THE RELATIONSHIP BETWEEN MUSCULAR CO-CONTRACTION AND DYNAMIC KNEE STIFFNESS IN ACL-DEFICIENT NON-COPERS

by

Emily S. Gardinier

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Emily S. Gardinier

Approved:_____
Lynn Snyder-Mackler, Sc.D.
Professor in charge of the thesis on behalf of the Advisory Committee

Approved:_____
Todd D. Royer, Ph.D.
Committee member from the Department of Health, Nutrition and Exercise Science

Approved:_____
James L. Glancey, Ph.D.
Committee member from the Board of Senior Thesis Readers

Approved:_____
Alan Fox, PhD.
Director, University Honors Program

DEDICATION

To my husband, Joe
in deep appreciation of your encouragement and support.

To my family,
in gratefulness for your confidence in me.

To my best friends,
Grace and Shelly
I love you like sisters.

Whatever your hand finds to do,
do it with all your might.
For in the grave where you are going,
there is neither working nor planning nor wisdom.

The race is not to the swift
or the battle to the strong,
nor does food come to the wise
or wealth to the brilliant
or favor to the learned;
but time and chance happen to them all.

Ecclesiastes 9:10-11

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ABSTRACT

Anterior cruciate ligament-deficient (ACL-D) individuals who fail to dynamically stabilize their knee (termed non-copers) are reported to adopt a neuromuscular strategy characterized by reduced knee flexion excursions, reduced external knee flexion moments and generalized co-contraction during gait. This unsuccessful neuromuscular strategy has been qualitatively described as the "knee-stiffening strategy," although dynamic knee stiffness has not been measured in the non-coper population. While the generally ascribed function of muscular co-contraction is to increase joint stiffness and stability, its relationship to dynamic knee stiffness during gait has not been evaluated. Establishment of a reliable relationship between these two measures would render stiffness a simpler means by which to infer co-contraction among individuals with ACL-D knees.

The purpose of this study was to determine (1) whether a strategy of generalized, increased co-contraction was present in the ACL-deficient limb, (2) whether ACL-D non-copers walk with a quantifiably stiffer involved knee and (3) whether dynamic knee stiffness correlates with muscular co-contraction in this dynamically unstable population. Kinematic and kinetic data were collected from 42 ACL-D non-copers (male, N=31; female, N=11; age, 27.8±10.1 yrs) during the weight acceptance phase of gait for

calculation of dynamic joint stiffness. Electromyography for 6 lower extremity muscles (M/L vastus, M/L hamstring, M/L gastrocnemius) were also collected for determination of co-contraction indices. No significant differences in co-contraction indices and dynamic knee stiffness were found between the involved and uninvolved limbs in this sample. Furthermore, dynamic knee stiffness did not correlate with co-contraction during gait. The lack of elevated co-contraction in the ACL-D limb is contrary to recent reports in the literature. However, the lack of differences in dynamic stiffness between limbs suggests that during gait, absence of the ACL does not result in a quantifiably discernible "knee-stiffening strategy." Because stiffness values did not correlate with co-contraction, we conclude that dynamic stiffness cannot be used as a surrogate for co-contraction during gait.

Chapter 1

INTRODUCTION

1.1 Neuromuscular Adaptations to ACL-Injury

Nearly half of all reported knee pathologies result from internal trauma, the most frequent of which is injury to the anterior cruciate ligament (ACL) [1, 2]. The principal function of the ACL is to prevent anterior translation of the tibia relative to the femur; thus ACL injury often leads to knee joint instability which inhibits individuals from participating in sports and daily activities.

Individuals with ACL rupture must achieve functional knee stability during daily activities despite their compromised mechanical restraints, and any significant variation in their gait from that of healthy individuals implies the presence of a compensation strategy. Several studies have examined patterns of lower-extremity kinematics, kinetics and muscle activation in ACL-deficient (ACL-D) individuals whose compensation strategies are unsuccessful in stabilizing their knee during dynamic activity (termed non-copers [3]). These individuals exhibit reduced knee flexion excursions [4-7]; reduced external knee flexion moments [4-8] and greater muscular co-contraction in the injured knee during walking and jogging [5, 6]. Often, this unsuccessful neuromuscular strategy adopted by non-copers is qualitatively labeled, “the knee-stiffening strategy.”

The strategies exhibited by ACL-injured patients are of particular interest because altered muscle activation is suggestive of abnormal distribution of contact forces at the articular surfaces, which does not bode well for long-term joint integrity and cartilage health [9, 10]. The increased magnitudes of generalized co-contraction observed in the non-coper gait suggest the presence of an adaptive strategy, which may contribute to the progression of knee osteoarthritis [11]. Not surprisingly, the relationship between ACL-rupture and the early onset of knee osteoarthritis has been well-established [11-13]. At least half of individuals sustaining ACL-rupture display radiographic signs of knee osteoarthritis 12 to 14 years after injury [11-13]. Further investigation of modified movement patterns assumed by non-copers may illuminate compensation-related mechanisms involved in progression of knee osteoarthritis in this population.

1.2 Co-contraction

Co-contraction is defined as simultaneous activation of antagonistic muscles crossing a joint. Its generally held purpose is to augment the ligament function in maintenance of joint stability, provide resistance to rotation at a joint, and equalize the pressure distribution at the articular surface [14, 15].

According to the classification scheme developed by Fitzgerald et al. [16] and used by the University of Delaware Physical Therapy Clinic, non-copers can be identified because of experiencing episodes of "giving-way," shifting or buckling on their injured knee. Episodes of giving-way are accompanied by the shear forces at the articular surface and are often interpreted by the patient as a sensation of instability [17]. A

neuromuscular strategy involving co-contraction of knee musculature during dynamic activity is identified as an attempt to stabilize the joint and to reduce shear, translational forces at the knee, both of which are detrimental to articular cartilage health [18, 19] and are un-restrained due to ACL deficiency.

Some have identified muscular co-contraction as a positive adaptation to trauma-induced instability, and indeed, from a mechanical standpoint, co-contraction can function as a joint-protective mechanism [15]. The development of torque by a single, unopposed agonist muscle group about a joint creates an uneven pressure distribution over the articular surfaces. Co-activation of an antagonist muscle group would therefore aid in the distribution of forces at the articular surface and simultaneously increase joint stability by increasing the degree of bone-to-bone fit of the articular ends.

Yet, co-contraction is widely viewed a potentially harmful strategy for long-term joint integrity [17, 20-23]. Despite its theorized ability to equalize joint pressure distribution, its overwhelming effect is an increase in the net compressive contact force at the articular surface. The activation of antagonist muscles crossing a joint has been shown to increase joint compressive forces [21], and have been linked to the progression of osteoarthritis [17, 21]. Further investigation of co-contraction strategies may elucidate the process by which this adaptive neuromuscular strategy contributes to the onset and progression of osteoarthritis in the ACL-injured population.

1.3 Dynamic Knee Joint Stiffness

In relation to the human body, the evaluation of the stiffness of biological tissues including muscle fibers, ligaments, bone and cartilage has enabled biomechanists to functional, behavioral models of biological subsystems. The use of "stiffness" measurements has been subsequently applied to quasi-elastic bodies ranging in complexity from an individual osteoblast to the entire human body.

The conception of stiffness is rooted in traditional physics, and is meant to characterize the elastic properties of a deformable body under the influence of an external force. An external force (F) is applied to a linear spring which deforms an observed distance (Δx). The proportionality constant, or stiffness (k), of the spring illustrates the relationship between the applied force and the resulting displacement according to Hooke's Law (Equation 1.1), and is indicated by the slope of the applied force plotted as a function of spring deformation (Figure 1.1a). Rotational springs can be viewed in a similar manner, with the rotational stiffness (k_r) designated as the slope the graph of the applied torque (T) plotted as a function of angular deformation ($\Delta\theta$) (Figure 1.1b).

Equation 1.1 Hooke's Law $F = k(\Delta x)$

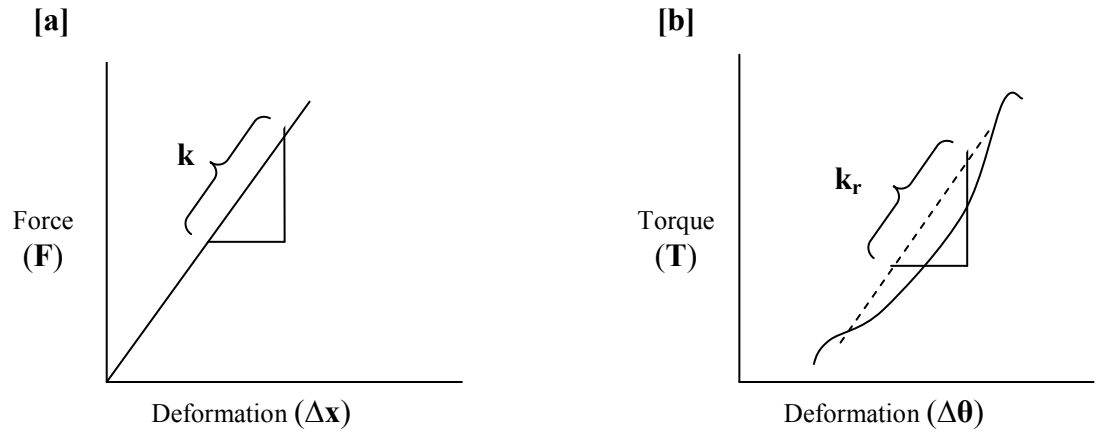

Figure 1.1 Hooke's Law applied to (a) linear stiffness and (b) torsional stiffness.

In concept, the stiffness of a human joint is determined by the collective physical properties of all active and passive structures that traverse it [24-27]. Most biomechanists agree that the quantification of *true* joint stiffness demands considerations for each of the individual components contributing to stiffness, including tendons, ligaments, muscles, cartilage and bone[24]. Additionally, provisions for viscosity, reflexive properties of muscles and central nervous system control need to be made. Therefore, rendering a model of *true* joint stiffness is quite complicated – and in most situations, wholly impractical.

Consequently, several distinct calculations of stiffness have been developed for use in sport and clinical biomechanics: vertical stiffness, leg stiffness, passive joint stiffness and dynamic joint stiffness. Leg stiffness is simply a modified measure of vertical stiffness developed by McMahon and Cheng [28]. Vertical and leg stiffness have been used largely for applications in which the leg can be modeled as a linear spring, such as in running and hopping [25, 28-33]. Alternatively, in the analysis of passive joint stiffness, a joint is modeled as a torsional spring; this measure is concerned with the elastic responses of passive soft tissues which cross a joint in response to an applied load. Passive joint stiffness must be measured in absence of active muscle contraction, which is often confirmed with inspection of electromyography (EMG) [24, 25]. Subsequently, when the contributions of active musculature to joint stability are of interest, the measure of dynamic joint stiffness is used. Dynamic joint stiffness is defined as the observed resistance that a joint (i.e. the active muscles and other passive soft tissue structures that cross the joint) offers during gait in response to an applied moment [26]. With the addition of active musculature to the joint tissues of interest, muscle activity becomes the dominant effector of joint movement. Thus, when studying the contribution of local muscle activity to motion at a single joint, dynamic joint stiffness proves the most appropriate measure of stiffness.

Some level of stiffness is required for neuromuscular control during daily activities; however too much or too little stiffness may lead to musculoskeletal injury [25]. Furthermore, increased stiffness is often accompanied by decreased joint excursions and increased peak joint reaction forces; these features typically lead to

increased loading rates and may subject the knee to abnormal joint compressive forces [21]. High levels of stiffness have been associated with bony injuries [32, 34], and conversely low levels of stiffness are suggested to be related to elevated incidence of soft tissue injuries due the allowance of excessive joint motion [32, 35].

Although muscle co-activity is clearly a major component of dynamic joint stiffness, the relationship between the two variables is not clear. Dynamic stiffness of the knee joint has been quantified during stepping down [36] and performance of a counter-movement jump [37] in elderly populations for comparison to young populations, as well as during walking in individuals with unilateral total knee arthroplasty for an assessment of gait symmetry [38]. To our knowledge, no studies have investigated dynamic knee joint stiffness in an ACL-D population during gait as a measure of limb symmetry and assessed its relationship to muscular co-contraction levels.

1.4 Clinical Relevance

Kinematic and kinetic motion analysis is widely used to obtain knee excursion and moment data; however EMG equipment necessary for the acquisition of muscular co-contraction levels is not globally available. There are several known factors which may make EMG undesirable for use, including: reliability of electrode placement, susceptibility to cross-talk and inherent signal noise due to motion artifact and ambient noise [39]. Overall, the process in which to collect EMG is tedious, labor-intensive, time-consuming and can result in inhibition of the natural movement patterns of subjects.

It is clinically relevant to establish whether a relationship exists between muscular co-contraction indices and dynamic knee stiffness in order to determine whether kinematic and kinetic motion analysis data could be used as a surrogate for EMG in assessing muscular co-contraction. Dynamic joint stiffness is determined using kinematic and kinetic data only and would provide a simpler means to infer co-contraction and, subsequently, abnormal joint compressive forces among individuals with ACL-deficient knees. The overall goal of this research is to determine whether dynamic knee joint stiffness, a biomechanically-derived variable, can act as a surrogate for generalized muscular co-contraction in inferring abnormal joint forces during gait in an ACL-deficient population.

1.5 Objectives and Hypothesis

This study had three objectives. The first was to compare co-contraction indices of four separate muscle pairs between the injured and non-injured limb in order to determine whether a strategy of generalized, increased co-contraction was present in the ACL-deficient limb. We hypothesized that co-contraction would be significantly higher for the involved limb, as was reported by Hurd [5].

The second objective was to compare dynamic stiffness values between the injured and non-injured limb in order to quantitatively determine whether a stiffened knee gait was being adopted by this sample of non-copers. We hypothesized that stiffness would be significantly higher for the involved knee.

The final objective was to determine whether a positive relationship exists between dynamic stiffness and muscular co-contraction for this dynamically unstable population. We hypothesized that co-contraction and stiffness would display a positively correlated relationship.

Chapter 2

METHODS

2.1 Subjects

The sample included 42 subjects (male, *N*=31; female, *N*=11; mean age=27.8 yrs) with acute ACL injury who had been classified as non-copers using the University of Delaware screening examination [16]. Patients underwent screening upon gaining full knee range of motion, minimal knee effusion, ≥70% quadriceps strength symmetry and the ability to hop on the injured limb without pain. Subjects were classified as non-copers if they met any one of the following criteria: four unilateral hop tests (timed hop <80%) [40], Knee Outcome Survey-Activities of Daily Living Scale (KOS-ADLS) (<80%), global rating of knee function (<60%), and the number of giving way episodes during daily activities since injury (>1) [16]. This screening examination and classification scheme has been established as an effective means of differentiation between individuals with ACL injury who are able to achieve functional knee stability during dynamic activities (copers) and those who cannot (non-copers) [16].

Non-copers who met the following inclusion criteria were included in the study: regular pre-injury participation in level I or II activities [41, 42], acute ACL rupture

within 7 months of their initial evaluation, and 13 to 55 years old. Each individual's healthy knee served as the control, for comparison with their ACL-deficient knee.

Exclusion criteria for the study included bilateral knee involvement and any lower extremity or low back injuries that prevented the completion of the screening examination. Individuals were also excluded if they had concomitant or symptomatic grade III injury to other knee ligaments, a repairable meniscus tear, or full-thickness articular cartilage defect greater than 1 cm^2. ACL ruptures were confirmed with magnetic resonance imaging scans, and through clinical examination with a KT 1000 arthrometer side-to-side measurement difference of >3mm.

The rights of human subjects were assured. This research work was approved by the University of Delaware Institutional Human Subjects Review Board. All individuals provided informed consent prior to testing.

2.2 Instrumentation

Kinematic data were collected using an eight camera, three-dimensional motion analysis system (VICON, Oxford Metrics Ltd., London, UK) with a sampling rate of 120 Hz. Residual errors were held below 0.50mm; marker data were low-pass filtered with a bidirectional 2^{nd} order Butterworth filter with a cutoff frequency of 6 Hz. Retro-reflective markers were secured with a double-sided marker fixing tape (VICON, Oxford Metrics Ltd., London, UK). Anatomical markers which were utilized solely during the standing calibration trial for identification of joint centers were placed bilaterally on the iliac crests, greater trochanters, medial and lateral femoral epicondyles, medial and lateral

malleoli, and the heads of the first and fifth metatarsals. For tracking lower extremity motion during dynamic trials, clusters comprised of four markers attached to rigid thermoplast were fixed to the postero-lateral thighs and shanks bilaterally; a three-marker cluster for tracking of the pelvis was placed on the sacrum. Walking speed was captured using two photoelectric beams placed 2.86 m apart, spanning the kinematic collection volume.

Kinetic data were collected using a six-component force plate (Bertec Corporation, Worthington, OH) embedded within the walkway. Ground reaction force data were sampled at 1,080 Hz and filtered with a 2^{nd} order bidirectional, phase corrected Butterworth filter using a low-pass frequency of 50 Hz. Data were collected unilaterally as subjects walked over the force plate at their self-selected walking speed, from heel strike to toe-off.

EMG data were collected with the use of two MA-300 EMG Systems (Motion Lab Systems, Baton Rouge, LA) with a sampling rate of 1080 Hz. Data was band pass filtered from 20-350 Hz. Electromyographic data were recorded from the vastus medialis, vastus lateralis, medial hamstring, lateral hamstring, medial gastrocnemius, and lateral gastrocnemius muscles of both limbs during gait. Kinematic, kinetic and electromyographic data were synchronized so that muscle activity could be analyzed with respect to biomechanically derived gait events.

2.3 Procedure

Anthropometric measurement of pelvic depth (from posterior superior iliac spine to umbilicus) was obtained for three-dimensional model construction purposes. Additionally, the subject's height was recorded for normalization purposes.

Electrodes were affixed to the 18 lower-extremity muscles and a ground electrode was placed over bilateral acromion processes. Skin preparation included shaving, light abrasion and cleaning with isopropyl alcohol prior to electrode placement. Dual, active surface electrodes made of surgical-grade stainless steel were placed in line with the muscle fibers on the prepared skin over the belly of each muscle and held in place with adhesive bandage (Cover-roll stretch, BSN-JOBST, Inc., Rutherford College, NC). Electrodes were additionally secured with elastic over-wrap (Superwrap, Fabrifoam Products, Exton, PA, USA).

Subjects performed isolated maximum voluntary isometric contractions (MVIC) in order to validate electrode placements and to provide a physiological reference for comparing the EMG amplitudes between muscle groups and among subjects. Verbal encouragement was provided to elicit maximum activation. The gastrocnemius was tested during standing with the subject's hands gripped underneath the countertop for self-resistance as they plantarflexed. Adjustable, padded cuffs secured to the supports of a padded table were used to restrain the subject's limbs for the remaining MVIC tests in the following positions (performed in the order listed): subject in a seated position with knee secured at the ankle in 60° flexion for testing quadriceps; subject in a long sitting position with the knees extended and ankles secured in a fully plantarflexed position for

testing tibialis anterior; subject lying prone with the ankles secured so that the knee is in 30° flexion for testing hamstrings; and subject in quadruped, with feet secured in full dorsiflexion and with hips and knees flexed > 90 degrees for testing soleus. Maximal contractions were held for two seconds, followed by two seconds of recorded resting activity for each test. During each muscle test, the channel gain was adjusted for each electrode so that the signal was just short of saturating the ± 5,000 bit output graph window, with no signal exceeding the ±8,000 bit maximum range. For normalization purposes, a separate, four-second resting trial was collected with the subject fully relaxed, lying prone.

A standing calibration was performed, capturing the subject's weight and knee angles for normalization purposes. Walking trials were then performed along a 13-meter walkway. Practice trials were used to obtain an average walking speed and to allow the subject to become comfortable walking through the collection volume without targeting the force plate in the walkway. Data were collected following the practice trials for the right leg, followed by the left leg. A usable trial was determined to be one in which there was no visible alteration in stride length, walking speed was within a ±5% range of the mean speed determined during practice trials, and the subject's foot landed entirely on the force plate. Trials in which both of the subject's feet struck the force plate were rejected. Data were collected unilaterally for the stance phase of gait, and a minimum of five usable right- and five left-foot strike trials were required of each subject.

2.4 Calculation of Variables of Interest

Electromyographic data reduction was accomplished with the use of a custom-built LabVIEW program (LabVIEW 8.0.1, National Instruments, Austin, TX). The EMG data were first filtered with a 350Hz lowpass Butterworth filter, then full-wave rectified and filtered again with a phase corrected 8^{th} order, 20Hz lowpass Butterworth filter to generate a linear envelope. Linear envelopes were normalized to the average activation during a 50ms window about the point of peak activation for each muscle. Peak activation was acquired from MVIC tests, except in cases where a greater activation level was found in one of the walking trials. During dynamic activities it is common to see levels of EMG activity that exceed levels measured during MVIC. Therefore, all walking trials were examined for maximum activations which exceeded that of MVIC. Linear envelopes were normalized to the peak activation level found among isometric tests and walking trials.

Co-contraction indices, which we defined as the simultaneous activation of antagonist muscles crossing a joint, were calculated for the following muscle pairs: vastus medialis-medial hamstrings (VMMH), vastus medialis-medial gastrocnemius (VMMG), vastus medialis-lateral gastrocnemius (VMLG), vastus lateralis-lateral hamstrings (VLLH), vastus lateralis-lateral gastrocnemius (VLLG), and vastus lateralis-medial gastrocnemius (VLMG). Muscle activity was analyzed from 100ms prior to initial contact (accounting for electromechanical delay [43]) to the point when toe-off occurred; this time interval was normalized to 100 data points. Co-contraction indices for each muscle pair were calculated according to Equation 2.1:

Equation 2.1 Calculation of co-contraction index according to Rudolph et al. [6]

$$\text{CC index} = \sum_{i=1}^{100} \left[\left(\frac{\text{Lower EMG}i}{\text{Higher EMG}i} \right) \times \left(\text{Lower EMG}i + \text{Higher EMG}i \right) \right] / 100$$

The terms "lower EMG*i*" and "higher EMG*i*" represent the activation of the less- and more-active muscles at *i* of 100 time points. The quotient of these two terms is arranged in order to avoid division by zero errors. This quotient of relative activity is multiplied by the sum of activity in both muscle groups for time point *i* in order to provide an estimate of the magnitude of co-activation as well as the relative activity observed in the two muscle groups. The resulting values for each of 100 data points are averaged to produce a single value which represents the co-contraction index for each muscle pair during the entire stance phase of one walking trial. Kinematically- and kinetically-determined time points for heel-strike and peak knee flexion are used to modify *i* in Equation 2 in order to produce a co-contraction index for the weight-acceptance portion of stance phase. Subsequently, the mean co-contraction index over five trials was calculated for the four muscle pairs of each limb.

Although data is available for the evaluation of co-contraction over the entire gait cycle, the weight acceptance phase (from 100ms prior to heel strike to peak knee flexion) was selected for analysis. This interval was chosen because at this time, the knee musculature must respond as the stance limb accepts the body weight in order to control joint motion. Dynamic knee stiffness is also approximately linear during this interval,

allowing for the comparison of time-normalized data for both stiffness and co-contraction.

Kinematic and kinetic data reduction was accomplished with the use of Visual 3D (C-Motion, Inc., Germantown, MD). Visual3D was used to compute sagittal plane knee joint angles bilaterally for the entire stance phase of gait. Joint angles during gait were normalized to those of the standing calibration. Visual3D was also used to compute sagittal plane knee joint moments during the stance phase of gait. All moments were normalized by the subject's body mass × height in order to account for variations in body size.

Dynamic knee stiffness was defined as the observed resistance that a joint (including all the structures that transverse it) offers to control its rotation in response to an applied moment. Stiffness values were computed with the use of a custom-built LabVIEW program (LabVIEW 8.0.1, National Instruments, Austin, TX) according to Equation 2.2. The mean knee joint moment and angle was calculated for the stance phase of the same five usable trials which were selected for EMG analysis. Knee moment was plotted with respect to joint angle and displayed for visual inspection by the investigator (Figure 2.1).

Equation 2.2 Calculation of dynamic knee stiffness according to Farley et al. [30]:

$$\text{Stiffness}_{(\text{joint})} = \frac{\Delta\text{Moment}}{\Delta\text{Angle}}$$

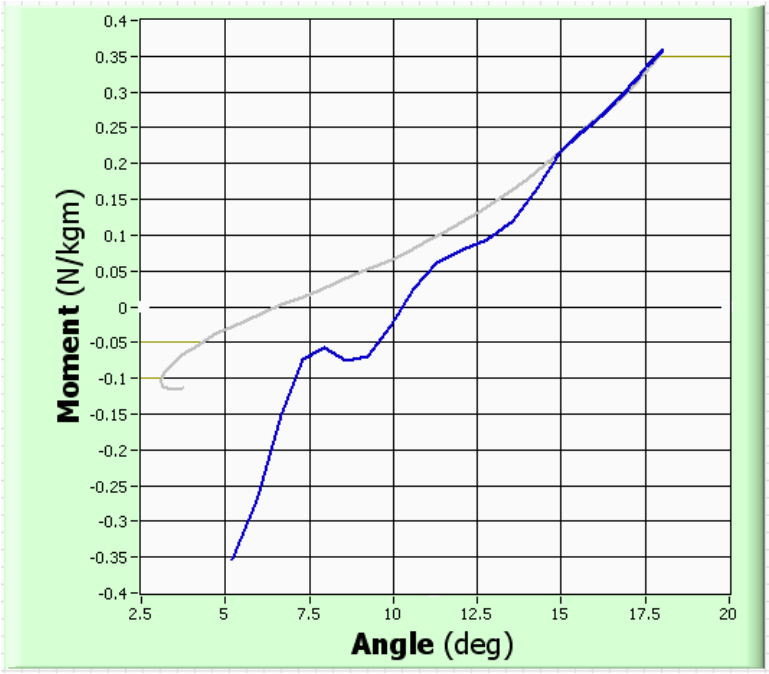

Figure 2.1 A typical knee moment-angle plot displaying the Weight Acceptance interval of gait for analysis in dark blue.

The interval for analysis was chosen to be the approximately linear portion of the moment-angle curve from its initial minimum (2-3% of stance phase) to its first maximum (peak knee flexion). A Best Linear Fit was applied to the moment-angle plot using a Least Square method, and the slope of this line was expressed as dynamic knee joint stiffness (k_r).

2.5 Statistical Analysis

Statistical Analysis of the data was performed with SPSS 16.0 (SPSS, Inc. Chicago, IL). Student's Paired T-Test was performed in order to evaluate differences in co-contraction indices and dynamic stiffness values between limbs. Significance was set

at p=0.05 for dynamic stiffness and p= 0.1 for co-contraction due to the high variability of EMG data. A Pearson product moment correlation was performed in order to determine the relationship between dynamic knee stiffness and co-contraction values during the above-specified portion of the weight-acceptance phase of gait. If correlations were found to be significant, linear regressions would be performed to determine the strength of the relationship.

Chapter 3

RESULTS

3.1 Comparison of Co-contraction Between Limbs

Student's Paired T-Tests revealed no significant differences in co-contraction indices for the six muscle pairs tested (Table 3.1). Mean values for quadriceps/hamstrings co-contraction indices (VL/LH and VM/MH) were higher in the involved limbs (Figure 3.1), although not significantly so.

Table 3.1. Significance values for differences in co-contraction indices between involved and uninvolved limbs for six muscle pairs.

Paired Muscles (Involved vs. Uninvolved)	Significance (n=41)
VL:LH	$p = 0.261$
VL:LG	$p = 0.373$
VL:MG	$p = 0.613$
VM:MH	$p = 0.124$
VM:MG	$p = 0.739$
VM:LG	$p = 0.919$

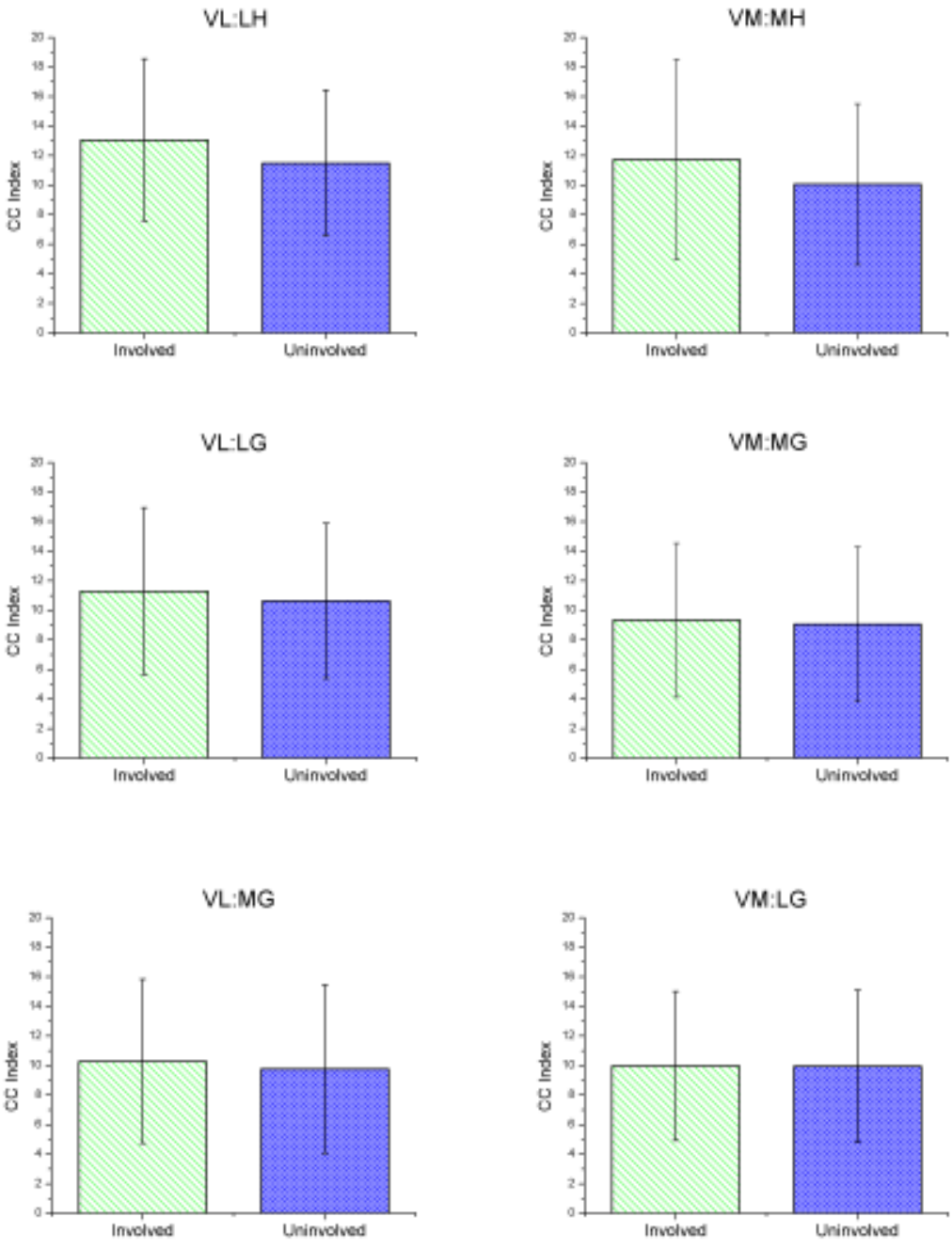

Figure 3.1 Co-contraction indices for involved and uninvolved limbs for six muscle pairs: VL/LH, VM/MH, VL/LG, VM/MG, VL/MG and VM/LG. Bars represent means ± 1 S.D.

3.2 Comparison of Dynamic Knee Stiffness Between Limbs

There was no significant difference in dynamic knee stiffness in the ACL-deficient knee compared to the control knee (Figure 3.2). Furthermore, a significant positive correlation was found between involved and uninvolved knee stiffness (Figure 3.3).

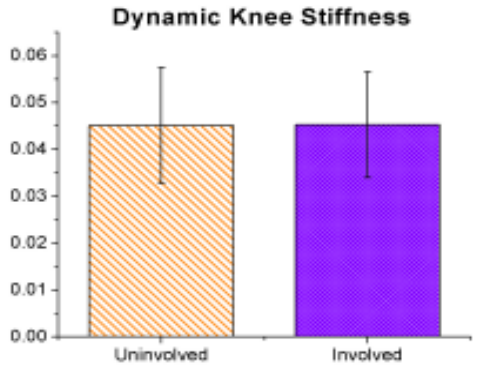

Figure 3.2 Dynamic knee stiffness values for involved and uninvolved limbs. Bars represent means, whiskers represent ±1.0 SD.

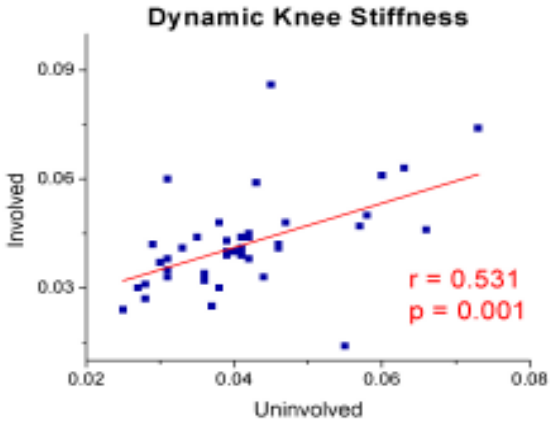

Figure 3.3 Pearson Product Moment Analysis showed dynamic knee stiffness to be linearly correlated between ACL-deficient and control knees.

3.3 Relationship Between Co-contraction and Dynamic Stiffness

Pearson's Product Moment Analysis revealed no significant correlations between dynamic knee stiffness and co-contraction for any of the six muscle pairs tested (Figure 3.4). Pearson's r-values and significance of correlations are visible in Table 3.2.

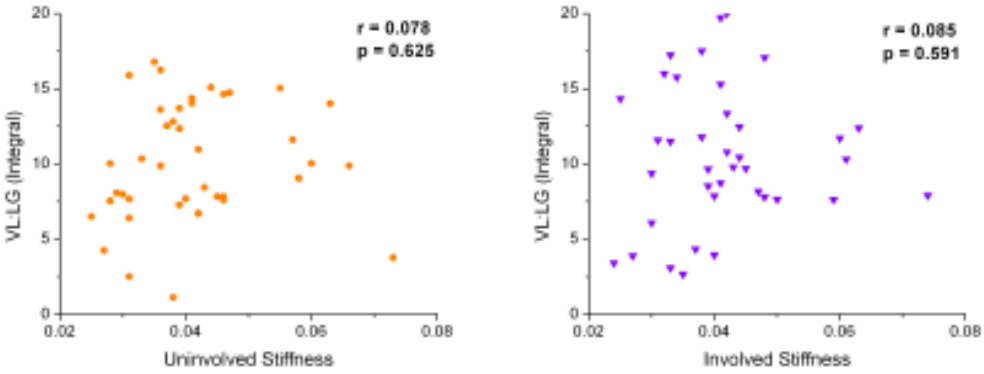

Figure 3.4. A typical set of graphs illustrating correlations of co-contraction and dynamic knee stiffness for involved and uninvolved limbs.

Table 3.2. Pearson's correlation coefficients (r) and their significance values (*p*) for Pearson Product Moment Analyses performed on each of six muscle pairs.

INV_STIFFNESS			UN_STIFFNESS	
r	*p*		r	*p*
0.085	0.591	**VL:LH**	0.027	0.866
0.053	0.738	**VL:LG**	0.078	0.625
-0.291	0.065	**VL:MG**	0.012	0.938
-0.033	0.839	**VM:MH**	-0.105	0.508
0.130	0.410	**VM:MG**	-0.037	0.814
-0.061	0.699	**VM:LG**	-0.001	0.994

Chapter 4

DISCUSSION

4.1 Differences in Co-contraction

Interestingly, our hypothesis regarding the presence of higher co-contraction in the ACL-deficient limb among non-copers was not supported by the data. This observation was different from reports of significantly elevated co-contraction in VL/LH and VM/MH of the involved limb during weight acceptance of walking by Hurd [5]. Significantly higher co-contraction was also reported in VL/MG of the involved limb during weight acceptance of jogging by Rudolph [6]; however, jogging is a more demanding task than normal walking, which may explain the presence of differences in co-contraction for weight acceptance of jogging where they are absent in normal gait.

Demographic data for the non-copers included in this study and for those reported by Hurd are displayed in Table 4.1. Subjects appear similar in both studies with respect to gender proportions and mean age, but not mean time from injury. Subjects in the present study were tested sooner after injury than those reported by Hurd. If the adoption of a neuromuscular compensation strategy in non-copers is viewed as a subconscious, experientially-based response to injury [44-47], then individuals who were tested further from the time of injury may develop strategies which were not discernible at an earlier

time point. Specifically, patients may complain of instability and report episodes of "giving-way," (defined as buckling or shifting on the injured knee). As time from injury increases, patients may be more likely to experience sensations of instability and begin to subconsciously develop a neuromuscular strategy of increased co-contraction in order to stabilize their injured knee and reduce shear forces. Comparisons between these two studies should be made with caution since it is possible that the asymmetrical co-contraction had not developed in our subject population at the time of testing.

Table 4.1 Comparison of non-coper sample demographics reported by Hurd [5] with those found in this study.

	Hurd 2007	**Present Study**
Sample size, n	21	42
Male ratio	71.4%	73.8%
Female ratio	28.6%	26.2%
Mean Age; (yrs) *(range)*	31.4 -	27.8 *(14-48)*
Mean Time from Injury (wks)	**11.4**	**8.6**

Despite the possibility that non-coper subject groups reported here and by Hurd [5] were different with respect to time from injury, the larger sample size tested in the present study (n=42) suggests the presence of more variability within the non-coper cohort than was initially postulated. A portion of the individuals initially classified as non-copers have been shown to successfully return to sport [48], implying that some non-copers possess viable neuromuscular strategies. Further evaluation of this potentially

variable cohort is needed to determine whether several distinct, consistent muscle activation patterns can be identified.

It is also possible that the differing results found in this study are affected by the methods used in interpretation of the EMG signal rather than inherent differences in the subject groups. For this reason, it is important to underscore the need for uniform methodology when comparing EMG-derived results between studies. There is no overall consensus on how to normalize EMG data, although there exist three predominantly-used methods [49]: (MEA) normalization to the mean amplitude of the EMG signal during a full gait cycle for a single subject, (MAX) normalization to 100% of the peak activity during a full gait cycle, and (MVC) normalization to the highest level of activity recorded during a discrete time window of either the maximum voluntary isometric contraction or dynamic trials recorded from each individual.

The MVC normalization method was used for this study, which is consistent with the methods reported by Rudolph [6] and Hurd [5]. However, it is important to note that slight algorithmic differences exist which may affect the comparability of the three non-coper studies: a 30ms discrete time window about the point of peak activation value was averaged for normalization purposes by Rudolph, whereas a 50ms window was used for this study (none was reported by Hurd). For EMG waveforms which rise, peak and fall quickly during dynamic activity, it is possible that a mean activity over a period of 30 and 50ms about a point of peak activation will result in a significantly different approximation of maximum activation for EMG normalization purposes.

4.2 Differences in Dynamic Knee Stiffness

Our hypothesis that dynamic joint stiffness would be different in the ACL-D knee compared to the intact knee was not supported by the data. Observations by other investigators of reduced knee motion accompanied by reduced external knee flexion moments in the involved limb during the ACL-D gait have been qualitatively identified as the "knee-stiffening strategy" [5-7, 45, 50, 51]. However, these references to stiffness should be regarded as conceptual descriptions only. Our results suggest that, during gait, the absence of the ACL does not result in a quantitatively discernible "knee-stiffening strategy" as measured by dynamic joint stiffness.

Although dynamic joint stiffness has been investigated as a parameter which may identify asymmetrical gait in patients with unilateral TKA [38], investigators reported a lack of asymmetry in the population which failed to confirm or deny the utility of dynamic joint stiffness in assessing asymmetrical gait. Results from the current study also fail to confirm the ability of this stiffness measure to successfully identify asymmetries during gait. Furthermore, the strong correlative relationship between involved- and uninvolved knee stiffness in this study suggest that dynamic knee stiffness may be found to be symmetrical between limbs for this population because it acts as a governing measure for the moment/excursion fluctuation during gait.

4.3 Relationships Between Co-contraction and Dynamic Stiffness

We hypothesized that stiffness would display a positively correlated relationship with co-contraction. However, our hypothesis was not supported; co-contraction varied

independently from dynamic stiffness during gait for the involved and the uninvolved limbs. Because these two independently-derived variables examined in this study are not quantitatively related, dynamic joint stiffness cannot be used as a surrogate for muscular co-contraction during walking for ACL-D individuals.

4.4 Conclusions

The non-coper population was selected for this study because they are known to exhibit an asymmetrical gait [4-8, 45, 52], and are reported to display aberrant levels of muscular co-contraction during dynamic activities [6, 50, 53, 54]. Our intent was to determine whether dynamic knee stiffness would reflect the asymmetry of the non-coper gait. Subsequently, we aimed to investigate the strength of the relationship between stiffness and co-contraction. Our expectation was that dynamic knee stiffness would indicate the extent to which co-activation is responsible for asymmetrical gait in the non-coper population.

The unexpected finding of the present work is that non-copers do not walk with significantly increased co-contraction in their involved limb when compared to their uninvolved limb. This differs from other, recent work concerning the non-coper neuromuscular strategy during gait. Further evaluation of this potentially variable cohort is needed to determine whether consistent neuromuscular patterns can be identified for comparison. Because dynamic joint stiffness was also found to be no different between limbs, it may be concluded that dynamic joint stiffness is not useful for characterizing asymmetries during walking. It may, in fact, act as a governing measure for the

moment/excursion fluctuation during gait. Due to the lack of statistical relationship between dynamic knee stiffness and co-contraction, we confirm that dynamic knee stiffness cannot be used as a surrogate for EMG data in discerning the presence of abnormal, asymmetrical or elevated co-contraction during gait.

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