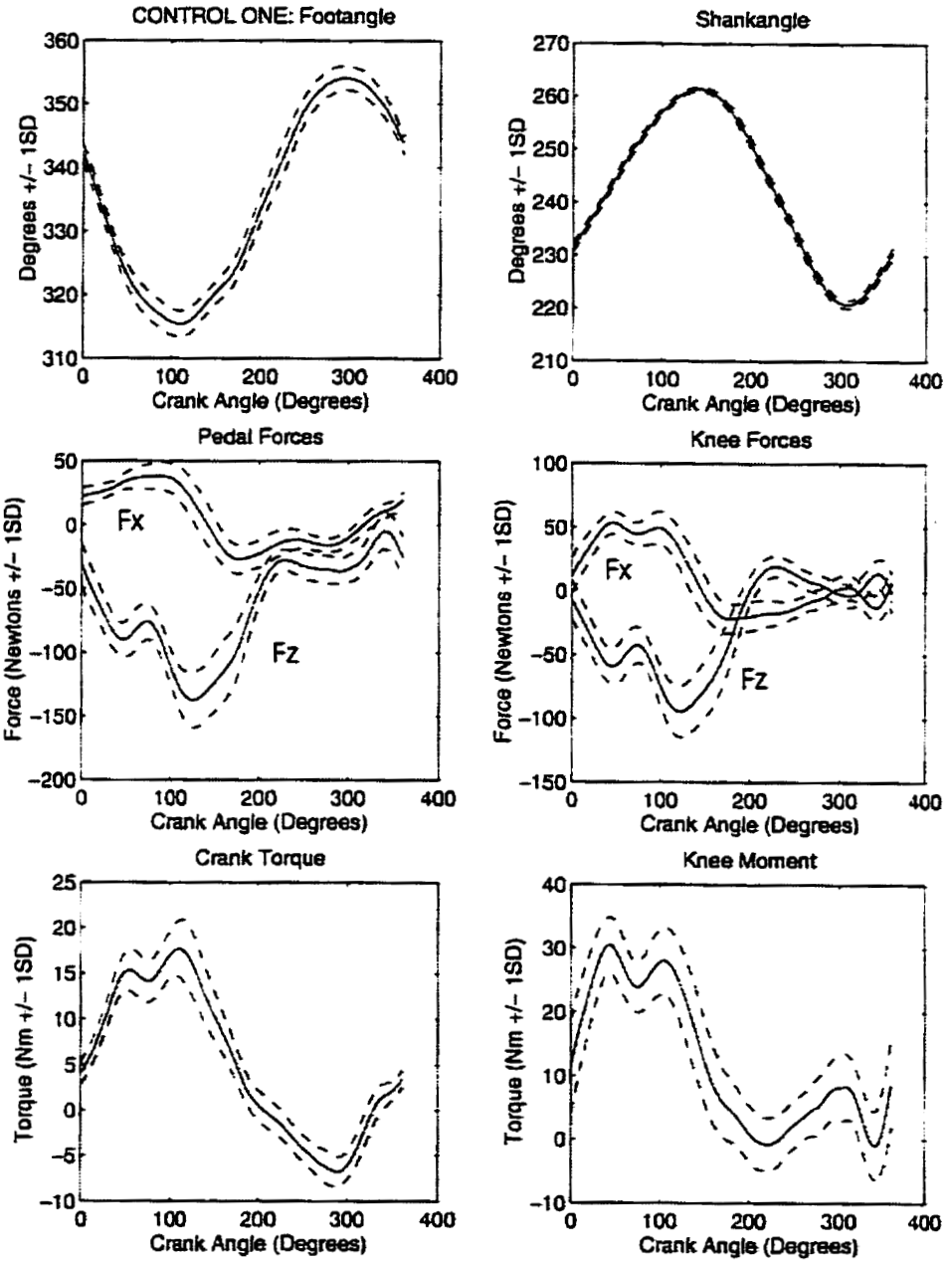
```
thighangle = thighangle*(57.29578);
mthighlength = mean(thighlength)
mshanklength = mean(shanklength)
mfootlength = mean(footlength)

%Calculate linear accelerations
Rdd(1,:) = [0,0,0,0,0,0];
for i=2:1500-1;
    Rdd(i,:) = [R(i+1,:) - 2*R(i,:) + R(i-1,:)] / dt^2;
end
Rdd(1500,:) = [0,0,0,0,0,0];

%load file
load control1.txt, control1;

%This file is for the processing of the pedal data
%Use a fourth order butterworth filter to smooth data
dt = 0.0041667;
order = 4;
fcut = (20/(0.5*240));
[b,a] = butter(order,fcut);
for i=2:5;
  filtered(:,i)=filtfilt(b,a,control1(:,i));
end;

%extract variables from filtered data
for i=2:3;
   RPedalx=filtered(:,2);
   RPedalz=filtered(:,3);
end;

%Decimate data from 240 Hz to 60 Hz
rightPedal_x_60 = decimate(RPedalx,4);
rightPedal_z_60 = decimate(RPedalz,4)

%input pedal gains
rightFx_gain=5
rightFz_gain=5

%eva calibration - convert from bits to volts
rightFx_cal=rightFx_gain/2047.0;
rightFz_cal=rightFz_gain/2047.0;

%calibrate relative to gain
Right_Pedalx=(rightPedal_x_60)*(rightFx_cal);
Right_Pedalz=(rightPedal_z_60)*(rightFz_cal);

%adjust pedal values (volts) for pedal offset
Right_Pedalx_off=[Right_Pedalx] - (1.410);
Right_Pedalz_off=[Right_Pedalz] - (3.430);

%convert Pedal force from volts to newtons
Pedal_force = [Right_Pedalx_off Right_Pedalz_off]
sens_matrix = [-0.017014 0.000315; -0.001456 -0.006056]
cal_matrix = inv(sens_matrix)
cal_force = (cal_matrix*(Pedal_force)')'
rightPedalx = cal_force(:,1);
```

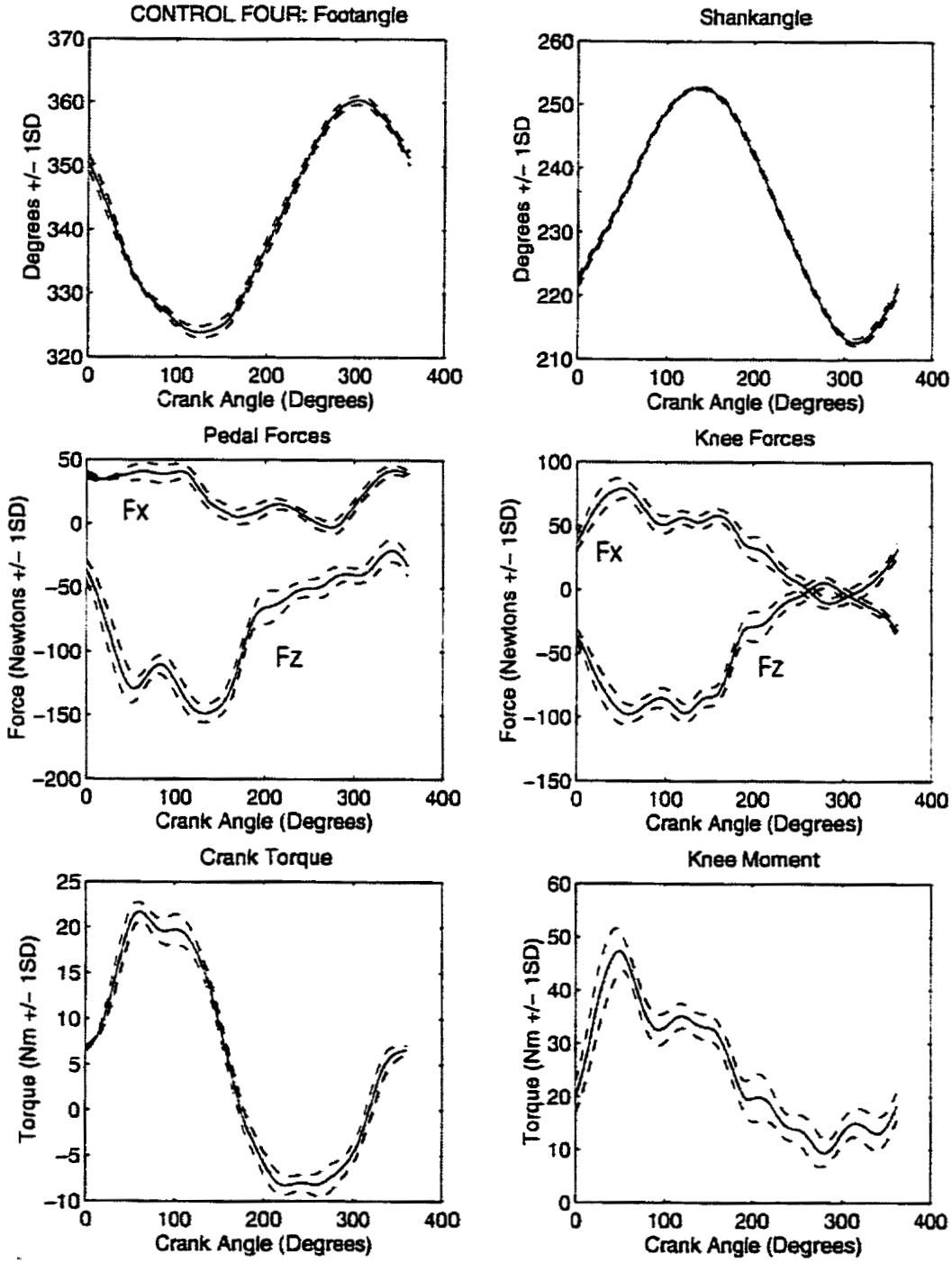
```
rightPedalz = cal_force(:,2);

%Change sign of rightPedal
rightPedalz = -(rightPedalz)

%coordinate transformation of Pedal force
for j=1:1500
          rightPedal_x(j,:) =(cos(pedalangle(j,:))*rightPedalx(j,:))-
          (sin(pedalangle(j,:))*rightPedalz(j,:))
end

for j=1:1500;
          rightPedal_z(j,:) = (sin(pedalangle(j,:))*rightPedalx(j,:))+
          (cos(pedalangle(j,:))*rightPedalz(j,:));
end

%compute crank_torque
r_cktq = [];
for j=1:1500;
   r_cktq(j,:) = (rightPedal_x(j,:)*cos(crankangle(j,:)))*.170 -
(rightPedal_z(j,:)*sin(crankangle(j,:)))*.170;
end

%compute power
power = [];
crk_ang_vel = [];

for i=1:1500-1;
   crk_ang_vel(i,:) = (crankangle(i+1,:) - crankangle(i,:))/0.01667;
end
crk_ang_vel(1500,:) = [0];

for j=1:1500;
  power(j,:) = r_cktq(j,:)*crk_ang_vel(j,:);
end

%This file is for the calculation of the kinetics
%Input anthropoemetric variables
%Subject's mass
m = 67;
%foot_segment_mass
FSM = m .* 0.0145;
%shank_segment_mass
SSM = m * 0.0465;
%Moment of Inertia
Foot_MofI = 0.0033;
Shank_MofI = 0.0463;

%Convert from ffp to fpf
fpfx = -(rightPedal_x);
fpfz = -(rightPedal_z);
%Calculate the force at the ankle in the x direction
fsfx = [];
fsfx =(FSM.*Rdd(:,5))- fpfx;

%calculate the force at the ankle in the z-direction
fsfz = [];
```

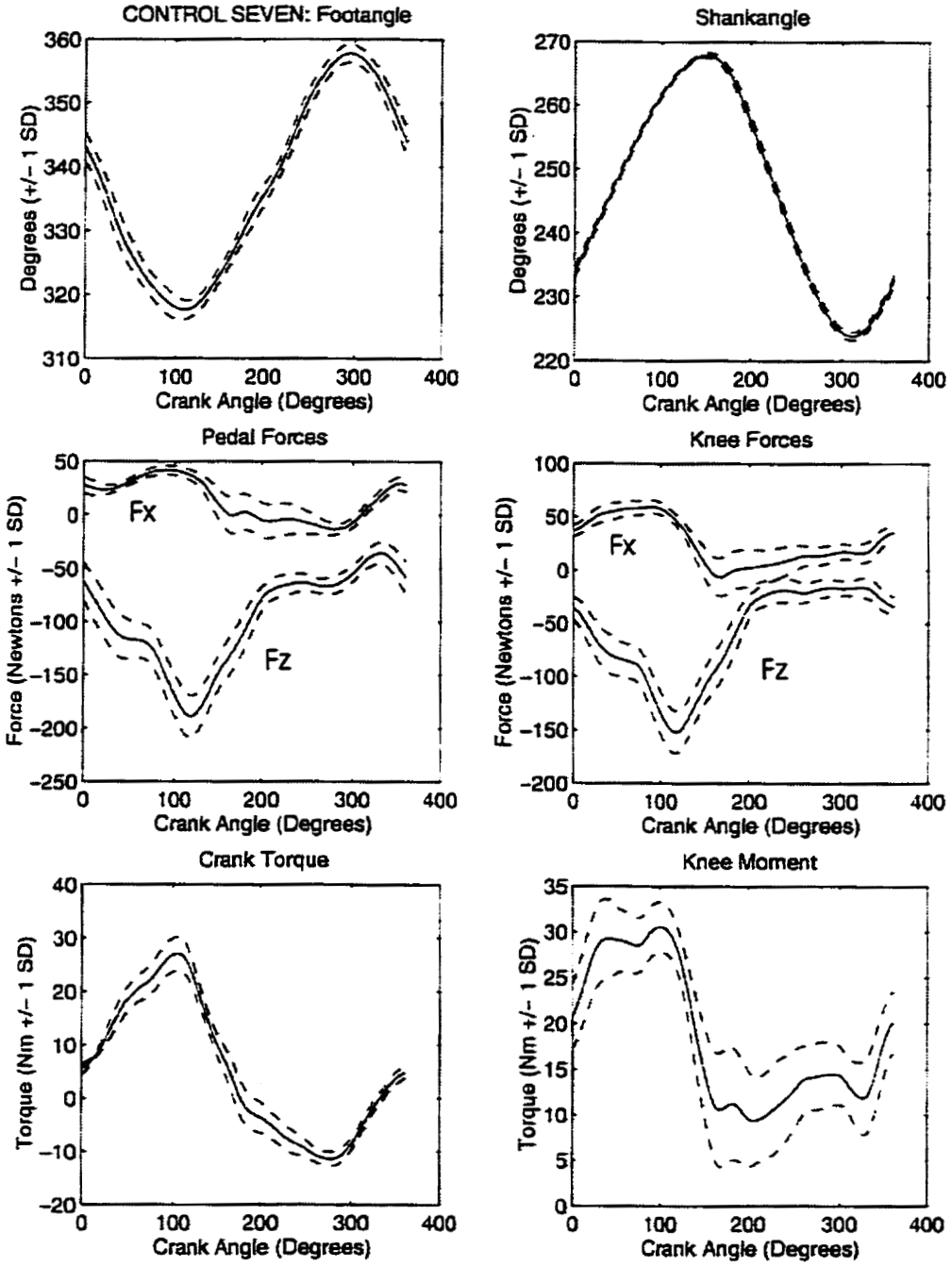
```
fsfz =FSM.*Rdd(:,6)  + (FSM.*9.81) - fpfz;

%Calculate the Moment at the ankle
Mfs = [];
for i=1:1500;
          Mfs(i,:) = -[-
(sin(footangle(i,:)))*fpfx(i,:)*((0.5)*(.1227))] -
          [fpfz(i,:)*((0.5)*(.1227))*cos(footangle(i,:))] -
          [-(sin(footangle(i,:)+pi))*fsfx(i,:)*((0.5)*(.1227))] -
          [fsfz(i,:)*((0.5)*(.1227))*cos(footangle(i,:)+pi)]+
          Foot_MofI*Add(i,3);
end

%where
ffsx = -1*fsfx;
ffsz = -1*fsfz;
Msf = -1*Mfs;

%Calculate the force at the knee in the x direction
ftsx = [];
ftsx =(SSM.*Rdd(:,3))- ffsx;

%calculate the force at the knee in the z direction
ftsz = [];
ftsz =(SSM.*Rdd(:,4)) + (SSM.*9.81)- ffsz;

%Do a coordinate transformation of the force at the knee from global to
shank coordinates
KF_x = [];
KF_z = [];

for i=1:1500;
   KF_x(i,:) = [cos(shankangle(i,:))*ftsx(i,:)]-
[sin(shankangle(i,:))*ftsz(i,:)];
end

for i=1:1500;
   KF_z(i,:) = -
[sin(shankangle(i,:))*ftsx(i,:)]+[cos(shankangle(i,:))*ftsz(i,:)];
end

%Calculate the moment at the knee
for i=1:1500;
       Mts(i,:)=Msf(i,:)[(sin(shankangle(i,:)))*
          (ffsx(i,:))*((0.567)*(0.4037))]-
          [(ffsz(i,:))*((0.567)*(0.4037))*cos(shankangle(i,:))]-
          [-(sin(shankangle(i,:)+pi))*ftsx(i,:)*((0.433)*(0.4037))] -
          [ftsz(i,:)*((0.433)*(0.4037))*cos(shankangle(i,:)+pi)]
          +Shank_MofI*Add(i,2);
end

%convert pedal angle and shank angle from radians to degrees
footangle = footangle*(57.29578);
shankangle = shankangle*(57.29578);
crankangle = crankangle*(57.29578);
pedalangle = pedalangle*(57.29578);
```

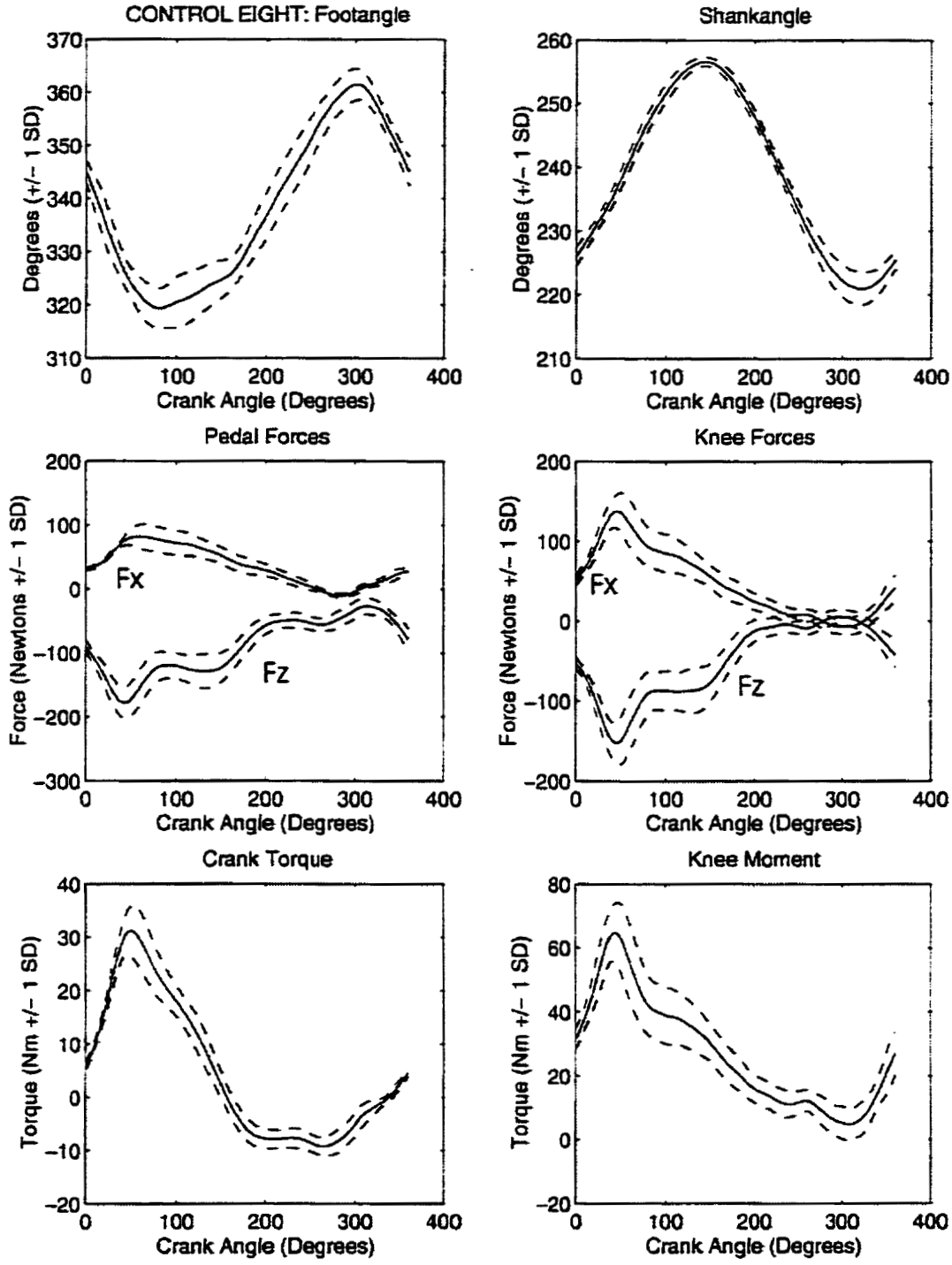
PART B: Individual control and subject data (complete kinetic files).

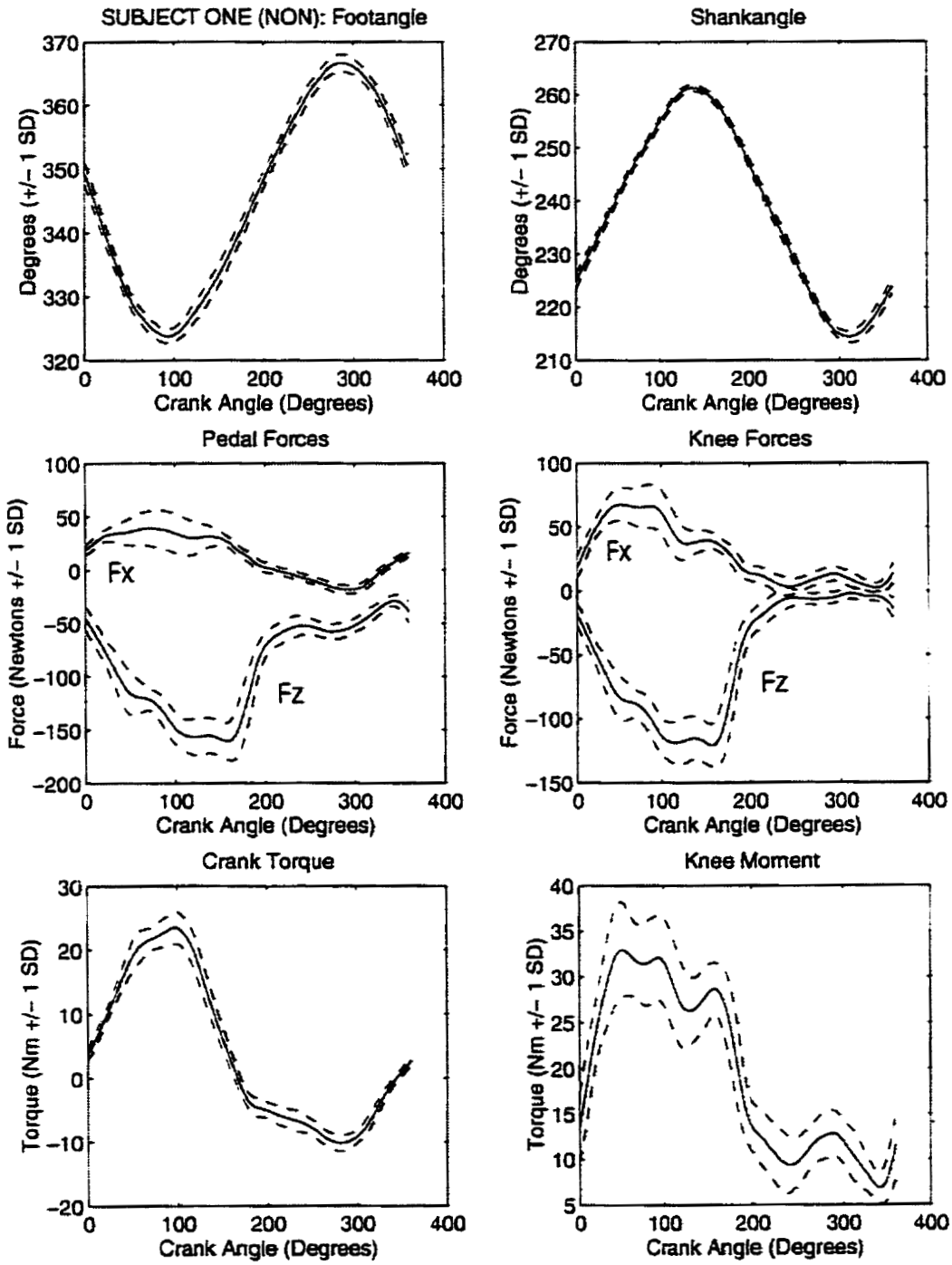
The following set of graphs includes the following information: the kinematic angles (footangle and shankangle) necessary for the knee moment calculation; the pedal forces (lab coordinate system) and knee forces (shank coordinate system); and the crank torque and knee moments.

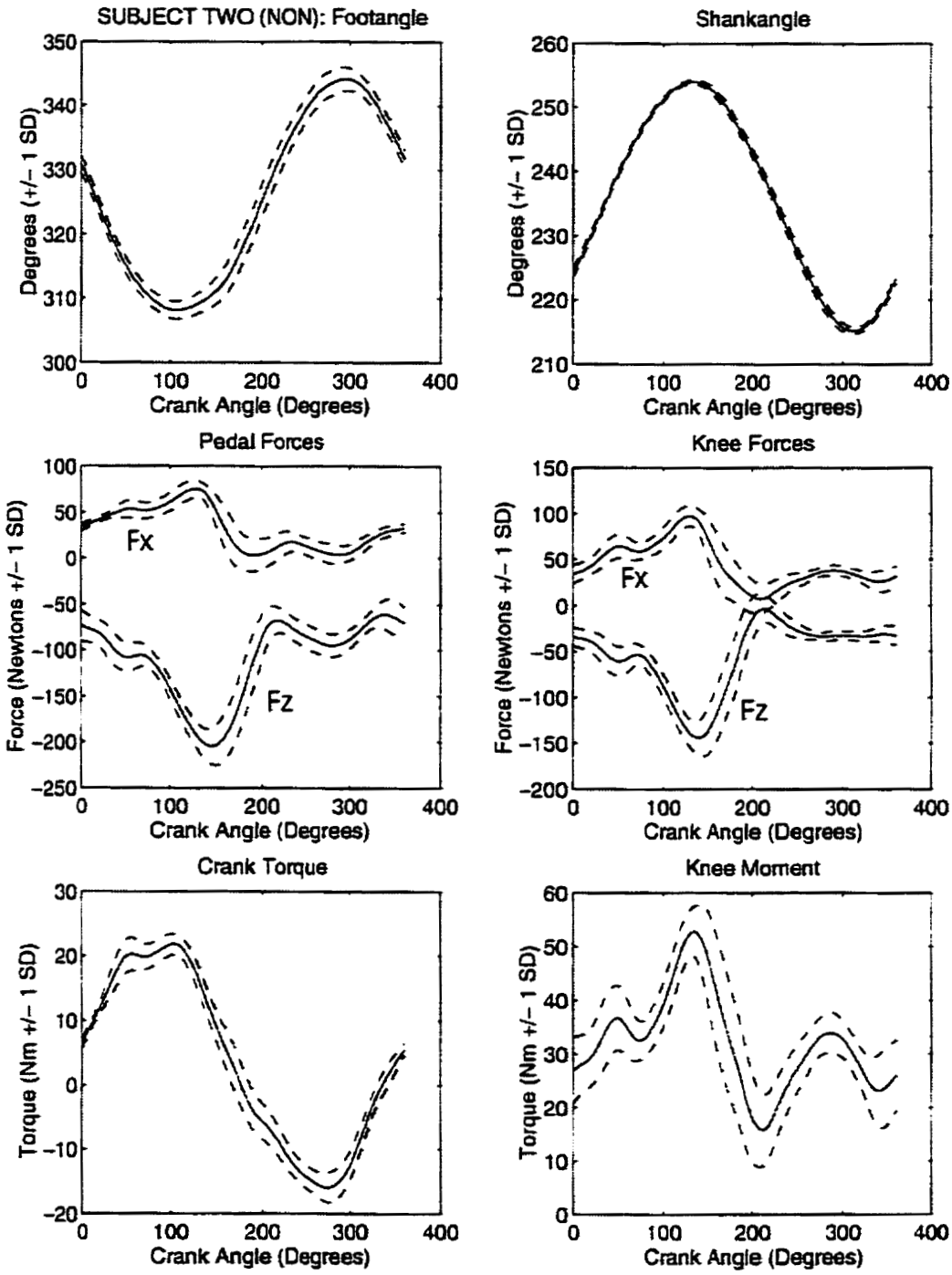


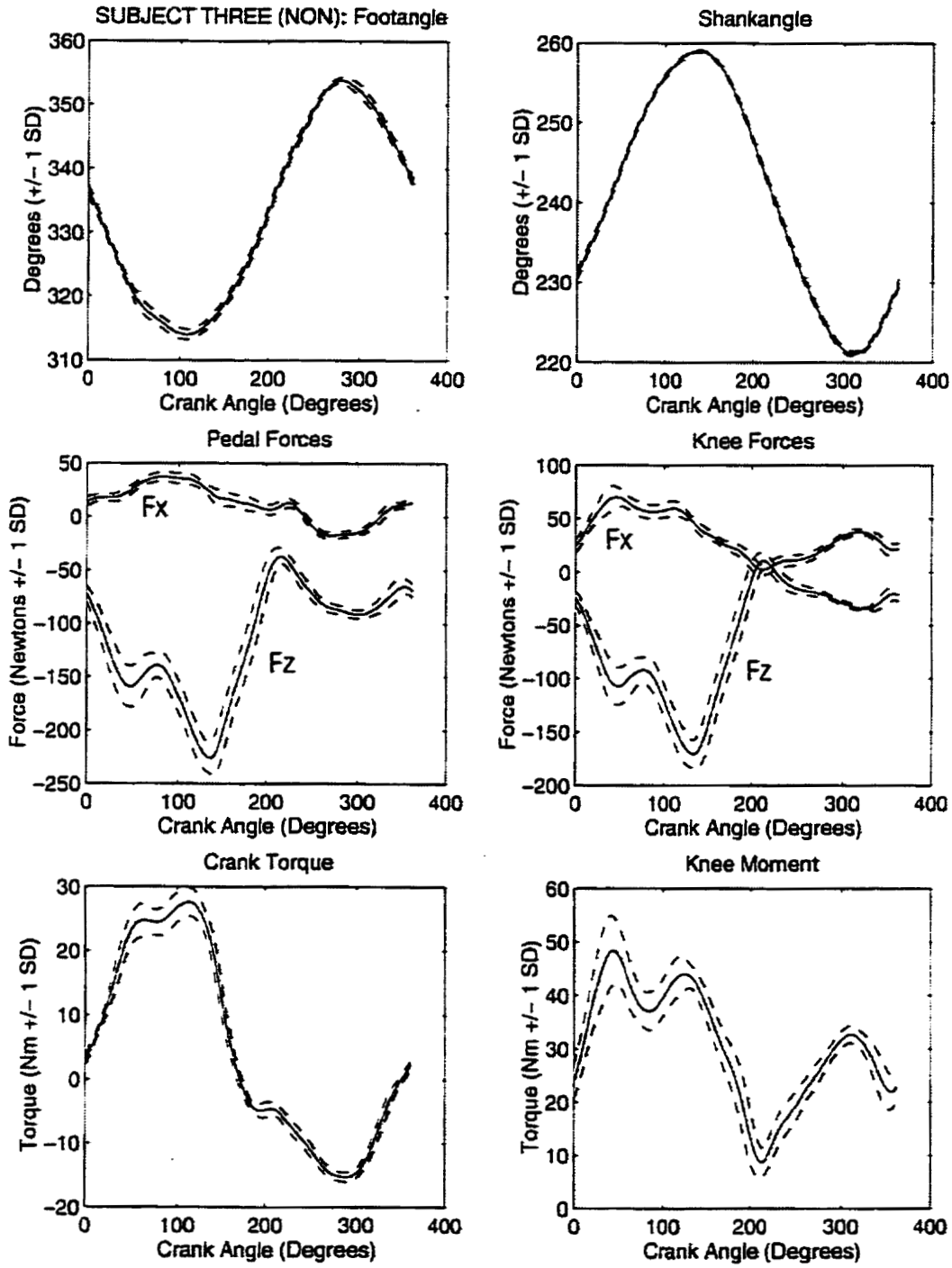

CONTROL FOUR: Footangle
Degrees +/- 1SD
370
360
350
340
330
320
0
100
200
300
400
Crank Angle (Degrees)
Shankangle
Degrees +/- 1SD
260
250
240
230
220
210
0
100
200
300
400
Crank Angle (Degrees)
Pedal Forces
Force (Newtons +/- 1SD)
50
0
-50
-100
-150
-200
Fx
Fz
0
100
200
300
400
Crank Angle (Degrees)
Knee Forces
Force (Newtons +/- 1SD)
100
50
0
-50
-100
-150
Fx
Fz
0
100
200
300
400
Crank Angle (Degrees)
Crank Torque
Torque (Nm +/- 1SD)
25
20
15
10
5
0
-5
-10
0
100
200
300
400
Crank Angle (Degrees)
Knee Moment
Torque (Nm +/- 1SD)
60
50
40
30
20
10
0
0
100
200
300
400
Crank Angle (Degrees)

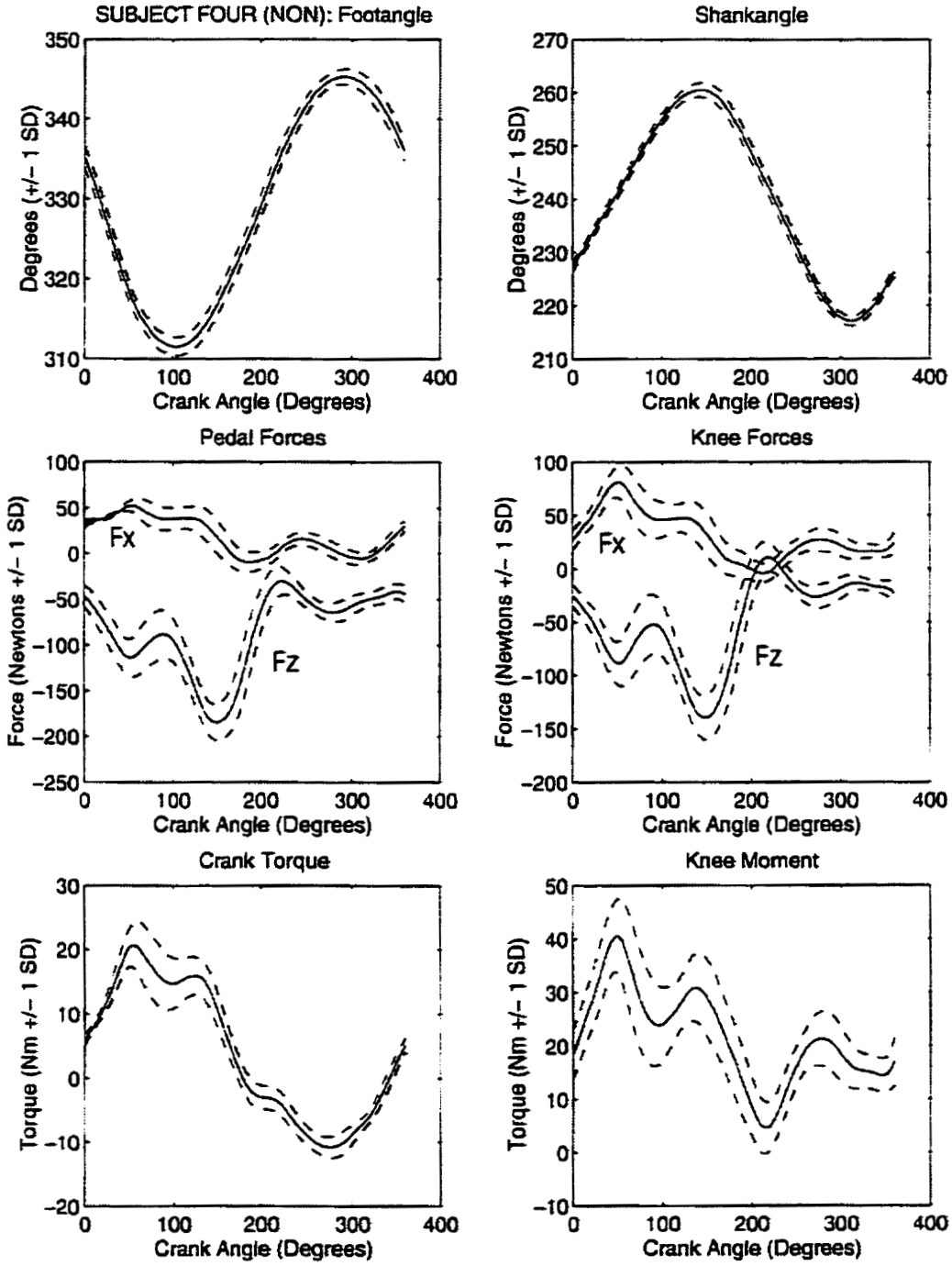

CONTROL SEVEN: Footangle
Degrees (+/- 1 SD)
Crank Angle (Degrees)
Shankangle
Degrees (+/- 1 SD)
Crank Angle (Degrees)
Pedal Forces
Fx
Fz
Force (Newtons +/- 1 SD)
Crank Angle (Degrees)
Knee Forces
Fx
Fz
Force (Newtons +/- 1 SD)
Crank Angle (Degrees)
Crank Torque
Torque (Nm +/- 1 SD)
Crank Angle (Degrees)
Knee Moment
Torque (Nm +/- 1 SD)
Crank Angle (Degrees)

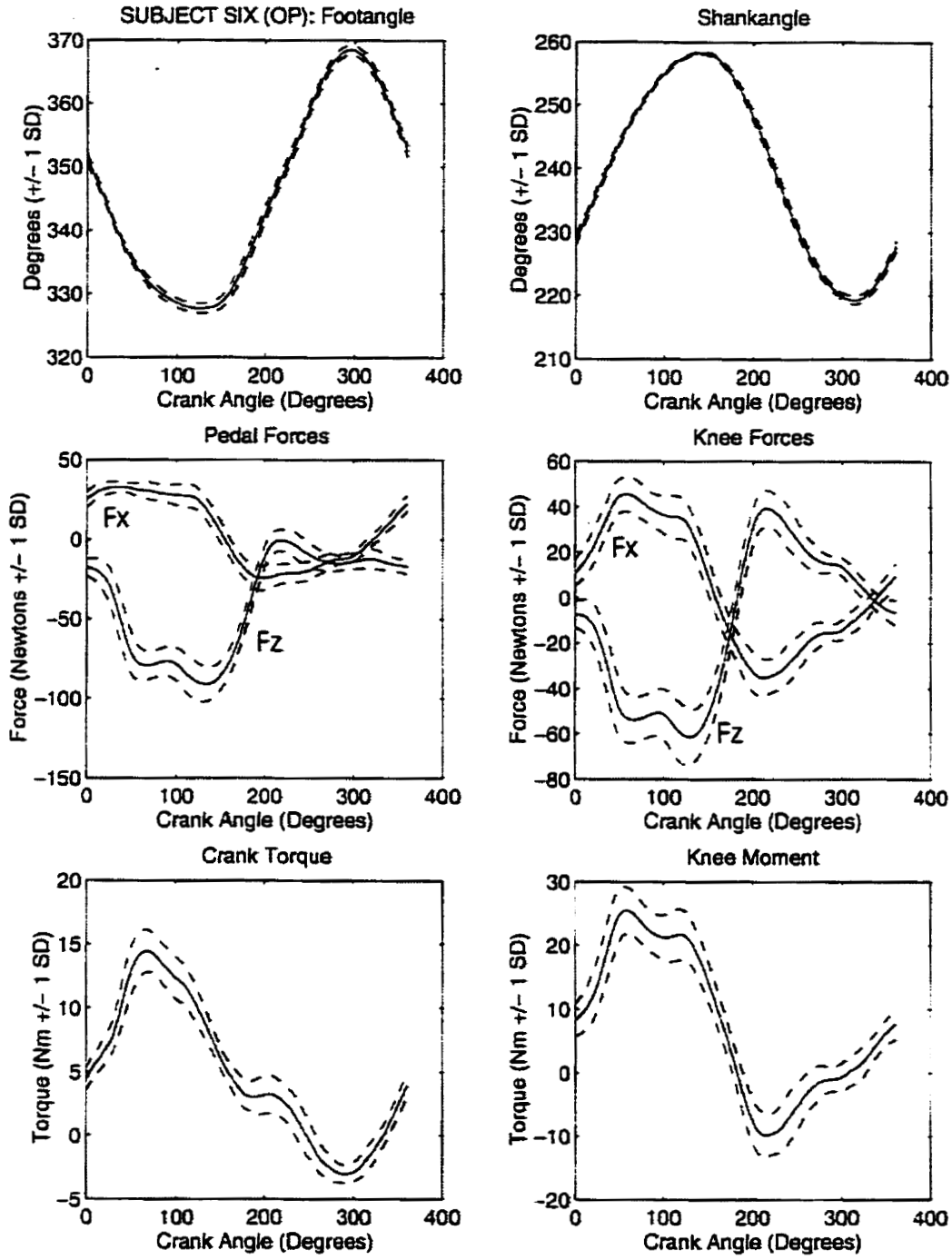

CONTROL EIGHT: Footangle
Degrees (+/- 1 SD)
Crank Angle (Degrees)
Shankangle
Degrees (+/- 1 SD)
Crank Angle (Degrees)
Pedal Forces
Force (Newtons +/- 1 SD)
Fx
Fz
Crank Angle (Degrees)
Knee Forces
Force (Newtons +/- 1 SD)
Fx
Fz
Crank Angle (Degrees)
Crank Torque
Torque (Nm +/- 1 SD)
Crank Angle (Degrees)
Knee Moment
Torque (Nm +/- 1 SD)
Crank Angle (Degrees)

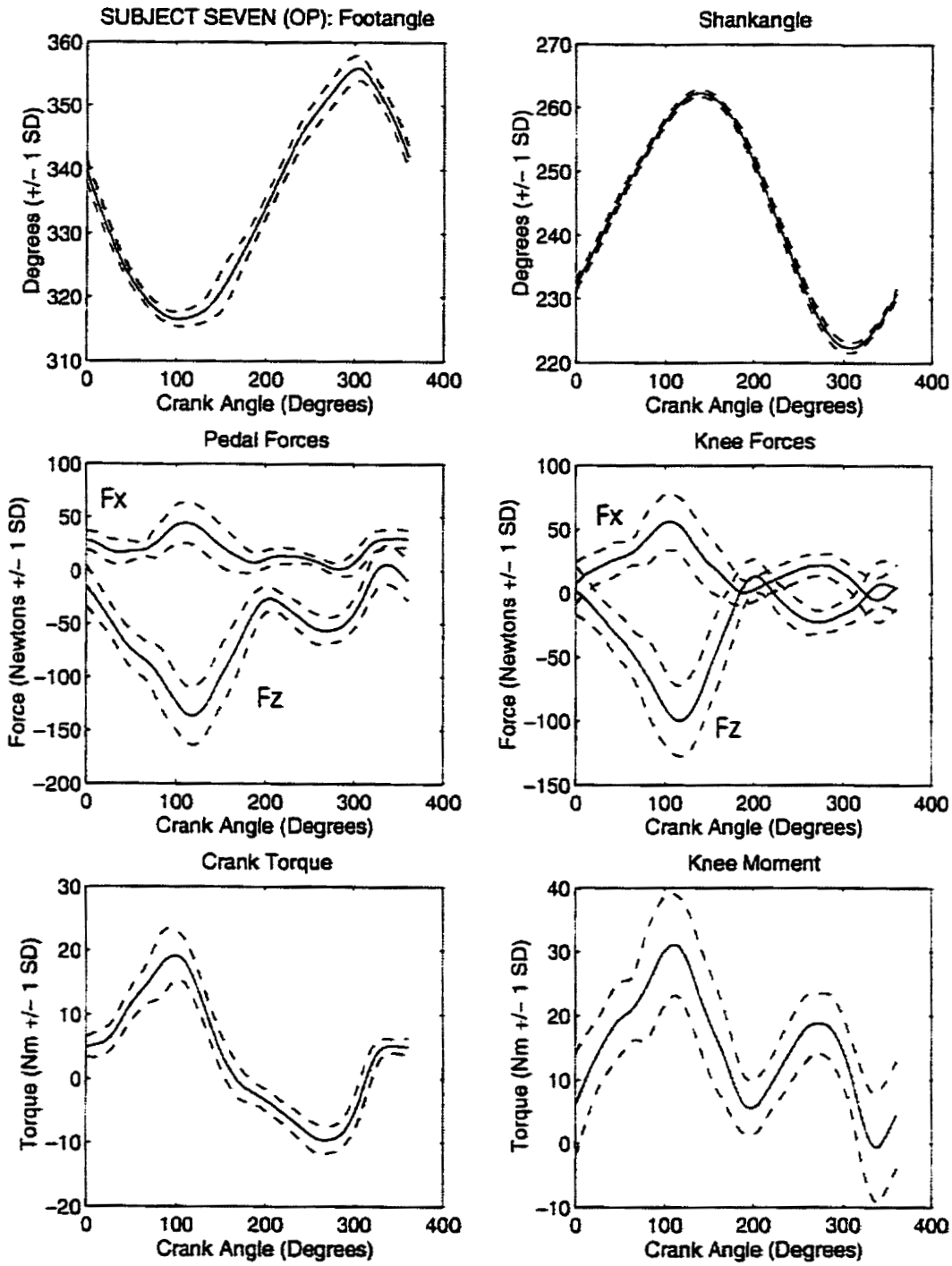

SUBJECT ONE (NON): Footangle
Degrees (+/- 1 SD)
Crank Angle (Degrees)
Shankangle
Degrees (+/- 1 SD)
Crank Angle (Degrees)
Pedal Forces
Force (Newtons +/- 1 SD)
Fx
Fz
Crank Angle (Degrees)
Knee Forces
Force (Newtons +/- 1 SD)
Fx
Fz
Crank Angle (Degrees)
Crank Torque
Torque (Nm +/- 1 SD)
Crank Angle (Degrees)
Knee Moment
Torque (Nm +/- 1 SD)
Crank Angle (Degrees)

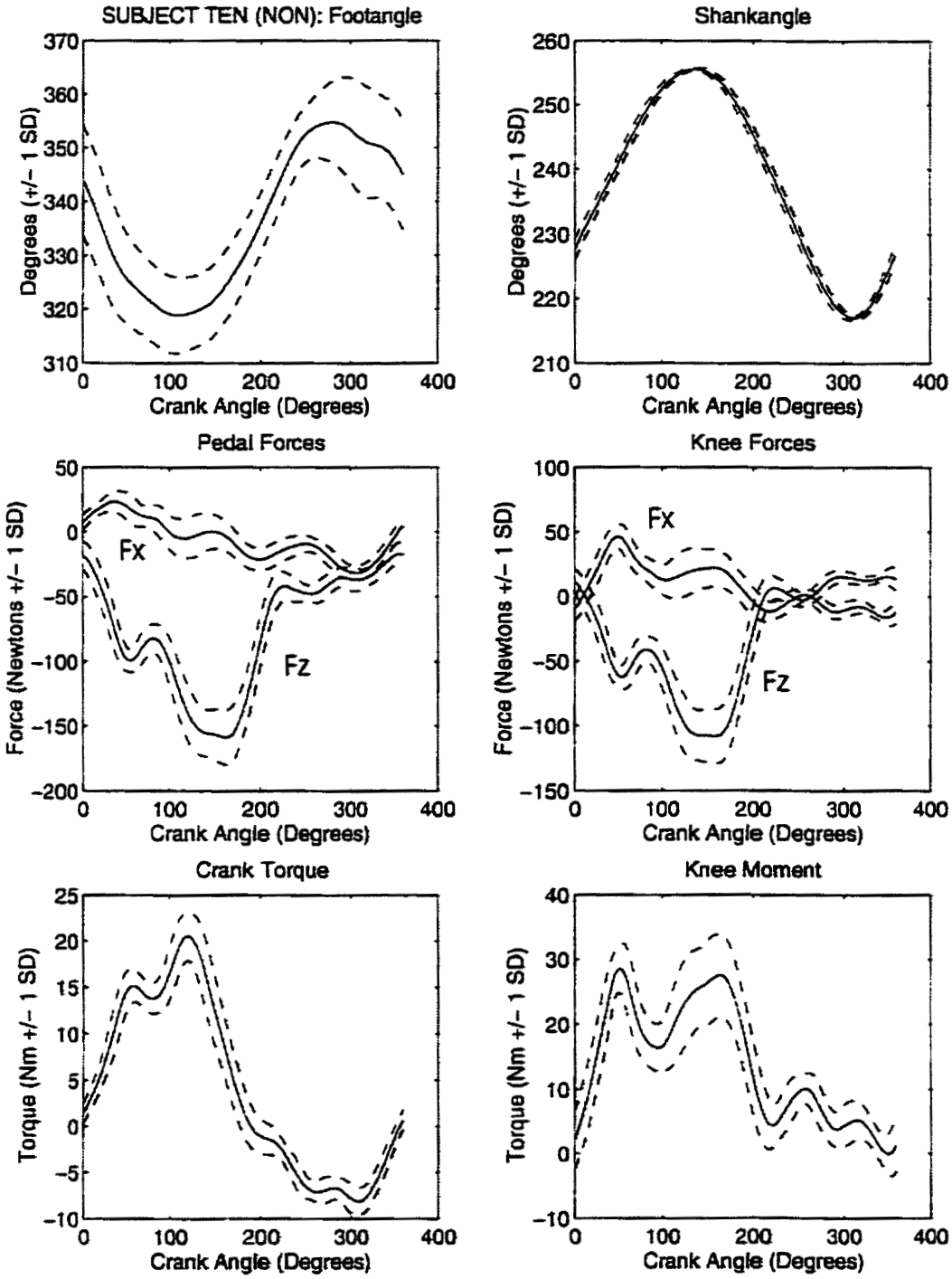

SUBJECT TWO (NON): Footangle
Degrees (+/- 1 SD)
Crank Angle (Degrees)
Shankangle
Degrees (+/- 1 SD)
Crank Angle (Degrees)
Pedal Forces
Force (Newtons +/- 1 SD)
Fx
Fz
Crank Angle (Degrees)
Knee Forces
Force (Newtons +/- 1 SD)
Fx
Fz
Crank Angle (Degrees)
Crank Torque
Torque (Nm +/- 1 SD)
Crank Angle (Degrees)
Knee Moment
Torque (Nm +/- 1 SD)
Crank Angle (Degrees)

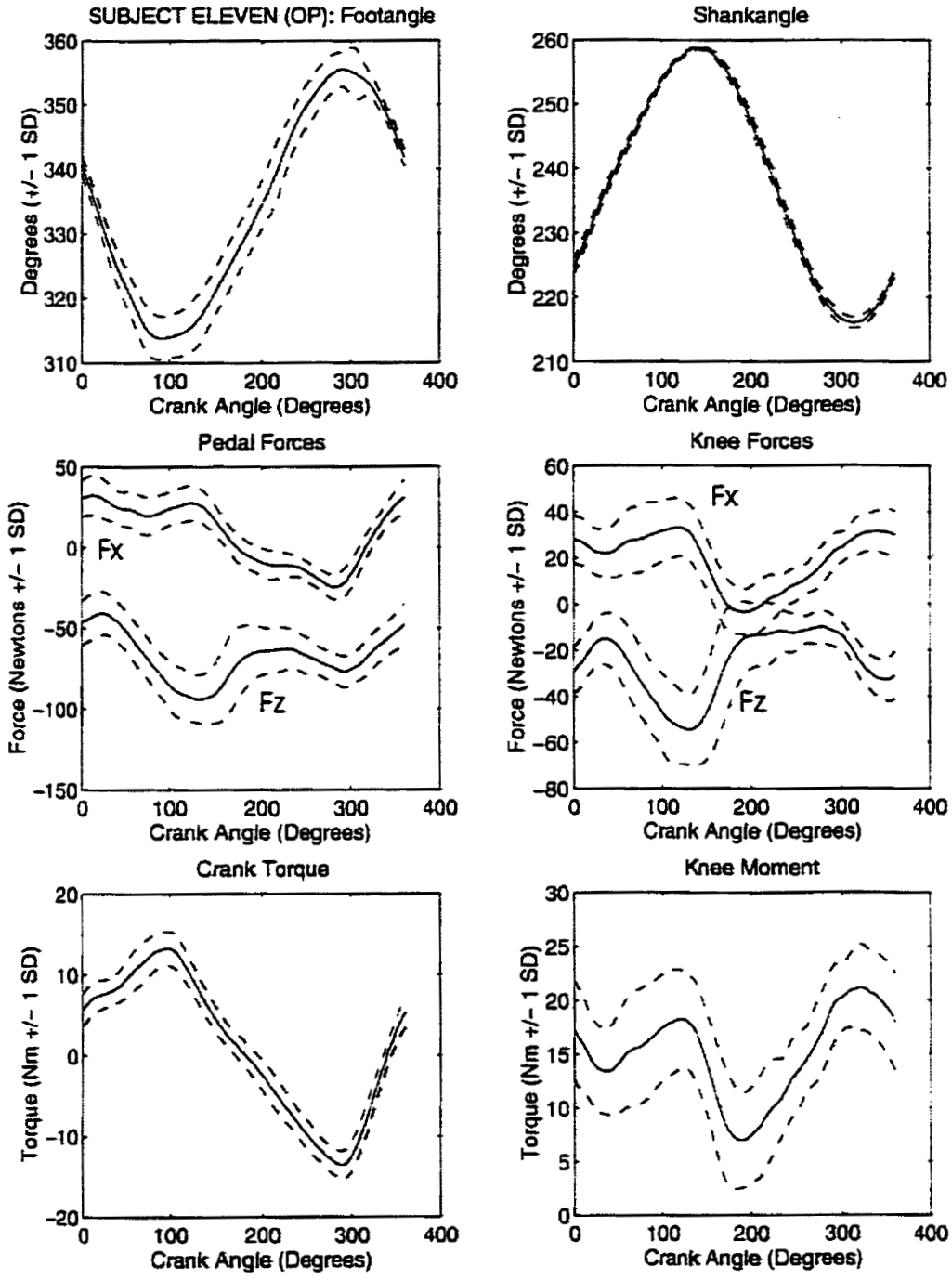

SUBJECT THREE (NON): Footangle
Degrees (+/- 1 SD)
Crank Angle (Degrees)
Shankangle
Degrees (+/- 1 SD)
Crank Angle (Degrees)
Pedal Forces
Fx
Fz
Force (Newtons +/- 1 SD)
Crank Angle (Degrees)
Knee Forces
Fx
Fz
Force (Newtons +/- 1 SD)
Crank Angle (Degrees)
Crank Torque
Torque (Nm +/- 1 SD)
Crank Angle (Degrees)
Knee Moment
Torque (Nm +/- 1 SD)
Crank Angle (Degrees)


SUBJECT FOUR (NON): Footangle
Degrees (+/- 1 SD)
Crank Angle (Degrees)
Shankangle
Degrees (+/- 1 SD)
Crank Angle (Degrees)
Pedal Forces
Force (Newtons +/- 1 SD)
Fx
Fz
Crank Angle (Degrees)
Knee Forces
Force (Newtons +/- 1 SD)
Fx
Fz
Crank Angle (Degrees)
Crank Torque
Torque (Nm +/- 1 SD)
Crank Angle (Degrees)
Knee Moment
Torque (Nm +/- 1 SD)
Crank Angle (Degrees)


SUBJECT SIX (OP): Footangle
Degrees (+/- 1 SD)
Crank Angle (Degrees)
Shankangle
Degrees (+/- 1 SD)
Crank Angle (Degrees)
Pedal Forces
Force (Newtons +/- 1 SD)
Fx
Fz
Crank Angle (Degrees)
Knee Forces
Force (Newtons +/- 1 SD)
Fx
Fz
Crank Angle (Degrees)
Crank Torque
Torque (Nm +/- 1 SD)
Crank Angle (Degrees)
Knee Moment
Torque (Nm +/- 1 SD)
Crank Angle (Degrees)


SUBJECT SEVEN (OP): Footangle
Degrees (+/- 1 SD)
Crank Angle (Degrees)
Shankangle
Degrees (+/- 1 SD)
Crank Angle (Degrees)
Pedal Forces
Fx
Fz
Force (Newtons +/- 1 SD)
Crank Angle (Degrees)
Knee Forces
Fx
Fz
Force (Newtons +/- 1 SD)
Crank Angle (Degrees)
Crank Torque
Torque (Nm +/- 1 SD)
Crank Angle (Degrees)
Knee Moment
Torque (Nm +/- 1 SD)
Crank Angle (Degrees)


SUBJECT TEN (NON): Footangle
Degrees (+/- 1 SD)
Crank Angle (Degrees)
Shankangle
Degrees (+/- 1 SD)
Crank Angle (Degrees)
Pedal Forces
Force (Newtons +/- 1 SD)
Fx
Fz
Crank Angle (Degrees)
Knee Forces
Force (Newtons +/- 1 SD)
Fx
Fz
Crank Angle (Degrees)
Crank Torque
Torque (Nm +/- 1 SD)
Crank Angle (Degrees)
Knee Moment
Torque (Nm +/- 1 SD)
Crank Angle (Degrees)


SUBJECT ELEVEN (OP): Footangle
Degrees (+/- 1 SD)
Crank Angle (Degrees)
Shankangle
Degrees (+/- 1 SD)
Crank Angle (Degrees)
Pedal Forces
Force (Newtons +/- 1 SD)
Fx
Fz
Crank Angle (Degrees)
Knee Forces
Force (Newtons +/- 1 SD)
Fx
Fz
Crank Angle (Degrees)
Crank Torque
Torque (Nm +/- 1 SD)
Crank Angle (Degrees)
Knee Moment
Torque (Nm +/- 1 SD)
Crank Angle (Degrees)