ETIOLOGY OF PATELLOFEMORAL PAIN SYNDROME: A PROXIMAL LINK TO A DISTAL PROBLEM

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ETIOLOGY OF PATELLOFEMORAL PAIN SYNDROME: A PROXIMAL LINK TO A DISTAL PROBLEM

ABSTRACT OF DISSERTATION

A dissertation submitted in partial fulfillment of the requirement for the degree of Doctor of Philosophy in the Graduate School at the University of Kentucky

By

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ABSTRACT OF DISSERTATION

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Patellofemoral pain syndrome (PFPS) is one of the most common, but least understood, knee disorders. Fulkerson (1997) believes that pathology may result from an excessive valgus force being applied to the patella. Researchers have historically examined quadriceps strength and neuromuscular activity and knee kinematics. However, results from these works have not provided conclusive answers. Powers (2003) has theorized that other structures can influence knee function, and researchers have shown that PFPS subjects can exhibit hip weakness and demonstrate altered hip kinematics during functional activities. Although they provide preliminary evidence regarding hip influences, investigations that simultaneously examine hip and knee function in PFPS subjects are needed.

The primary purpose of this study was to determine functional performance, strength, neuromuscular activity (amplitudes and onset timing differences), and kinematics of the hip and knee for people diagnosed with PFPS. Eighteen females diagnosed with PFPS and 18 asymptomatic female controls participated. Subjects initially completed a 10-cm visual analog scale. Next, they completed two functional performance tests and underwent a strength assessment for the hip abductors, hip external rotators, and knee extensors. Surface electromyography (EMG) electrodes and reflective markers were donned in order to collect EMG and kinematic data during a stair-stepping task. For this purpose, subjects ascended and descended two 20-cm high steps at a standardized rate. Seven PFPS and seven control subjects were retested five to seven days later to establish measurement reliability.

A repeated measures analysis of variance was used to determine group differences. Correlation coefficients were calculated to identify associations between pain and dependent measures; intraclass correlation coefficients were calculated to determine measurement reliability for both control and PFPS subjects. Results from this study showed group differences for functional performance, strength, and EMG amplitudes but none for onset timing differences or kinematics. A strong association was found between pain and hip external rotator strength and EMG amplitudes during stair-stepping.
Most tests provided reliable measures with repeat testing. PFPS subjects demonstrated quadriceps dysfunction but even greater hip weakness that was correlated more with pain. Contemporary rehabilitation has focused on quadriceps strengthening; however, results from this study support the importance of the hip.

KEYWORDS: patellofemoral pain syndrome, strength, electromyography, kinematics, hip

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DISSERTATION

Lori Ann Bolgla

The Graduate School
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2005
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CHAPTER ONE

The Continuing Problem

Patellofemoral pain syndrome (PFPS) is a common problem experienced by active adults and adolescents (Brody & Thein, 1998; Thomee, Augustsson, & Karlsson, 1999); however, its etiology has remained vague and controversial (Powers, 1998; Witvrouw, Lysens, Bellemans, & Peers, 2000; Witvrouw et al., 2005). Most often, patients complain of diffuse peripatellar and retropatellar pain that may limit their ability to perform activities of daily living that require loading on a flexed knee. Such activities include ascending and descending stairs, squatting, and sitting for prolonged periods of time (Doucette & Goble, 1992; Fulkerson, 2002; Heinjes et al., 2004; Witvrouw, Lysens, Bellemans, & Peers, 2000).

Researchers have described PFPS as abnormal lateral patella movement on the femur during non-weight bearing knee extension (Doucette & Goble, 1992; Fulkerson, 2002); however, PFPS patients typically report impairments during weight bearing activities, like squatting and stair climbing. Therefore, differences in patellar tracking may exist during weight bearing and non-weight bearing activities (Powers, 2000).

Powers et al. (2003) examined movement of the femur and patella during weight bearing and non-weight bearing knee extension using kinematic magnetic resonance imaging. They reported lateral patella movement on the femur during non-weight bearing exercise. Moreover, they found increased femoral internal rotation under a relatively stable patella during the weight bearing activity. This finding demonstrated that excessive hip internal rotation, not patella movement, caused relative lateral tracking. These results are important clinically because they implicated the hip in patellofemoral joint pathology.

Recently, researchers have examined hip neuromuscular influences on the knee. Brindle et al. (2003) reported a greater delay in gluteus medius (GM) activation relative to the vastus medialis oblique (VMO) during stair climbing in subjects diagnosed with PFPS. Nyland et al. (2004) compared GM and vastus medialis (VM) activation amplitude ratios in varying positions of femoral internal rotation and found lower amplitudes in subjects exhibiting increased femoral internal rotation. Other researchers have reported significant hip weakness in subjects diagnosed with PFPS (Ireland,
Willson, Ballantyne, & Davis, 2003; Niemuth, Johnson, Myers, & Thieman, 2005). Although these studies provide preliminary evidence regarding hip influences, additional investigations that simultaneously examine hip and knee function in PFPS subjects are needed.

Clinicians use various evaluation tools to identify impairments related to strength, muscle activation, and movement. Examples of measurement tools have included functional performance tests (Loudon, Wiesner, Goist-Foley, Asjes, & Loudon, 2002), strength (Andrews, Thomas, & Bohannon, 1996; Bohannon, 1997), surface electromyography (EMG) (Cowan, Bennell, Hodges, Crossley, & McConnell, 2001), and motion analysis (Brechter & Powers, 2002). A review of the literature failed to identify any studies that specifically provided kinematic and EMG measurement reliability for PFPS subjects. Only one has examined measurement reliability for PFPS-specific functional performance tests (Loudon et al., 2002). Since clinicians assess impairments using these tools, identification of those capable of differentiating subjects diagnosed with and without PFPS may enhance the evaluation process.

**Purpose**

The primary purpose of this research project was to investigate the role of the hip on PFPS. A secondary purpose was to determine measurement reliability for tools clinicians commonly use when evaluating people diagnosed with PFPS. This study was designed to address the following questions:

1. Do functional performance tests, hand-held dynamometry, surface EMG, and motion analysis provide reliable measures of function for people diagnosed with PFPS?
2. Can functional performance tests, hand-held dynamometry, surface EMG, and motion analysis discriminate between people diagnosed with and without PFPS?
3. Do people diagnosed with PFPS demonstrate excessive hip weakness compared to age-matched controls and, if so, do strength differences result in altered muscle activation patterns?
4. Do people diagnosed with PFPS demonstrate excessive femoral internal rotation, femoral adduction, and knee valgus during the descent phase of stair stepping?
Overview

Information specific to each question has been synthesized into the following sequence. Chapter 2 summarizes issues related to measurement reliability. Chapter 3 examines the interrelationships between functional performance tests, muscle strength, and EMG activity during the descent phase of stair stepping. Chapter 4 compares hip and knee kinematics between people diagnosed with and without PFPS. Chapter 5 summarizes findings from all aspects of the study to determine which parameters may be more indicative of a person having PFPS.

Operational Definitions

For purposes of this study, the following definitions were used:

Patellofemoral Pain Syndrome

PFPS was defined as retropatellar or peripatellar pain. It excluded pathology resulting from osteoarthritis, direct trauma, soft tissue injury, or specific neurological dysfunction.

Subject Inclusion Criteria

Female subjects diagnosed with PFPS participated in this study if they complained of: 1) anterior knee pain during the descent phase of stair stepping and 2) pain during two of the following provocative activities: a) stair ascent, b) squatting, c) kneeling, or d) excessive sitting. They also rated usual knee pain over the previous week at a minimum of 3 on a 10-cm visual analog scale (Cowan, Bennell, & Hodges, 2000). The most affected lower extremity was tested for PFPS subjects (Powers, Landel, & Perry, 1996).

Control subjects participated in this study if they had 1) no history or diagnosis of knee pathology, 2) no pain with any of the above-named provocative activities, and 3) no history of hip pathology. The right lower extremity was tested for control subjects (Mohr, Kvitne, Pink, Fideler, & Perry, 2003; Owings & Grabiner, 2002).

Subject Exclusion Criteria

Female subjects were excluded from the study if they had 1) previous knee surgery or significant injury, 2) traumatic patellar dislocation, 3) any neurologic involvement that would affect gait, or 4) previous hip surgery or significant injury (Brindle et al., 2003; Powers et al., 1996). Inclusion and exclusion criteria were
consistent with other published literature (Brechter & Powers, 2002; Brindle et al., 2003; Cowan et al., 2001; Crossley, Bennell, Green, Cowan, & McConnell, 2002; Ireland et al., 2003; Powers, Chen, Reischl, & Perry, 2002; Powers et al., 2003).

**Functional Performance Tests**

Functional performance tests were used to evaluate overall lower limb function in subjects diagnosed with PFPS (Risberg & Ekeland, 1994). These tests were conducted under controlled clinical conditions to assess the lower extremity during specific activities that typically elicit pain and dysfunction in people diagnosed with PFPS.

**Strength**

Strength was defined as the maximum isometric torque that subjects generated for specific hip and knee muscles during manual muscle testing. Isometric torque represented the force recorded on a hand-held dynamometer (HHD) multiplied by the perpendicular distance of the HHD from the specific joint center of rotation.

**Stair-Stepping Task**

The stair-stepping task required subjects to walk across a level platform, ascend and descend 2 steps (using a reciprocal pattern), and continue walking across the level platform. Subjects typically took 3 strides prior to and immediately following stair stepping. The stairs consisted of steps having a 20-cm height, 30-cm tread depth, and 47-cm width (See Figure 1.1).

**Stair Descent**

Hip and knee EMG activity and kinematic data were collected during stair descent. Stair descent began at the point of initial foot contact as the subject descended the third step and ended at the point of ipsilateral foot contact onto the floor (Yu, Kienbacher, Grownney, Johnson, & An, 1997). Clinically, PFPS patients typically complain of pain and dysfunction during the stance phase of stair descent. Mascal et al. (2003) also demonstrated a possible relationship between faulty hip motion and PFPS when observing subjects during the stance phase of stair descent. Therefore, this study only examined EMG activity and kinematics during the stance phase.

To identify differences in EMG activity throughout the stance phase of stair descent, stance was subdivided into the following intervals: 1) loading response, 2) single leg stance, and 3) preswing (Mohr et al., 2003). Loading response began at the initial
point where any part of the ipsilateral foot contacted the step and ended as subjects lifted the contralateral foot off the previous step (e.g., initial double leg stance). Single leg stance occurred when the test extremity supported the entire body mass during stair descent. Preswing began when any part of the contralateral foot contacted the ground and ended as subjects lifted the test extremity’s foot off the stair (e.g., terminal double leg stance). Figures 1.2 through 1.4 illustrate each interval.

Based on temporal data collected from the subjects during stair descent, the cycle for stair descent was divided into the following intervals: 1) loading response = 0% to 7% of stair descent, 2) single leg stance = 8% to 46% of stair descent, and 3) preswing = 47% to 58% of stair descent. The remaining 42% of stair descent represented the swing phase. See Appendix G for a more detailed explanation.

Assumptions

The following assumptions were made for this study:

1. It was assumed that all subjects provided their best effort during strength and functional performance testing.
2. It was assumed that PFPS subjects provided an accurate history concerning insidious onset of patellofemoral joint pain and no history of any other lower extremity injury.
3. It was assumed that PFPS subjects had no other knee pathology if not found on clinical examination.
4. It was assumed that all control subjects accurately reported no previous history of lower extremity injury.
5. It was assumed that control subjects had no knee pathology if not found on clinical examination.
6. It was assumed that no subjects had osteoarthritic changes to the patellofemoral joint.
Limitations

This study was limited by the following factors:

1. A sample of convenience was utilized for this study.
2. Some subjects might have had previous exposure to the type of skills used for functional performance testing.
3. Most subjects in both groups represented college-aged students from the University of Kentucky.
4. Some subjects might have practiced the type of skills used for testing between initial and repeat testing.
5. The primary investigator was not blinded to group assignment during data collection or data analysis.

Figure 1.1
Stairs Used for the Stair-Stepping Task.
Figure 1.2
Loading Response Interval of Stair Descent. Loading response begins at initial foot contact with the third step and ends when the contralateral foot is lifted off the second step (e.g., initial double leg stance).
Figure 1.3
Single Leg Stance Interval of Stair Descent. Single leg stance begins when the contralateral foot is lifted off the second step and ends when the contralateral foot touches the floor.
Figure 1.4
Preswing Interval of Stair Descent. Preswing begins when the contralateral foot contacts the ground and ends as test extremity’s foot is lifted off the third step (e.g., terminal double leg stance).
CHAPTER TWO

Reliability of Evaluation Tools for Assessing Patellofemoral Pain Syndrome

PFPS has remained one of the most commonly seen and clinically challenging pathologies (Wilk, Davies, Mangine, & Malone, 1998; Witvrouw, Lysens, Bellemans, & Peers, 2000). Dye (1997) has described PFPS as the “Black Hole of Orthopaedics” because of differences in reported etiology. As such, investigators have examined knee strength (Natri, Kannus, & Jarvinen, 1998; Thomee, Renstrom, Karlsson, & Grimby, 1995; Witvrouw, Lysens, Bellemans, Peers, & Vanderstraeten, 2000), quadriceps activation patterns (Cowan, Bennell, Crossley, Hodges, & McConnell, 2002; Cowan, Bennell, & Hodges, 2002; Cowan et al., 2001; Owings & Grabiner, 2002; Voight & Wieder, 1991; Witvrouw et al., 2003), and knee kinematics (Brechter & Powers, 2002; Crossley, Cowan, Bennell, & McConnell, 2004; Nadeau, Gravel, Hebert, Arsenault, & Lepage, 1997; Powers, Heino, Rao, & Perry, 1999) in an attempt to better understand PFPS etiology.

Recently, there has been a focus on the importance of the hip musculature on knee function. Researchers have found an association between hip weakness (Ireland et al., 2003; Niemuth et al., 2005) and delayed GM activation relative to that of the VM and VL in subjects diagnosed with PFPS (Brindle et al., 2003). Furthermore, preliminary research has shown that people diagnosed with knee pain respond favorably to rehabilitation programs emphasizing hip strength (Fredericson et al., 2000; Mascal et al., 2003; Pettitt & Dolski, 2000). Results from these studies suggest that the hip can positively influence knee function; however, additional investigations are needed to firmly establish this relationship.

Many measurement tools are available to assess hip and knee function. Clinicians routinely assess strength using HHD and functional performance tests (FPT). Researchers have employed surface EMG to determine muscle activation amplitudes and temporal characteristics and motion analysis to calculate lower extremity joint angles during dynamic activities. Together, these measurement tools can help identify interactions between the hip and knee that may result in patellofemoral pathology.

An important concept related to measurement is reliability. Portney and Watkins (2000a) have defined reliability as “the extent to which a measurement is consistent and
free from error.” It means that observed changes with repeat testing occur from true
differences in the parameter being assessed. Measurement reliability is important
because it provides evidence that the evaluation tool can detect true differences in subject
behavior, if in fact they exist. For example, improved quadriceps strength should reflect
an increase in torque generated rather than a different application of the measurement
tool.

Many investigators have examined the reliability of HHD; yet few have
determined the reliability of FPT, surface EMG, and motion analysis. Furthermore, most
reliability studies have been conducted using a normal subject population. With the
exception of FPT (Loudon et al., 2002), none have determined the reliability of these
measurement tools specific to subjects diagnosed with a pathology, like PFPS. Although
it would be expected that these tools would provide reliable measures for PFPS subjects,
data are needed to support this premise.

Review of the Related Literature

Functional Performance Tests

Rivera (1994) states that function depends on the optimal integration of all joints
and muscles involved in a particular action. It cannot be adequately measured, trained, or
predicted from the isolation of a particular muscle group while performing an abnormal
movement pattern. Researchers have designed FPT that simulate the stresses about the
knee encountered during functional or athletic activities (Lephart et al., 1992). FPT are
important in assessing lower extremity function because they encompass many variables
such as pain, neuromuscular coordination, muscle strength, and joint stability (Barber,
Noyes, Mangine, & DeMaio, 1992).

Patients diagnosed with PFPS typically complain of pain during activities that
require loading on a flexed knee. Loudon et al. (2002) first described the following FPT
designed to simulate demands placed on the patellofemoral joint during functional
activities: 1) anteromedial lunge, 2) step-down, 3) single-leg press, 4) bilateral squat, and
5) balance and reach. They determined intrarater reliability by testing PFPS and control
subjects on 2 occasions, 48 to 72 hours apart, and calculating intraclass correlation
coefficients (ICC [3,1]) and standard errors of measurement (SEM). They also calculated
Pearson correlation coefficients to determine associations between FPT values and pain.
Results from this study showed that the step-down test was the most reliable measurement (ICC = .94; SEM = .53) and was moderately correlated to pain ($r = .57$). The anteromedial lunge had a strong correlation with pain ($r = .73$) and good reliability (ICC = .82; SEM = .38). Although the remaining tests provided reliable measures, none had greater association with pain than the step-down and anteromedial lunge tests. Based on these results, this study used the step-down and anteromedial lunge FPT because of their acceptable intrarater reliability and higher correlations with pain.

**Strength**

Strength measurements are an important part of assessment because they provide baseline data and information concerning improvement and intervention efficacy (Wikholm & Bohannon, 1991). Historically, clinicians have assessed strength using manual muscle testing (MMT), a system based on a 5-point grading scale. Although MMT has been reported to be a reliable tool (Wadsworth, Krishnan, Sear, Harrold, & Nielsen, 1987), it has inherent flaws. Wikholm and Bohannon (1991) have shown that MMT may depend on the examiner’s ability to exert sufficient strength to counteract the muscle action being tested. Another limitation of MMT is the inability to detect subtle changes in strength (Wadsworth et al., 1987).

HHD is an alternative method for measuring strength. HHD may be superior to MMT because it enables an objective manner for estimating strength (force applied to the HHD times the external moment arm) and is more sensitive to subtle changes. Although HHD may improve measurement precision, it has potential flaws that deserve consideration.

First, like MMT, the examiner must ensure proper stabilization of the limb segments to avoid substitution from other muscle groups. Second, the HHD should be applied securely to the test limb. Agre et al. (1987) have shown that inadequate application can affect a subject’s ability to exert a maximum contraction. Third, exertion of a maximum contraction depends on the amount of examiner resistive force applied. This means that a smaller-sized examiner may not have sufficient strength to securely hold the HHD against resistance applied from a larger subject (Wikholm & Bohannon, 1991).
To alleviate a bias from examiner size or strength, Kramer et al. (1991) investigated the efficacy of using a resistive belt to collect HHD measures. They hypothesized that subjects would exert a more consistent force against an immovable strap. To test this hypothesis, the test-retest reliability of hip abductor isometric strength was determined under 2 different test conditions using a group of younger and older females. The first condition required that subjects generate force against a HHD held by the examiner. The second condition was a belt-resisted method, where each subject exerted force against a HHD secured to the lateral aspect of the thigh by an immovable strap. They tested subjects on 2 separate occasions; calculated ICCs ranged from .84 to .98. Lower ICCs were found in the younger subjects under the examiner-resisted condition. Higher ICCs were reported for the older subjects under both conditions and younger subjects under the belt-resisted condition.

Because younger subjects were expected to be stronger than older subjects, their efforts could have depended on the resistance provided by the examiner. Therefore, differences in examiner resistance applied may have accounted for the younger subjects’ greater variability. Alternatively, the belt-resisted method provided a strong, consistent resistance. All subjects generated greater force using the belt-resisted method, regardless of age, and the researchers believed that patients “had a greater tendency to trust their own resistance, over which they had control, and were less hesitant to perform strong contractions in this manner.”

Agre et al. (1987) found similar results as the Kramer study. In this study, they examined intrarater and interrater reliability of HHD for the upper and lower extremities. Four subjects performed maximal contractions three to four times for each muscle group being tested. Pearson correlation coefficients for the upper extremities ranged from .85 to .99; those for the lower extremity ranged from -.20 to .96. The researchers attributed differences in reliability coefficients to intratrial variability. For example, during upper extremity testing, the examiner could easily stabilize the HHD. They could also provide greater resistance to the muscle action, which resulted in less variability (5.1% to 8.3%) between individual trials. However, examiners had greater difficulty stabilizing the HHD and resisting muscle action for many of the lower extremity muscles. This resulted in intratrial variability that was much higher than upper extremity muscles (11.3% to
Agre et al. concluded that intratrial variability of 10% or less should improve HHD reliability.

More recent studies have found good reliability for measuring lower extremity strength. Nadler et al. (2000) examined intrarater reliability of the hip abductors and extensors using a HHD that had a specially designed anchoring station (to improve HHD stability and applied resistance). ICCs for this study ranged from .94 to .98 and coefficients of variation between trials did not exceed 8.06%. Click Fenter, Bellew, Pitts, & Kay (2003) conducted a similar study and reported similar ICCs. Together, these studies demonstrate that reliable measures could be obtained for lower extremity HHD with the use of adequate stabilization and resistance.

A limitation of these studies has been unreported data for other lower extremity muscles. It has been hypothesized that hip external rotation strength is associated with PFPS (Ireland et al., 2003); however, no studies have assessed the reliability of these measures. With respect to the quadriceps, researchers have only established reliability with subjects positioned in 90 degrees of knee flexion. However, many researchers (Mohr et al., 2003; Powers, 2000; Powers, Perry, Hsu, & Hislop, 1997; Selseth, Dayton, Cordova, Ingersoll, & Merrick, 2000) have tested the quadriceps in 60 degrees of flexion because it is thought that subjects can generate a stronger contraction in this position. No studies have determined quadriceps strength reliability with the knee positioned in 60 degrees of flexion using HHD.

In summary, HHD can provide reliable measures of lower extremity strength assuming that testing is conducted with adequate HHD stabilization and applied resistance. The current review of the literature has recommended the average of 3 trials having coefficients of variation less than 10%.

Surface Electromyography

It has been theorized that delayed onset of the VM relative to the VL can cause lateral tracking of the patella and contribute to PFPS. Although researchers (Cowan et al., 2001; Owings & Grabiner, 2002; Powers et al., 1996; Sheehy, Burdett, Irrgang, & Van Swearingen, 1998; Voight & Wieder, 1991; Witvrouw et al., 2003) have examined VM to VL onsets, only two have examined the reliability of such measures. Gillett, McConnell, & Parsons (1998) determined the test-retest reliability of relative VMO and
VL activity onset using 3 asymptomatic subjects during stair stepping. In this study, they reported an ICC [3,1] of .78. It should be noted that EMG signals were full-wave rectified and low pass filtered using a 3 Hz cutoff frequency. Hodges and Bui (1996) have stated that excessive signal smoothing can hinder proper determination of onsets and suggest that applying a low pass filter of 50 Hz can result in accurate and consistent determinations of EMG onsets. Therefore, the manner in which Gilleard et al. processed their data might have affected proper identification of muscle onsets.

Cowan et al. (2000) tested 10 asymptomatic subjects on 2 occasions to determine test-retest reliability of detecting onset activity of the VMO and VL. Subjects in this study ascended and descended 2 steps (20-cm in height) at a rate of 96 beats per minute, a rate used previously by Gilleard et al. (1998). They chose this rate to standardize performance for purposes of increasing repeatability. EMG data were preamplified at a gain of 1000, sampled at 1000 Hz, and band pass filtered between 20 and 500 Hz. Raw EMG signals were then full wave rectified and low pass filtered at 50 Hz.

Cowan et al. (2000) used a computer algorithm, in combination with visual inspection, to identify EMG onsets since an algorithm was thought to increase the objectivity of analysis (Hodges & Bui, 1996). Specifically, they defined an onset of muscle activity as the point in which the signal deviated by more than 3 standard deviations, for a minimum of 25 ms, over the baseline level taken 200 ms before the trial began. All onsets were also visually confirmed since movement artifact could have caused onset activity. They quantified onset differences by subtracting the VMO onset from the VL onset. A negative difference meant a delay in VMO activation relative to the VL where as a positive difference signified VMO preactivation.

The researchers reported ICCs of .91 and .96 for the concentric and eccentric phases of stair stepping, respectively. These values suggest that the stair stepping test and signal processing parameters are reliable measures for determining muscle activity onset of the VMO and VL. A limitation of the study was that it only used asymptomatic subjects. If quadriceps onset timing differences are important parameters to evaluate, then additional studies are needed to determine if such differences are reliable for subjects diagnosed with PFPS. For purposes of the current study, the methods described by Cowan et al. (2000) were duplicated to enable comparison among studies.
Researchers have also examined EMG amplitudes during non-weight bearing knee extension (Owings & Grabiner, 2002; Powers, 2000), ambulation (Powers et al., 1996), and stair stepping (Mohr et al., 2003; Sheehy et al., 1998) between subjects diagnosed with and without PFPS. Some have reported decreased quadriceps amplitudes in subjects diagnosed with PFPS where as others have found greater activation. Contrasting results support the need for additional studies to better understand muscle activity in PFPS patients. A limitation of these studies has been the varied methods for collecting and analyzing EMG activity. Therefore, the determination of a single, reliable method for investigating EMG amplitudes would enhance the current body of knowledge.

Motion Analysis

People diagnosed with PFPS typically complain of pain when descending stairs. Stair descent has represented an important functional activity that may be a more informative clinical evaluation tool than level walking, especially for people diagnosed with PFPS (Yu et al., 1997). In order to use kinematic data during stair-stepping as an assessment tool, its reproducibility must be established.

Few researchers have examined lower extremity kinematics during stair-stepping and the reliability of these measures. Andriacchi et al. (1980) initially investigated sagittal plane lower extremity kinematics but did not assess reliability. McFadyen and Winter (1988) examined the intrasubject and intersubject variability of sagittal plane motion; however, their study only included 3 subjects. Sagittal plane of motion can provide important information; however, the transverse and frontal planes of motion might provide more clinically relevant data (Powers, 2003).

Yu et al. (1997) are the only researchers who have determined intrasubject reproducibility of frontal and transverse plane lower extremity kinematics in asymptomatic subjects during stair-stepping. They collected video data using Expert Vision (Motion Analysis Corporation, Santa Rosa, CA) and analyzed data using OrthoTrak software (Motion Analysis Corporation). They normalized all kinematic data to 100% of the gait cycle; defined as the time from foot contact on a step until the next foot contact of the same foot. For testing purposes, subjects performed 3 trials of the task. Coefficients of multiple correlations for all joint angles were calculated to determine reliability. Although flexion-extension angles were more reproducible for all
lower extremity joints, those for hip and knee adduction-abduction and hip internal-external rotation were acceptable as evidenced by correlations of .85. These results inferred that the researchers obtained reliable measures of kinematics during stair-stepping.

Although Yu et al. (1997) provided preliminary results regarding motion analysis reliability during stair-stepping, additional studies are indicated. First, this study only examined intrasubject reproducibility during a single testing session. If clinicians use motion analysis as an evaluation tool throughout a rehabilitation period, then they need information regarding between-day reliability. When studying normal gait, Kadaba and colleagues (Kadaba, Ramakrishnan, & Wootten, 1990; Kadaba et al., 1989) found variability resulting from day-to-day marker reapplication. Because of this potential bias, further studies should determine day-to-day reliability specific to stair-stepping.

Second, all previous studies have only included normal subjects. It is not known if subjects diagnosed with PFPS will utilize similar, or consistent, movement patterns of the hip and knee during stair-stepping. Performance may be highly variable when assessing a symptomatic subject group, which may limit the clinical utility (ability to identify true changes in behavior) of motion analysis. Therefore, this study will examine the reliability of motion analysis in a group of subjects diagnosed with PFPS.

**Purpose and Research Hypothesis**

The purpose of this study was to compare the test-retest reliability of FPT, HHD, surface EMG, and motion analysis for subjects diagnosed with and without PFPS. It was hypothesized that all tests would provide reliable measures as evidenced by ICCs exceeding .75 (Portney & Watkins, 2000a).

**Methodology**

**Subjects**

Seven females diagnosed with PFPS (age = 22.9 ± 2.7 years, height = 1.69 ± .1 m, body weight = 588.6 ± 54.9 N, pain = 4.8 ± 2.0 cm, duration of symptoms = 14.4 ± 12.8 months) and 7 asymptomatic females (age = 24.0 ± 2.9 years, height = 1.66 ± .1 m, body weight = 588.6 ± 103.0 N) participated in this study. All subjects met the inclusion criteria, as summarized in Chapter 1, and signed an informed consent approved by the University of Kentucky Institutional Review Board prior to participation.
Procedures

First, subjects completed a 10-cm visual analog scale reflecting usual pain during the past week (Crossley, Bennell, Cowan, & Green, 2004). Next, they rode a stationary bicycle ergometer for 3 minutes in a pain-free range of motion at a submaximal speed and practiced each functional performance test 3 to 5 times (Loudon et al., 2002). Subjects then performed the step-down and anteromedial functional performance tests, as described in Appendix E, in a random order to reduce ordering bias. The number of repetitions performed for each lower extremity within a 30-second period was documented.

Next, the distances from the greater trochanter to the lateral femoral condyle and the lateral knee joint line to the lateral malleolus were measured. These measurements were conducted to determine the perpendicular distance from the HHD to the hip and knee joints, respectively. This information was used to report all strength values as measures of torque in units of newton*meters (N*m).

Subjects’ skin was prepared for EMG instrumentation by shaving, abrading, and cleansing it with isopropyl alcohol prior to application of surface electrodes. Bi-polar Ag-AgCl surface electrodes (Medicotest, Rolling Meadows, IL), measuring 5 mm in diameter with an interelectrode distance of approximately 20 mm, were placed in parallel arrangement over the muscle bellies of the GM, VM, and VL. The GM electrode was placed 1/3rd the distance between the iliac crest and greater trochanter (Cram & Kasman, 1998). The VM electrode was placed approximately 4 cm superior to and 3 cm medial to the superomedial border of the patella and oriented 55° to the vertical (Cowan et al., 2000). The VL electrode was placed 5 to 7 cm superior to and 6 to 8 cm lateral to the superolateral border of the patella and oriented 15° to the vertical (Cram & Kasman, 1998). Electrodes were further secured to the skin with an adhesive tape to prevent slippage during testing. Electrode placement sites were recorded on a data collection sheet so that they could be repositioned correctly during repeat testing. A ground electrode was placed on the ipsilateral clavicle. Electrode placements were visually confirmed on an oscilloscope using manual muscle testing techniques. A 3-second standing "quiet" file was also recorded to exclude ambient noise.
Following EMG placement, strength measures were taken for the hip abductors, hip external rotators, and knee extensors. Subjects were positioned as described in Appendix E. For testing, subjects produced a maximal isometric contraction using the “make” test (Andrews et al., 1996; Bohannon, 1997) to the beat of a metronome set at 60 beats per minute. They generated maximum force over a 2-second period and maintained this force for an additional 5 seconds to the beat of the metronome. Subjects performed one practice (Andrews et al., 1996; Bohannon, 1997) and 3 test trials, with a 30-second rest period between trials. A coefficient of variation was calculated and an additional trial was taken, if necessary, to ensure that subjects had 3 measures with variability less than 10% (Agre et al., 1987). The order of muscle testing was counterbalanced to account for any potential bias. All measures were recorded in newtons (N) of force. EMG activity was simultaneously collected for the GM, VM, and VL during strength testing to determine a maximum voluntary isometric contraction (MVIC) for each muscle.

Next, retroreflective markers, with a diameter of 20 mm, were placed on subjects using a standard Cleveland Clinic marker setup. After collecting an anatomic calibration file, subjects were shown the stair stepping task. They were instructed to ascend and descend two 20-cm high steps, ensuring that the test extremity lifted and lowered the body on the first and third steps, respectively. Subjects also took a minimum of 3 strides prior to and immediately following stair stepping in order to maintain a continuous movement pattern. Because movement velocity may influence EMG activity, subjects performed the task at a standardized rate of 96 beats per minute (Cowan et al., 2000; Gilleard et al., 1998). Subjects performed 5 practice trials prior to data collection.

Subjects performed 10 test trials. During this time, EMG data were sampled at 960 Hz and recorded synchronously with the video data, which were sampled at 60 Hz. Data from the last 5 trials were analyzed because of potential learning effects that might have been associated with earlier trials, even though subjects had performed 5 practice trials. Refer to Appendix C for unit specifications of the EMG and motion analysis equipment used in this study.
All subjects returned to the laboratory within 5 to 7 days for repeat testing. They performed all tests in the same manner described above. Subjects completed another visual analog scale because differences in pain may affect functional test performance. Loudon et al. (2002) recommended that repeat visual analog scale scores be ± 0.5 cm of the original score to prevent confounding of the pain variable. All subjects participated in the second part of this study.

**Data Processing**

**Functional performance tests.** For each FPT, the total number of repetitions completed by subjects on the involved (PFPS) or the right lower extremity (controls) was used for statistical analysis.

**Strength.** Strength was expressed in units of torque by multiplying the force recorded on the HHD by the perpendicular distance from the HHD to the joint center of rotation. Average torque was then normalized to subject height and weight (% [body weight (N) * height (m)] = torque * {100/[body weight (N) * subject height (m)]}) to allow for comparison among subjects (Fredericson et al., 2000). These values were used for statistical analysis.

**EMG data.** Raw EMG signals were processed in the manner described in Appendix D. To determine muscle activation amplitudes, EMG data from the last 5 trials were root mean square (RMS) smoothed using a 55 ms time constant and normalized to 100% of the stair descent cycle. They were then ensemble averaged and expressed as a % MVIC. Datapac software (Run Technologies, Mission Viejo, CA) then calculated the average % MVIC EMG amplitude for each muscle during the 1) loading response, 2) single leg stance, and 3) preswing intervals of stair descent (see Appendix G). The resulting values were used for statistical analysis.

Muscle activation onsets were determined at the beginning of stair descent. After processing EMG signals and identifying muscle onsets (See Appendix D), Datapac software calculated timing differences. The program subtracted the GM onset from the VM onset and VL onset, respectively, to quantify timing differences between the hip and knee musculature. A negative difference signified a delay in GM activation relative to the VM and VL where as a positive difference meant GM preactivation. The software also subtracted the VM onset from the VL onset to quantify quadriceps timing
differences. A negative difference meant a delay in VM activation relative to the VL where as a positive difference signified VM preactivation. The average from the last 5 trials was later used for statistical analysis.

**Kinematics.** Video data were processed in the manner described in Appendix D. Hip transverse plane, hip frontal plane, and knee frontal plane angles for individual trials were calculated by OrthoTrak 5.0 software (Motion Analysis Corporation) using methods described by Grood and Suntay (1983). Table 2.1 summarizes the conventions used to describe joint angles for the current study. The last 5 individual trials were then normalized to 100% of the gait cycle and ensemble averaged. Average joint angles from the normalized data during the entire stance phase of stair descent were used for statistical analysis.

**Statistical Analysis**

ICCs (Shrout & Fleiss, 1979) were used to determine between day reliability; standard errors of measurement (SEM) were used to determine measurement precision (Denegar & Ball, 1993) for all dependent measures. ICC [3, 1] was calculated for FPT since these measures represented a single value. ICC [3, 3] was calculated for all strength measures since they represented the average of 3 trials; ICC [3, 5] was calculated for all EMG and kinematic values since they represented the average of 5 trials. Statistical analyses were performed using SPSS version 12.0 (SPSS, Inc., Chicago, IL). Level of significance was established at the 0.05 level.

**Results**

Tables 2.2 through 2.6 summarize between day ICCs and SEMs for PFPS subjects and controls for FPT, strength, EMG activation amplitudes, EMG timing differences, and kinematics. Tables 2.7 through 2.12 summarize means and standard deviations for all dependent measures for testing days 1 and 2. With the exception of the anteromedial lunge test for PFPS subjects, ICC [3, 1] for FPT measures exceeded .76. ICC [3, 3] for strength measures exceeded .85 for control subjects. PFPS had slightly lower ICCs for the hip muscles; however, they had excellent reproducibility for the knee extensors (ICC [3, 3] = .97). ICC [3, 5] for average EMG amplitudes exceeded .71 for all intervals of stair descent for controls, except for GM single leg stance, GM preswing, and VL preswing. PFPS subjects also had similar ICCs as controls except for VM loading.
response, VM single leg stance, VM preswing, and VL preswing. All groups had acceptable reliability for the EMG timing differences. ICC [3,5] for average kinematic data during the stance phase of stair descent for the control groups exceeded .70; however, PFPS subjects demonstrated acceptable reliability only for hip abduction and knee valgus angles.

**Discussion**

Measurement reliability is critical for data analysis. It insures that changes in a specific measure represent a true change in performance and not one attributable to chance alone (Loudon et al., 2002). Overall, results demonstrated that the evaluation tools used in this study provided reliable measures. Most ICCs exceeded .75, an acceptable level of reliability (Portney & Watkins, 2000a). The control group generally had higher ICCs than subjects with PFPS.

**Functional Performance Tests**

The step-down test provided similar measures of reliability for both groups (ICC [3, 1] = .76 and .78 for control and PFPS subjects, respectively). Although these coefficients were acceptable, they were much lower than the .94 coefficient reported by Loudon et al. (2002).

Results from the current study suggested less reproducibility of the step-down test. An identical step height was used in order to compare results to the Loudon study; however, some subjects appeared to have greater difficulty with this task. Difficulty could have resulted from variations in heel cord flexibility because adequate ankle dorsiflexion was necessary to perform this task. Therefore, greater tightness on a particular day could have adversely affected performance. Because heel cord flexibility was not measured, this influence could not be answered in the current study. Future investigators should employ a procedure to allow adequate heel cord stretching prior to testing.
Results for the anteromedial lunge test also differed from Loudon et al (2002). Their ICC [3, 1] was .82 whereas the current study ICCs were .89 and .33 for controls and PFPS subjects, respectively. For the current study, control subjects completed this test more consistently as evidenced by the higher ICC. However, this test did not provide a reliable measure for PFPS subjects as they showed significant improvements in performance on the second day of testing (See Table 2.7). PFPS subjects might have anticipated increased discomfort with the lunge activity during initial testing. However, they reported similar pain ratings (a rating ± .05 cm of the original score) prior to the second day of testing. Therefore, PFPS subjects might have performed repeat testing more aggressively due to a lower pain expectation, which would account for the lower ICC calculation. Based on this finding, caution should be taken when using the anteromedial lunge test.

Strength

Strength reliability for control subjects was good to excellent (ICC [3, 5] range from .85 to .97) for all muscles tested; however, hip musculature reliability in PFPS subjects was lower (ICC [3, 5] = .69 and .63 for the hip abductors and hip external rotators, respectively). For each test, the HHD was positioned and stabilized, and data collected, as recommended by previous researchers (Agre et al., 1987; Andrews et al., 1996; Kramer et al., 1991; Nadler et al., 2000). With respect to the hip musculature, these procedures resulted in much higher ICCs in controls compared to PFPS subjects. Although lower for PFPS subjects, the SEM associated with these hip measures was quite small. This finding suggested limited variability between measures (Bolgla & Keskula, 1997). Therefore, these methods may still provide reliable measures of hip strength.

For the knee extensor test, control subjects had an ICC of .89; PFPS subjects had an ICC of .97. The belt-resisted method was used to facilitate subject’s willingness to perform a maximum contraction (Kramer et al., 1991). Although every effort was made to provide the maximum resistance possible, variations could have occurred. As will be explained in Chapter 3, control subjects demonstrated significantly greater knee extensor strength than PFPS subjects. If the belt felt more stable on one day but not the other, then it might have affected the effort exerted by control subjects (Wikholm & Bohannon,
Alternatively, resistance applied for PFPS subjects might have been adequate enough so that they provided a consistent day-to-day effort.

**Surface EMG**

**Muscle activation amplitudes.** Researchers have quantified quadriceps EMG amplitudes during rehabilitation exercises (Gryzlo, Patek, Pink, & Perry, 1994; Selseth et al., 2000), level ambulation (Powers et al., 1996), and stair-stepping (Gilleard et al., 1998; Mohr et al., 2003). However, limited information has existed regarding measurement reliability. Some studies (Winter & Yack, 1987; Yang & Winter, 1984) have examined within day intrasubject and intersubject variability of lower extremity muscles during normal gait. Researchers calculated coefficients of variation for isometric and dynamic normalization methods and recommended dynamic normalization methods because of their lower variability. However, these studies were conducted to describe patterns of normal gait. The purpose of the current study was to identify EMG amplitude differences between groups of subjects.

Knutson et al. (1994) stated that lower coefficients of variation inferred group homogeneity. Many studies are conducted to determine differences in parameters between subjects. If researchers use tools that have limited variability (low intersubject variability), then they may not be able to identify differences between groups. Knutson et al. compared gastrocnemius activation differences in subjects with and without an anterior cruciate ligament - deficient knee using a MVIC and 2 dynamic normalization methods. Although dynamic methods had lower intrasubject and intersubject coefficients of variation, ICCs using the MVIC method were higher for both groups of subjects. These results supported the use of the MVIC method because it successfully discriminated between groups.

The current study normalized data based on a MVIC. ICCs for control subjects implied acceptable reliability for all stance intervals except the GM during single leg stance and preswing (ICC = .49 and .47, respectively) and the VL during preswing (ICC = .55). However, these intervals had low standard errors of measurement (SEM). The small SEM indicated high measurement precision and limited variability between measures, findings that support the reliability of these measures.
ICCs for PFPS subjects varied from controls for the GM and VM. Surprisingly, PFPS subjects had higher ICCs for the GM, which implied consistent activation for both testing days. As will be explained in Chapter 3, the GM can help stabilize knee frontal plane motion. This stabilizing effect might have required more consistent muscle activation and resulted in more reproducible measurements. Regarding VM activation, PFPS subjects had less knee extensor strength (See Chapter 3), and weakness could have affected their ability to provide a consistent effort.

**Muscle onset timing differences.** Cowan et al. (2000) are the only researchers who examined reliability for VM and VL timing differences using parameters identical to those in the current study. They reported ICC [3, 5] equal to .91 and .96 during the concentric and eccentric phases of stair stepping in normal subjects. ICCs for the current study (ICC [3, 5] = .89 for eccentric phase) inferred good to excellent reliability but were lower than those reported by Cowan et al. Since generalization of results would depend on study replication, additional investigations are needed to conclusively determine reliability for these timing differences.

The current study was the first to determine this method’s reliability specifically for subjects diagnosed with PFPS. This determination is important because it is unknown if these measures would be reproducible in a patient population. PFPS subjects demonstrated less reproducibility (ICC [3, 5] = .70). Although a higher ICC was desirable, this value may still be acceptable due to the inherent variability associated with EMG measures.

No study has determined the test-retest reliability of GM, VM, and VL timing differences. Although Brindle et al. (2003) highlighted relationships between hip and knee muscle timing onsets, they did not establish measurement reliability. Findings from this study revealed acceptable reliability (ICC [3, 5] ranges from .84 to .90), for both control and PFPS subjects, providing support for the use of these measures in further investigations.
Kinematics

Control subjects demonstrated acceptable test-retest reliability for all measures during the stance phase of stair descent. Frontal plane reliability was higher than transverse plane and consistent with normal gait (Kadaba et al., 1989). Although PFPS subjects had acceptable reliability for hip and knee frontal plane motion, hip transverse plane motion revealed moderate reproducibility (ICC = .55). Yu et al. (1997) stated that variance in kinematic data may result from variation in motor performance. Therefore, PFPS subjects might have used different movement patterns during repeat testing, and the resulting variability could have accounted for lower ICC values.

Another source of between day variability for both groups could have been marker misalignment (Kadaba et al., 1990). Kadaba et al. (1989) showed that marker misalignment can introduce a constant offset to some joint angle measurement patterns. For normal gait, they found that an offset had a greater affect on transverse and frontal planes of motion. Marker misalignment could have contributed to lower ICCs in the current study. Although every attempt was made to apply markers in a consistent manner, variations could have occurred.

Conclusion and Future Direction

Results from this study indicated acceptable reliability for most of the measures examined. These findings have important clinical implications because measurement reliability is paramount for evaluating changes in patient impairments throughout the rehabilitation process. The step-down test provided a more reliable measure of functional performance compared to the anteromedial lunge test. HHD proved to be a very reliable tool; however, the clinician must ensure proper application and stabilization.

Surface EMG provided reproducible data for activation amplitudes during loading response and single leg stance. It also demonstrated good reliability for determining timing differences. It should be noted that subjects completed the stair-stepping task at a standard rate (96 beats per minute); it is unknown if similar ICCs would be calculated using other cadences. Finally, motion analysis can provide important information regarding joint angles but a continuing problem exists when measuring frontal and transverse plane motion.
The purpose of this dissertation was to identify differences in hip and knee functional performance, strength, muscle activation amplitudes, muscle onsets, and kinematics between subjects diagnosed with and without PFPS. Prior to investigating these relationships, it was imperative to determine measurement reliability. Chapter 2 has supported the use of these tools for the current study.
Table 2.1
Summary of Kinematic Variables and Descriptions of Joint Motion

<table>
<thead>
<tr>
<th>Joint</th>
<th>Plane of Motion</th>
<th>Positive Value</th>
<th>Negative Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hip</td>
<td>Transverse</td>
<td>Internal Rotation</td>
<td>External Rotation</td>
</tr>
<tr>
<td>Hip</td>
<td>Frontal</td>
<td>Adduction</td>
<td>Abduction</td>
</tr>
<tr>
<td>Knee</td>
<td>Frontal</td>
<td>Adduction (Varus)</td>
<td>Abduction (Valgus)</td>
</tr>
</tbody>
</table>

Table 2.2
Summary of Between Day Intraclass Correlation Coefficients and Standard Errors of Measurement for Functional Performance Tests

<table>
<thead>
<tr>
<th>Functional Performance Test</th>
<th>Controls ICC</th>
<th>Controls SEM</th>
<th>PFPS ICC</th>
<th>PFPS SEM</th>
</tr>
</thead>
<tbody>
<tr>
<td>Step-down</td>
<td>.76</td>
<td>2</td>
<td>.78</td>
<td>2</td>
</tr>
<tr>
<td>Anteromedial Lunge</td>
<td>.89</td>
<td>1</td>
<td>.33</td>
<td>1</td>
</tr>
</tbody>
</table>

ICC = intraclass correlation coefficient model [3,1]
SEM = standard error of measure expressed as number of repetitions
PFPS = patellofemoral pain syndrome
Table 2.3
Summary of Between Day Intraclass Correlation Coefficients and Standard Errors of Measurement for Strength Measures

<table>
<thead>
<tr>
<th>Muscle</th>
<th>Controls ICC</th>
<th>Controls SEM</th>
<th>PFPS ICC</th>
<th>PFPS SEM</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hip Abductors</td>
<td>.97</td>
<td>.46</td>
<td>.69</td>
<td>.49</td>
</tr>
<tr>
<td>Hip External Rotators</td>
<td>.85</td>
<td>.31</td>
<td>.63</td>
<td>.32</td>
</tr>
<tr>
<td>Knee Extensors</td>
<td>.89</td>
<td>.64</td>
<td>.97</td>
<td>.40</td>
</tr>
</tbody>
</table>

ICC = intraclass correlation coefficient model [3,3]
SEM = standard error of measure expressed as strength values normalized to subject weight and height
PFPS = patellofemoral pain syndrome
Table 2.4
Summary of Between Day Intraclass Correlation Coefficients and Standard Errors of Measurement for Electromyographic Amplitude Measures

<table>
<thead>
<tr>
<th>Muscle and Phase</th>
<th>Controls ICC</th>
<th>Controls SEM</th>
<th>PFPS ICC</th>
<th>PFPS SEM</th>
</tr>
</thead>
<tbody>
<tr>
<td>GM Load</td>
<td>.71</td>
<td>3</td>
<td>.96</td>
<td>6</td>
</tr>
<tr>
<td>VM Load</td>
<td>.88</td>
<td>6</td>
<td>.66</td>
<td>21</td>
</tr>
<tr>
<td>VL Load</td>
<td>.93</td>
<td>5</td>
<td>.89</td>
<td>11</td>
</tr>
<tr>
<td>GM SLS</td>
<td>.49</td>
<td>3</td>
<td>.70</td>
<td>9</td>
</tr>
<tr>
<td>VM SLS</td>
<td>.85</td>
<td>9</td>
<td>.64</td>
<td>12</td>
</tr>
<tr>
<td>VL SLS</td>
<td>.93</td>
<td>5</td>
<td>.89</td>
<td>5</td>
</tr>
<tr>
<td>GM Preswing</td>
<td>.47</td>
<td>4</td>
<td>.87</td>
<td>2</td>
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<tr>
<td>VM Preswing</td>
<td>.84</td>
<td>8</td>
<td>.52</td>
<td>13</td>
</tr>
<tr>
<td>VL Preswing</td>
<td>.55</td>
<td>6</td>
<td>.50</td>
<td>11</td>
</tr>
</tbody>
</table>

ICC = intraclass correlation coefficient model [3,5]
SEM = standard error of measure expressed as a percent maximum voluntary isometric contraction
PFPS = patellofemoral pain syndrome
GM = gluteus medius
VM = vastus medialis
VL = vastus lateralis
Load = loading response
SLS = single leg stance
Table 2.5
Summary of Between Day Intraclass Correlation Coefficients and Standard Errors of Measurement for Electromyographic Onset Timing Differences

<table>
<thead>
<tr>
<th>Onset Difference</th>
<th>Controls</th>
<th>PFPS</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>ICC</td>
<td>SEM</td>
</tr>
<tr>
<td>VM – GM</td>
<td>.89</td>
<td>18</td>
</tr>
<tr>
<td>VL – GM</td>
<td>.90</td>
<td>16</td>
</tr>
<tr>
<td>VL – VM</td>
<td>.89</td>
<td>2</td>
</tr>
</tbody>
</table>

ICC = intraclass correlation coefficient model [3,5]
SEM = standard error of measure expressed in milliseconds
PFPS = patellofemoral pain syndrome
Table 2.6
Summary of Between Day Intraclass Correlation Coefficients and Standard Errors of Measurement for Kinematic Measures

<table>
<thead>
<tr>
<th></th>
<th>Controls</th>
<th></th>
<th></th>
<th>PFPS</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>ICC</td>
<td>SEM</td>
<td>ICC</td>
<td>SEM</td>
<td></td>
</tr>
<tr>
<td>Hip Transverse Plane</td>
<td>.75</td>
<td>4</td>
<td>.55</td>
<td>5</td>
<td></td>
</tr>
<tr>
<td>Hip Frontal Plane</td>
<td>.81</td>
<td>1</td>
<td>.74</td>
<td>1</td>
<td></td>
</tr>
<tr>
<td>Knee Frontal Plane</td>
<td>.88</td>
<td>4</td>
<td>.70</td>
<td>2</td>
<td></td>
</tr>
</tbody>
</table>

ICC = intraclass correlation coefficient model [3,5]
SEM = standard error of measure expressed in degrees of motion
PFPS = patellofemoral pain syndrome
Table 2.7
Means and Standard Deviations for Functional Performance Tests

<table>
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</tr>
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<td>Step-down</td>
<td>24.7</td>
<td>6.0</td>
<td>27.6</td>
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<tr>
<td>Anteromedial Lunge</td>
<td>12.1</td>
<td>1.9</td>
<td>12.7</td>
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<td></td>
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</tr>
<tr>
<td>Step-down</td>
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<td>22.7</td>
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<tr>
<td>Anteromedial Lunge</td>
<td>9.9</td>
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<td>11.7</td>
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* Expressed as number of repetitions
SD = standard deviation
Table 2.8
Means and Standard Deviations for Strength Measures

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<tr>
<td>HAD</td>
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<td>6.2</td>
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<tr>
<td>HER</td>
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<td>KE</td>
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<td>8.0</td>
<td>2.1</td>
<td>0.10</td>
<td></td>
</tr>
<tr>
<td><strong>Patellofemoral Pain Syndrome Subjects</strong></td>
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<td></td>
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</tr>
<tr>
<td>HAD</td>
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<td>0.77</td>
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<tr>
<td>HER</td>
<td>2.2</td>
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<td>2.4</td>
<td>0.6</td>
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<tr>
<td>KE</td>
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<td>2.1</td>
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<td></td>
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</table>

* Expressed as normalized strength value \{\% \text{ [body weight} \times \text{ height}] = \text{torque} \times \frac{100}{\text{[body weight (N) \times subject height (m)]}} \}\}

SD = standard deviation
HAD = hip abductors
HER = hip external rotators
KE = knee extensors
Table 2.9  
Means and Standard Deviations for Electromyographic Amplitude Measures for Control Subjects

<table>
<thead>
<tr>
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<th></th>
<th>Day 2</th>
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</tr>
</thead>
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<td>Mean*</td>
<td>SD</td>
<td>Mean*</td>
<td>SD</td>
<td>p-value</td>
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<tr>
<td>GM Load</td>
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<td>6.7</td>
<td>21.6</td>
<td>7.0</td>
<td>0.91</td>
</tr>
<tr>
<td>VM Load</td>
<td>36.6</td>
<td>15.0</td>
<td>37.2</td>
<td>23.4</td>
<td>0.89</td>
</tr>
<tr>
<td>VL Load</td>
<td>44.6</td>
<td>17.5</td>
<td>42.1</td>
<td>22.3</td>
<td>0.56</td>
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<td>GM SLS</td>
<td>9.9</td>
<td>5.6</td>
<td>9.7</td>
<td>3.5</td>
<td>0.95</td>
</tr>
<tr>
<td>VM SLS</td>
<td>31.9</td>
<td>20.0</td>
<td>37.1</td>
<td>25.1</td>
<td>0.43</td>
</tr>
<tr>
<td>VL SLS</td>
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<td>33.0</td>
<td>14.1</td>
<td>0.42</td>
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<tr>
<td>GM Preswing</td>
<td>6.9</td>
<td>5.0</td>
<td>4.9</td>
<td>2.0</td>
<td>0.27</td>
</tr>
<tr>
<td>VM Preswing</td>
<td>20.4</td>
<td>14.7</td>
<td>25.0</td>
<td>26.1</td>
<td>0.48</td>
</tr>
<tr>
<td>VL Preswing</td>
<td>23.4</td>
<td>8.0</td>
<td>17.9</td>
<td>9.0</td>
<td>0.16</td>
</tr>
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</table>

* Expressed as a percent maximum voluntary isometric contraction  
SD = standard deviation  
GM = gluteus medius  
VM = vastus medialis  
VL = vastus lateralis  
Load = loading response  
SLS = single leg stance
Table 2.10
Means and Standard Deviations for Electromyographic Amplitude Measures for Patellofemoral Pain Syndrome Subjects

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<th>Day 2</th>
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<th>p-value</th>
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<td>Mean*</td>
<td>SD</td>
<td>Mean*</td>
<td>SD</td>
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</tr>
<tr>
<td>GM Load</td>
<td>46.0</td>
<td>27.0</td>
<td>43.4</td>
<td>32.3</td>
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<tr>
<td>VM Load</td>
<td>64.0</td>
<td>33.6</td>
<td>71.0</td>
<td>36.7</td>
<td>0.63</td>
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<tr>
<td>VL Load</td>
<td>47.4</td>
<td>23.1</td>
<td>54.1</td>
<td>42.0</td>
<td>0.43</td>
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<tr>
<td>GM SLS</td>
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<td>19.3</td>
<td>24.7</td>
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<tr>
<td>VM SLS</td>
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<td>19.6</td>
<td>59.4</td>
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<td>VL SLS</td>
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<td>41.7</td>
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<td>0.89</td>
</tr>
<tr>
<td>GM Preswing</td>
<td>9.3</td>
<td>5.9</td>
<td>6.9</td>
<td>5.1</td>
<td>0.10</td>
</tr>
<tr>
<td>VM Preswing</td>
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<td>23.5</td>
<td>13.9</td>
<td>11.3</td>
<td>0.22</td>
</tr>
<tr>
<td>VL Preswing</td>
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<td>20.2</td>
<td>10.9</td>
<td>7.2</td>
<td>0.18</td>
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* Expressed as a percent maximum voluntary isometric contraction
SD = standard deviation
GM = gluteus medius
VM = vastus medialis
VL = vastus lateralis
Load = loading response
SLS = single leg stance
Table 2.11
Means and Standard Deviations for Electromyographic Onset Timing Differences

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<td>Mean§</td>
<td>SD</td>
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<td>p-value</td>
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<td></td>
<td></td>
</tr>
<tr>
<td>VM – GM*</td>
<td>-100.1</td>
<td>56.9</td>
<td>-90.0</td>
<td>49.6</td>
<td>0.43</td>
<td></td>
</tr>
<tr>
<td>VL – GM‡</td>
<td>-100.8</td>
<td>54.5</td>
<td>-88.8</td>
<td>49.5</td>
<td>0.32</td>
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<tr>
<td>VL – VM†</td>
<td>0.0</td>
<td>7.0</td>
<td>-1.6</td>
<td>5.2</td>
<td>0.27</td>
<td></td>
</tr>
<tr>
<td><strong>Patellofemoral Pain Syndrome Subjects</strong></td>
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<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>VM – GM</td>
<td>-60.9</td>
<td>28.7</td>
<td>-72.1</td>
<td>38.7</td>
<td>0.28</td>
<td></td>
</tr>
<tr>
<td>VL – GM</td>
<td>-56.4</td>
<td>32.4</td>
<td>-71.4</td>
<td>42.0</td>
<td>0.10</td>
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</tr>
<tr>
<td>VL – VM</td>
<td>-6.7</td>
<td>8.3</td>
<td>-4.4</td>
<td>4.4</td>
<td>0.38</td>
<td></td>
</tr>
</tbody>
</table>

§ Expressed in milliseconds
* A negative value represents a delay in gluteus medius (GM) activation relative to the vastus medialis (VM).
‡ A negative value represents a delay in GM activation relative to the vastus lateralis (VL).
† A negative value represents a delay in VM activation relative to the VL.
SD = standard deviation
Table 2.12
Means and Standard Deviations for Kinematic Measures

<table>
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<td>SD</td>
<td>Mean*</td>
<td>SD</td>
<td>p-value</td>
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</tr>
<tr>
<td><strong>Control Subjects</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Hip Internal/ (External) Rotation</td>
<td>1.7</td>
<td>12.0</td>
<td>1.3</td>
<td>6.1</td>
<td>0.90</td>
<td></td>
</tr>
<tr>
<td>Hip Adduction/ (Abduction)</td>
<td>3.2</td>
<td>4.6</td>
<td>2.7</td>
<td>2.8</td>
<td>0.68</td>
<td></td>
</tr>
<tr>
<td>Knee Varus/ (Valgus)</td>
<td>1.0</td>
<td>8.2</td>
<td>3.1</td>
<td>11.0</td>
<td>0.08</td>
<td></td>
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<tr>
<td><strong>Patellofemoral Pain Syndrome Subjects</strong></td>
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<td></td>
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<td></td>
</tr>
<tr>
<td>Hip Internal/ (External) Rotation</td>
<td>2.1</td>
<td>8.6</td>
<td>1.5</td>
<td>5.6</td>
<td>0.88</td>
<td></td>
</tr>
<tr>
<td>Hip Adduction/ (Abduction)</td>
<td>2.1</td>
<td>2.9</td>
<td>0.8</td>
<td>1.3</td>
<td>0.13</td>
<td></td>
</tr>
<tr>
<td>Knee Varus/ (Valgus)</td>
<td>4.9</td>
<td>6.4</td>
<td>4.2</td>
<td>3.4</td>
<td>0.73</td>
<td></td>
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</table>

* Expressed in degrees of motion
SD = standard deviation
CHAPTER THREE
Function, Strength, and Neuromuscular Activity in Subjects Diagnosed With and Without Patellofemoral Pain Syndrome

Historically, research on PFPS etiology has focused primarily on the knee joint. Researchers have shown that PFPS results from either quadriceps weakness (Doucette & Goble, 1992; Malone, Davies, & Walsh, 2002; Natri et al., 1998; Thomee et al., 1995) or delayed activation of the VM relative to the VL (Cowan, Bennell, Crossley et al., 2002; Fulkerson, 2002; Witvrouw, Sneyes, Lysens, Victor, & Bellemans, 1996). It has been hypothesized that quadriceps weakness, especially of the VMO, or delayed onsets can lead to abnormal patella tracking and irritation to the patellofemoral joint (Grabiner, Koh, & Draganich, 1994; Neptune, Wright, & van den Bogert, 2000; Powers, 1998). Based on this theory, quadriceps strengthening has been the gold standard intervention and its use has been supported by the literature (Arroll, Ellis-Pegler, Edwards, & Sutcliffe, 1997; Harrison, Sheppard, & McQuarrie, 1999; Roush et al., 2000; Witvrouw, Lysens, Bellemans, Peers et al., 2000). To date, the exact mechanism of how quadriceps strengthening can decrease patellofemoral joint pain has remained elusive (Grabiner et al., 1994; Powers, 1998).

More current research has implicated the hip musculature in PFPS etiology. Both hip weakness (Ireland et al., 2003; Niemuth et al., 2005) and altered hip-to-knee muscular activation patterns (Brindle et al., 2003) have been identified in subjects diagnosed with PFPS. More important, preliminary research has shown that people diagnosed with knee pain respond favorably to rehabilitation programs targeting the hip musculature (Fredericson et al., 2000; Mascal et al., 2003; Pettitt & Dolski, 2000). Although studies have shown that the hip may positively influence knee function, further studies are needed to firmly establish this relationship. Therefore, instead of looking solely at the knee, it has been suggested that researchers adopt a more novel approach for investigating PFPS etiology—examining the lower extremity kinetic chain in its entirety (Powers, 2003).

Strength, neuromuscular patterns, and kinematics contribute to human movement and deserve consideration when examining the lower extremity kinetic chain and PFPS etiology. As explained in Chapter 1, this dissertation will address functional
performance, strength and neuromuscular influences in this chapter and kinematics in Chapter 4. Chapter 5 will integrate findings from Chapters 3 and 4 and explain the interrelationships between these factors.

Review of the Related Literature

Functional Anatomy of the Hip Musculature

The gluteus medius (GM) originates from the iliac crest and inserts onto the lateral surface of the greater trochanter. Although the GM is commonly known as a strong hip abductor, it is functionally more important as a hip stabilizer (Gottschalk, Kourosh, & LeVeau, 1989). During a functional activity like level walking, the GM maintains a level pelvis during the single leg stance portion of gait (Neumann & Hase, 1994). However, GM weakness can cause an increase in: 1) hip adduction (Neumann, 2002a), 2) knee valgus (Simoneau, 2002), and 3) lateral patella compressive forces (Mizuno et al., 2001).

The hip external rotators (see Table 3.1) also play an intricate role for hip stabilization. Delp, Hess, Hungerford, & Jones (1999) evaluated 4 hemi-pelvic specimens to assess the internal and external rotation moment arms of the quadratus femoris, obturator internus, obturator externus, and piriformis. Although the piriformis had an internal moment arm at 90 degrees hip flexion, all others displayed an external moment arm independent of hip position. However, the piriformis had an external moment during lesser amounts of hip flexion (positions closer to hip extension). Many functional activities, like gait, are performed in positions of minimal hip flexion, and these findings highlight the stabilizing effects that the hip external rotators can provide.

Results from this study support the importance of the hip external rotators during functional activities. Fibers from these muscles are horizontally oriented and can influence the amount of pelvic-on-femoral rotation. For example, with the lower extremity firmly contacted on the ground (e.g. during single limb stance phase of stair descent), concentric action of the stance leg hip external rotators moves the pelvis and trunk posteriorly to the fixed femur (Neumann, 2002a). Such movement places the femur in an externally rotated position relative to the pelvis.

Eccentric action of the hip external rotators also influences pelvic-on-femoral rotation control. For example, the pelvis and femur internally rotate during the early
stance of the gait cycle. Throughout this interval, the hip external rotators eccentrically contract to control the amount of hip internal rotation (anteriorly-directed movement of the contralateral iliac crest). Clinically, Simoneau (2002) has stated that inadequate hip external rotator strength or control may lead to excessive hip internal rotation, a position that may increase patellofemoral joint contact pressures (Lee, Morris, & Csintalan, 2003; Mizuno et al., 2001).

**Hip Weakness and PFPS**

Clinicians have incorporated hip strengthening as a comprehensive part of a PFPS rehabilitation program because of its stabilizing effects on hip, and ultimately knee, position (Crossley et al., 2002; Fulkerson, 2002; Loudon, Gajewski, Goist-Foley, & Loudon, 2004; Mascal et al., 2003). Although clinicians believe that hip strength can improve PFPS impairments, few studies have quantified the extent of hip weakness specific to this patient population.

Ireland et al. (2003) were the only researchers to compare hip abductor and hip external rotator strength among females diagnosed with and without PFPS. Using HHD, they measured isometric hip abductor and external rotator strength. Their results showed that PFPS subjects demonstrated 26% less hip abductor strength and 36% less hip external rotation strength compared to controls. Although hip abductor weakness has been referenced more in the literature, hip external rotator weakness might have a greater association with PFPS.

Niemuth et al. (2005) recently examined hip muscle weakness and overuse injuries in a group of injured and uninjured male and female recreational runners. They measured isometric strength of the entire hip musculature using test positions described in Appendix E for hip abduction and hip external rotation. Although some injured subjects had pathology other than PFPS, all demonstrated significant hip abductor weakness. However, unlike Ireland et al. (2003), Niemuth et al. did not report significant hip external rotator weakness. A possible reason for this finding was that their subjects were of mixed gender and included other overuse injuries besides PFPS. It is not known if the results would have found hip external rotator weakness if only female subjects diagnosed with PFPS had been the focus.
Neuromuscular Factors and PFPS

**EMG activation amplitudes.** A limited number of researchers have examined quadriceps EMG activation amplitudes in subjects diagnosed with PFPS during functional activities. MacIntyre and Robertson (1992) investigated quadriceps amplitudes in female runners with and without PFPS but found no differences. They concluded that changes in running patterns between these subjects were undetectable by EMG changes. However, they normalized data based on the maximum amplitude per running cycle, a dynamic normalization method similar to that used in gait studies (Winter & Yack, 1987; Yang & Winter, 1984). As explained in Chapter 2, dynamic normalization methods reduce intersubject variability and may not identify true group differences. Knutson et al. (1994) have recommended normalizing data based on a percent maximum voluntary isometric contraction (% MVIC) to determine group differences.

Powers et al. (1996) compared mean VMO, vastus medialis longus, VL, vastus intermedius, and rectus femoris amplitudes during level ambulation, ramp ambulation, and stair-stepping in 29 PFPS and 10 control subjects. Unlike MacIntyre and Robertson (1992), they normalized all data to a % MVIC. PFPS subjects demonstrated decreased activity during level and ramp ambulation, a finding suggestive of a quadriceps avoidance pattern (Berchuck, Andriacchi, Bach, & Reider, 1990). Adoption of a quadriceps avoidance pattern would minimize knee joint reaction forces that occur during knee flexion and possibly reduce patellofemoral pain (Perry, 1992). However, Powers et al. did not find differences in amplitudes during stair ascent or descent. They concluded that stair-stepping required greater muscular demands that were unavoidable. In other words, PFPS subjects could not perform stair-stepping using a quadriceps avoidance pattern.

A possible limitation of Powers et al. (1996) was the manner in which EMG data were analyzed; they combined data from all muscles. However, PFPS has been characterized by weakness, or inhibition, of the VMO, and not necessarily other quadriceps muscles. It is not known if differences in VMO activity existed but went undetected during stair-stepping.
Sheehy et al. (1998) compared amplitudes by calculating a ratio for peak VMO and VL activity during stair-stepping. Instead of determining mean activity throughout the task, they calculated ratios for the concentric and eccentric phases. Similar to Powers et al. (1996), PFPS and controls subjects had similar ratios. Together, these studies suggested that PFPS subjects generated EMG amplitudes similar to asymptomatic subjects. However, further comparisons are not possible due to differences in data processing.

Mohr et al. (2003) conducted a similar stair-stepping study but analyzed data in a more detailed fashion. They collected EMG data for 13 subjects diagnosed with PFPS associated with patellar subluxation (11 females and 2 males) and 11 controls (3 females and 8 males) while ascending and descending stairs at self-selected pace. All data were expressed as a % MVIC and normalized to 100% of the gait cycle. Instead of calculating mean cycle amplitudes or VMO/VL peak amplitude ratios, they determined median amplitudes for each 2% interval of the entire stair-stepping cycle.

During stair descent, PFPS subjects had greater VMO and VL amplitudes during all phases of the gait cycle. They concluded that PFPS subjects required greater EMG activity to complete the task because of quadriceps weakness (Powers, 2000). However, they could not determine the presence or extent of quadriceps weakness, since strength was not assessed.

A possible limitation of Mohr et al. (2003) was the imbalance between male and female subjects in each group. Zeller, McCrory, Kibler, & Uhl (2003) examined EMG activation of the lower extremity muscles during a single-leg squat, a task with demands similar to stair descent. Gender differences were identified since females exhibited greater EMG amplitudes during the task. Sheehy et al. (1998) accounted for this possible confounding factor by using equal numbers of male and female subjects.

Mohr et al. (2003) compared amplitudes of symptomatic subjects (2 males and 11 females) to a group of primarily healthy controls (8 males and 3 females), which could have underestimated control subjects’ amplitudes due to the predominance of male subjects. Likewise, the experimental group data might also have been overestimated (due to a greater number of female subjects) as compared to controls for the same reason.
Emerging studies, especially for ACL injury, have highlighted neuromuscular differences among gender (Lephart, Ferris, Riemann, Myers, & Fu, 2002; Zeller et al., 2003).

This review of the literature indicates mixed findings with respect to greater amplitudes for PFPS subjects. These varied conclusions may be the result of a possible gender bias. Therefore, future studies should examine same gender differences as this approach might provide new insight regarding PFPS etiology (Souza & Gross, 1991).

**EMG onset timing differences.** Fulkerson (2002) has cited abnormal patella tracking due to an imbalance of VMO and VL muscle onsets as a common cause of PFPS pathology. It has been hypothesized that a delayed contraction of the VMO relative to the VL can cause lateral patella tracking and an increase in lateral patellofemoral joint compressive forces (Grabiner et al., 1994; Neptune et al., 2000). Many researchers have investigated temporal characteristics of the vasti muscles. As summarized below, some have identified timing differences whereas others have not.

Voight and Wieder (1991) examined reflex response times of the VMO and VL in subjects with and without PFPS. They chose this method because it eliminated a potential confounding factor from voluntary quadriceps control. Pilot data suggested that normal subjects activated the VMO prior to the VL during a patellar tendon tap. They hypothesized that PFPS subjects would have a delayed VMO response during a similar tendon tap. Findings from this study supported their initial hypothesis. PFPS subjects demonstrated faster VL response times than the VMO; control subjects exhibited faster VMO response times than the VL. They concluded that delayed VMO activation could contribute to increased lateral patella tracking and patellofemoral joint irritation.

Witvrouw et al. (1996) conducted an identical study and found similar results as Voight and Wieder (1991). Although altered response times differentiated between subjects with and without PFPS, the clinical relevance of such differences was not known. To address clinical relevance, Witvrouw et al. (2003) determined reflex response times in PFPS subjects prior to beginning a quadriceps strengthening program, at the end of the 5-week program, and 3-months following the end of the program. Although subjects demonstrated significant functional improvements, they continued to have a similar pattern of delayed VMO response times following a patellar tendon tap.
A potential limitation of these studies may have been the assessment of afferent pathways. People diagnosed with PFPS typically complain of pain during dynamic, volitional activities. According to Karst and Willet (1995), it is not known if the quadriceps muscles are activated in a similar manner during functional activities. They investigated response times by replicating the Voight study and determining VL-VMO onset differences during voluntary non-weight bearing and weight bearing knee extension activity.

VL-VMO response time differences following a patellar tendon tap were similar for PFPS (0.01 ± 0.44 ms) and control (-0.19 ± 0.52 ms) subjects. Likewise, both groups had similar VL-VMO onset timing differences (all differences less than 4 ms) during non-weight bearing and weight bearing voluntary knee extension exercise. Karst and Willet also conducted a correlation analysis and did not find any associations between relative timing of VMO and VL onsets during reflex and voluntary activities. This lack of association suggested that reflex testing was not a good indicator of muscle activity onsets during voluntary knee extension. Therefore, activation timing during reflex and voluntary contractions may not be related in a functionally intuitive manner (Owings & Grabiner, 2002).

Powers et al. (1996), Sheehy et al. (1998), and Brindle et al. (2003) investigated onset timing differences during more functional activities, such as ambulation and stair-stepping. Like Karst & Willet (1995), neither reported significant VL-VMO timing differences in subjects diagnosed with PFPS. Although these studies reached similar conclusions, it has been difficult to compare results because of differences in data collection, EMG signal processing, and muscle onset identification.

In order to make meaningful comparisons among studies, researchers should employ a standard methodology for collecting, processing, and analyzing data. Hodges and Bui (1996) have recommended use of computer algorithms, in combination with close visual inspection, to objectify muscle onset identification. Cowan et al. (2000) tested different computer algorithms; each developed to identify quadriceps onsets during a stair-stepping task for the PFPS population. They defined a muscle onset as the point in which the signal deviated by more than 3 standard deviations, for a minimum of 25 ms, over the baseline level taken 200 ms before the trial began. Cowan et al. recommended
this algorithm because of its test-retest reliability ICC [3, 5] of .91 for the concentric phase and .96 for the eccentric phase of stair-stepping.

Using this algorithm, Cowan et al. (2001) compared VL-VMO onsets in PFPS and control subjects during stair-stepping. They found that asymptomatic subjects had nearly synchronous VL and VMO activation during stair ascent and descent. However, they reported delayed VMO activation (15.80 ms and 19.39 ms during the concentric and eccentric phases, respectively) in PFPS subjects. Cowan et al. inferred that such delays could result in excessive lateral patella tracking and increased patellofemoral joint loading. Neptune et al. (2000) reported that a delay as small as 5-ms could cause patellofemoral joint irritation based on a musculoskeletal model and simulation of running. It is not conclusively known from either study if a 5-ms or greater delay in VMO activation is clinically relevant.

Cowan et al. (2002) conducted a study in which PFPS subjects were randomly assigned either into a rehabilitation group or a placebo group. The rehabilitation group participated in a 6-week McConnell-based program that included functional VMO training, hamstring stretching, patella taping, and gluteus medius strengthening exercise. The placebo group received placebo taping, inoperative ultrasound, and light application of a nontherapeutic gel. Researchers assessed VL-VMO timing differences during stair-stepping as described above. Both groups had similar VMO delayed activation at the beginning of the study. At the end of the study, subjects in the rehabilitation group reported significant improvements in pain. VMO onset preceded the VL during the concentric phase and occurred at the same time during the eccentric phase of stair-stepping.

The authors concluded that the rehabilitation program enhanced VMO activation and contributed to functional improvement. It is not known if quadriceps strength improvements that might have contributed more to pain reduction, since this parameter was not measured. It should also be noted that Cowan et al. (2002) reported significant VL-VMO timing differences (using a specific stair-stepping protocol) during voluntary activities whereas others have not. Therefore, study replication from outside laboratories is needed to support their findings.
More recent research has focused on the influence of the hip musculature on knee function. Brindle et al. (2003) were among the first researchers to determine differences in GM activation to that of the VMO and VL during stair-stepping. They determined GM, VM, and VL onsets during a stair stepping task in PFPS and control subjects. In agreement with other studies, they reported simultaneous VMO and VL activation. The PFPS and control groups both had delayed GM activation in relation to the VMO (52 ms and 33 ms GM delay in PFPS and controls, respectively) and the VL (112 ms and 61 ms GM delay in PFPS and controls, respectively) during stair descent. Delays for PFPS subjects were significantly different from controls. The authors concluded that altered GM activity could affect movement of the rest of the lower extremity and that additional studies were needed to understand the role of the gluteus medius and PFPS etiology.

The above literature review suggests that little is definitively known regarding function, strength, and neuromuscular patterns in subjects diagnosed with PFPS. Discrepancies between studies have resulted from differing methodologies, various EMG processing methods, and mixed gender subject groups. Studies examining ACL injury have identified gender differences for strength and neuromuscular factors (Huston & Wojtys, 1996; Lephart et al., 2002; Myer, Ford, & Hewett, 2005; Zeller et al., 2003). Therefore, future studies should: 1) include subjects of the same gender, 2) employ reliable testing procedures, and 3) process and analyze data in ways that permit comparison to other studies.

**Purpose and Research Hypotheses**

The purpose of this study was to identify differences in function, strength, and neuromuscular patterns for females diagnosed with and without PFPS. Since recent studies have suggested gender differences associated with strength and neuromuscular activation patterns, only female subjects were included (Huston & Wojtys, 1996; Lephart et al., 2002; Myer et al., 2005; Zeller et al., 2003). It was hypothesized that female subjects diagnosed with PFPS would demonstrate the following:

1. Subjects diagnosed with PFPS would have an average limb symmetry index (LSI) less than 90%; control subjects would have average LSI greater than 95% for the step-down and anteromedial lunge functional performance tests.
2. Subjects with PFPS would demonstrate significantly less hip abductor, hip external rotator, and knee extensor strength than control subjects.

3. Subjects with PFPS would exhibit greater EMG amplitudes of the GM, VM, and VL during stair descent than control subjects.

4. Subjects with PFPS would demonstrate simultaneous activation of the VM and VL at the onset of stair descent.

5. Subjects with PFPS would demonstrate a greater delay in GM activation compared to the VM and VL than control subjects.

**Methodology**

**Subjects**

Eighteen females diagnosed with PFPS (age = 24.5 ± 3.2 years, height = 1.68 ± 0.1 m, body mass = 618.0 ± 89.3 N, pain = 4.4 ± 1.5 cm, duration of symptoms = 14.4 ± 12.8 months) and 18 asymptomatic females (age = 23.9 ± 2.8 years, height = 1.67 ± 0.1 m, body mass = 608.2 ± 83.4 N) participated in this study. All subjects met the inclusion criteria, as summarized in Chapter 1, and signed an informed consent approved by the University of Kentucky Institutional Review Board prior to participation.

**Procedures**

First, subjects completed a 10-cm visual analog scale reflecting usual pain during the past week (Crossley, Bennell et al., 2004). Next, they rode a stationary bike for 3 minutes in a pain-free range of motion at a submaximal speed and practiced each functional performance test 3 to 5 times (Loudon et al., 2002). Subjects then performed the step-down and anteromedial functional performance tests, as described in Appendix E, for each lower extremity in a random order to reduce ordering bias. The number of repetitions performed within a 30-second period for each lower extremity was documented.

Next, the distances from the greater trochanter to the lateral femoral condyle and the distance from the lateral knee joint line to the lateral malleolus were measured. These measurements were completed to establish the perpendicular distance from the HHD and the hip and knee joints, respectively. This information was used to report all strength values as measures of torque in units of newton*meters (N*m).
Subjects’ skin was prepared for EMG instrumentation by shaving, abrading, and cleansing with isopropyl alcohol prior to application of surface electrodes. Bi-polar Ag-AgCl surface electrodes (Medicotest, Rolling Meadows, IL), measuring 5 mm in diameter with an interelectrode distance of approximately 20 mm, were placed in parallel arrangement over the muscle bellies of the GM, VM, and VL. The GM electrode was placed 1/3rd the distance between the iliac crest and greater trochanter (Cram & Kasman, 1998). The VM electrode was placed approximately 4 cm superior to and 3 cm medial to the superomedial border of the patella and oriented 55° to the vertical (Cowan et al., 2000). The VL electrode was placed 5 to 7 cm superior to and 6 to 8 cm lateral to the superolateral border of the patella and oriented 15° to the vertical (Cram & Kasman, 1998). Electrodes were further secured to the skin with an adhesive tape to prevent slippage during testing. A ground electrode was placed on the ipsilateral clavicle. Electrode placements were visually confirmed on an oscilloscope using manual muscle testing techniques. A 3-second standing “quiet” file was also recorded to exclude ambient noise.

Following EMG placement, strength measures were taken for the hip abductors, hip external rotators, and knee extensors. Subjects were positioned as described in Appendix E. For testing, subjects produced a maximum voluntary isometric contraction (MVIC) using the “make” test (Andrews et al., 1996; Bohannon, 1997) to the beat of a metronome set at 60 beats per minute. They generated maximum force over a 2-second period and maintained this force for an additional 5 seconds to the beat of the metronome. Subjects performed one practice (Andrews et al., 1996; Bohannon, 1997) and 3 test trials, with a 30-second rest period between trials. A coefficient of variation was calculated and an additional trial was taken, if necessary, to ensure that subjects had 3 measures with variability less than 10% (Agre et al., 1987). The order of muscle testing was counterbalanced to account for any potential bias. All measures were recorded in newtons (N) of force. EMG activity was simultaneously collected for the GM, VM, and VL during strength testing to determine a MVIC for each muscle.

Next, retroreflective markers, with a diameter of 20 mm, were placed on subjects using a standard Cleveland Clinic marker setup. This allowed use of video data to demarcate the start and end of stair descent. Subjects were then shown the stair stepping
task and allowed 5 practice trials. They were instructed to ascend and descend two 20-
cm high steps, ensuring that the test extremity lifted and lowered the body on the first and
third steps, respectively. Subjects also took a minimum of 3 strides prior to and
immediately following stair stepping in order to maintain a continuous movement pattern.
Because movement velocity may influence EMG activity, subjects performed the task at
a standardized rate of 96 beats per minute (Cowan et al., 2000; Gilleast et al., 1998).

After demonstrating proficiency with the test, subjects performed 10 test trials.
During this time, EMG data were sampled at 960 Hz and recorded synchronously with
the video data, which were sampled at 60 Hz. Data from the last 5 trials were analyzed
because of potential learning effects that might have been associated with earlier trials,
even with subjects having performed 5 practice trials. Refer to Appendix C for unit
specifications of the EMG and motion analysis equipment used in this study.

Data Processing

Functional performance tests. For each FPT, the total number of repetitions
completed by subjects on the involved (PFPS) or the right lower extremity (controls) was
recorded. Data were normalized by calculating a limb symmetry index ([number of
repetitions completed by the test lower extremity/ number of repetitions completed by the
contralateral lower extremity] * 100%). The resulting values were used for statistical
analysis.

Strength. Strength was expressed in units of torque by multiplying the force recorded
on the HHD by the perpendicular distance from the HHD to the joint center of rotation.
Average torque was then normalized to subject height and weight (% [body weight (N) *
height (m)] = torque * {100/[body weight (N) * subject height (m)]}) to allow for
comparison among subjects (Fredericson et al., 2000). These values were used for
statistical analysis.

EMG data. Raw EMG signals were processed in the manner as described in
Appendix D. To determine muscle activation amplitudes, EMG data from the last 5 trials
were root mean square (RMS) smoothed using a 55 ms time constant and normalized to
100% of the stair descent cycle. They were then ensemble averaged and expressed as a
% MVIC. Datapac software (Run Technologies, Mission Viejo, CA) then calculated the
average % MVIC EMG amplitude for each muscle during the: 1) loading response, 2)
single leg stance, and 3) preswing intervals of stair descent (see Appendix G). The resulting values were used for statistical analysis.

Muscle activation onsets were determined at the beginning of stair descent. After processing EMG signals and identifying muscle onsets (see Appendix D), Datapac software calculated timing differences. It subtracted the GM onset from the VM onset and VL onset, respectively, to quantify timing differences between the hip and knee musculature. A negative difference signified a delay in GM activation relative to the VM and VL where as a positive difference meant GM preactivation. The software also subtracted the VM onset from the VL onset to quantify quadriceps timing differences. A negative difference meant a delay in VM activation relative to the VL where as a positive difference signified VM preactivation. The average from 5 trials was used for statistical analysis (Cowan et al., 2001).

Statistical Analysis

Independent \( t \)-tests were used to determine group differences in age, height, and weight. A 2 X 2 (group X functional performance test) analysis of variance (ANOVA) for repeated measures on FPT was used to determine differences in LSI for the step-down and anteromedial lunge tests. A 2 X 3 (group X muscle) ANOVA for repeated measures on muscle was used to determined differences in strength. Separate 2 X 3 (group X interval) ANOVAs for repeated measures on stance interval were used to identify EMG amplitude differences for the GM, VM, and VL, respectively. A 2 X 3 (group X timing difference) ANOVA for repeated measures on muscle was used to determine EMG onset timing differences. An independent 1-group \( t \)-test was conducted to determine if timing differences varied significantly from 0 (meaning simultaneous VM and VL activation) for the PFPS and control groups (Cowan et al., 2001). All statistical analyses were performed using SPSS version 12.0 (SPSS, Inc., Chicago, IL). Level of significance was established at the 0.05 level; the sequentially rejective Bonferroni (Bonferroni-Holm) post hoc test (Holm, 1979) was used to determine the significance of interactions for the two-factor ANOVAs.
Results

Independent *t*-tests for subject demographics revealed similar age, height, and weight (*p* > .44) characteristics for both groups. Results from separate ANOVAs showed a significant main effect for group for the functional performance tests (*p* < .002) and strength measures (*p* < .006). PFPS subjects had significantly lower LSI scores (Figure 3.1) and produced less torque during strength testing (Figure 3.2).

For the GM and VM, a group X interval interaction effect existed for EMG amplitudes. PFPS subjects generated higher EMG amplitudes during the loading response but had similar amplitudes during single leg stance and preswing. For the VL, subjects generated similar EMG amplitudes throughout the entire stance phase (*p* > .066). Figures 3.3 through 3.5 summarize this data.

No differences were identified with respect to EMG timing parameters (*p* > .55). Results from independent 1-group *t*-test to determine if VL - VM onsets differed significantly from 0 were not significant (meaning both groups had simultaneous VM and VL activation). Table 3.2 summarizes descriptive data for the EMG timing differences.

Discussion

Historically, researchers have examined knee function and its influence on PFPS etiology. Recently, attention has focused on the hip and results from more current studies have shown an association between hip weakness and PFPS etiology (Ireland et al., 2003; Powers et al., 2003). Although researchers have concluded that the hip might have influenced knee function, they did not concurrently examine the hip and knee. In contrast, the current study simultaneously examined hip and knee function, strength, EMG amplitudes, and EMG timing differences between subjects diagnosed with and without PFPS.

Functional Performance Tests

Clinicians typically assess function using functional performance tests (FPT) designed to simulate the stresses about the knee encountered during athletic activities (Lephart, Perrin, Fu, & Minger, 1991). Such activities include running, jumping, and cutting for people diagnosed with ACL injury (Barber et al., 1992; Lephart et al., 1992). However, patients diagnosed with PFPS typically complain of pain and dysfunction during activities like squatting and kneeling. Loudon et al. (2002) developed five FPTs
that simulated loading on a flexed knee and established their measurement reliability. For the current study, only the step-down and anteromedial lunge tests were used since they had the highest reliability and greatest correlation with pain.

In the Loudon et al. (2002) study, PFPS subjects had LSI of 80.0% and 85.9% for the step-down and anteromedial lunge tests, respectively, while LSI for control subjects exceeded 95.1%. Control subjects in the current study had similar LSI for both tests; PFPS subjects had higher LSI than subjects in the Loudon study for both tests. A primary reason for the discrepancy between the step-down test may have been the manner of administration. As discussed in Chapter 2, subjects had greater difficulty performing the test, regardless of group membership. Therefore, instead of touching the bottom of the contralateral heel to the ground (as described by Loudon et al.), subjects in the current study brushed any portion of that foot. PFPS subjects in the Loudon et al. study might have had greater difficulty performing the test, which could have accounted for their lower reported LSI.

Subjects in the current study performed the anteromedial lunge test as described by Loudon et al. (2002). Control subjects demonstrated LSI very similar to those reported by Loudon et al. (97% for both studies). However, PFPS subjects for this study had LSI of 92%, which was higher than that reported by the Loudon et al. (85.9%). A possible reason for the differences could have been the subject sample used for each study. Subjects in the current study had more chronic symptoms and reported average pain at a 4.4 on a 10-cm VAS. Although subjects in the Loudon study had a 5.5 month average duration of symptoms, they did not report average pain. Therefore, they might have had higher pain ratings that led to poorer performance, as compared to the contralateral extremity. Greater pain to the test extremity could have accounted for a lower reported LSI.

**Hip and Knee Strength**

**Hip abductor and hip external rotators.** Fredericson et al. (2000) were among the first researchers to report an association between hip weakness and knee overuse injury. Although subjects in their study had iliotibial band syndrome (ITBS), it was thought that hip weakness contributed to the knee pathology. As explained above, Fredericson et al.
normalized torque to subject height and weight. The same method was used to allow comparisons between studies.

Fredericson et al. (2000) reported average hip abductor torque of 7.82 and 10.19 for female ITBS and control subjects, respectively, which were higher than the current findings (4.78 for PFPS and 6.45 for controls). This discrepancy might have been attributable to differences in the subject sample. Subjects in the Fredericson et al. study were recreational runners who most likely demonstrated greater fitness levels (as reflected by greater strength measures) than subjects in the current study. However, percent differences between groups for each study were similar. ITBS subjects in the Fredericson et al. study demonstrated a 23% strength deficit and PFPS subjects in the current study had a 26% strength deficit compared to controls.

Ireland et al. (2003) measured the force applied to a HHD during hip abduction in the same manner as this study. However, they did not express measures as a unit of torque by multiplying the force applied to the HHD by the perpendicular distance from the hip joint. They did normalize force values to subject body weight and found that PFPS subjects applied 26% less force to the HHD than control subjects.

The fact that force measurements in the current study were taken in a manner similar to Ireland et al. (2003) enabled comparison of results. To enable this comparison, the force (N) recorded on the HHD by subjects in the current study was expressed as a percent body weight (% BW). Under this method, hip abductor force for PFPS subjects was 22.5 ± 5.9 % BW, which agreed with the results reported by Ireland et al. (23.3 ± 6.9 % BW). Although force does not accurately represent strength (torque), the fact that PFPS subjects in both studies had significantly lower hip abductor force values indicated hip weakness for this patient population.

Ireland et al. (2003) also examined hip external rotator function and reported that PFPS subjects applied 36% less force on the HHD than controls. For the current study, PFPS subjects demonstrated 27% less hip external rotator strength than controls. However, PFPS force values expressed as a % BW were similar (like the hip abductors, both studies recorded the force applied to the HHD during hip external rotation in a similar manner). PFPS subjects in the current study had values of 11.1 ± 3.1 % BW whereas PFPS subjects in the Ireland study had values of 10.8 ± 4.0 % BW. These
findings indicated that variations in percent differences between groups were attributable to control subject differences.

**Knee extensors.** Results from this study supported previous works showing a relationship between quadriceps weakness and PFPS (Grabiner et al., 1994; Malone et al., 2002; Powers et al., 1997; Stiene, Brosky, Reinking, Nyland, & Mason, 1996; Thomee et al., 1995). PFPS subjects demonstrated 20% less quadriceps strength compared to control subjects. These findings are clinically relevant because PFPS patients have responded favorably to quadriceps strengthening programs (Malone et al., 2002; Natri et al., 1998).

In summary, although PFPS subjects demonstrated quadriceps weakness, they demonstrated even higher hip abductor and external rotator strength deficits. Findings from this study could not determine if hip weakness contributed to or resulted from PFPS. Therefore, additional prospective studies should address this question.

**EMG Activation Amplitudes**

Subjects diagnosed with PFPS demonstrated significantly higher EMG amplitudes for the VM during the loading response with a trend toward significance ($p = 0.018$) during the single leg stance interval of stair descent. These findings are in partial agreement with those reported previously for subjects diagnosed with lateral patella instability (Mohr et al., 2003; Powers, 2000). Mohr et al. (2003) had subjects descend stairs at a self-selected pace and data were analyzed at 2% intervals of the entire stair descent cycle. Data were expressed as a % MVIC and analyzed on a natural log-transformed scale, a transformation that approximated medians of the data. Data in the current study were also expressed as a % MVIC but analyzed based on the average values for loading response, single leg stance, and preswing. Overall, values in the current study exceeded those reported by Mohr et al.; however, patterns of EMG activity between groups were very similar.

Mohr et al. (2003) concluded that quadriceps weakness contributed to the greater EMG activity required for stair descent even though they did not measure strength. The current study differed because it examined EMG activity and strength concurrently. PFPS subjects in the current study exhibited quadriceps weakness and showed an inverse relationship between strength and muscle activation.
Results from the current study also differed from Mohr et al. (2003) because PFPS and control subjects had similar VM amplitudes during preswing. Sheehy et al. (1998) identified 2 peaks of eccentric EMG activity for the VM and VL during stair descent. The first corresponded with weight acceptance (loading response). During this interval, researchers have reported greater hip muscle activation in response to decelerating and controlling forward and downward motion of the body onto the step (Lyons, Perry, & Gronley, 1983; McFadyen & Winter, 1988). Higher VM amplitudes for PFPS subjects during loading response most likely reflected the need for greater activation when knee flexion moments are greater (Kadaba et al., 1989). Sheehy et al. referred to the second peak of activity as body lowering, which corresponded to preswing in the current study (movement of the center of mass past the stance leg). During this interval, the body was likely positioned with the center of mass located more centrally over the foot, which would provide a stable base and require less muscle activation (McFadyen & Winter, 1988).

Results for the VL also differed from Mohr et al. (2003). No VL differences were identified during stair descent and agreed with previous works (MacIntyre & Robertson, 1992; Powers et al., 1996; Sheehy et al., 1998). The current study also found differences between VM and VL amplitudes, which could possibly imply VM insufficiency relative to the VL. Souza and Gross (1991) found relative differences in VMO and VL activity for PFPS subjects during stair-stepping. Unlike the current study, they reported decreased (not increased) VMO activity relative to the VL. It is unclear why Souza and Gross found less VM activity compared to the VL. However, they did not normalize the EMG data and had a smaller sample size compared to the current study. These methodological differences might account for the conflicting findings (Powers et al., 1996).

Researchers have reported greater GM activation during the loading response of stair descent in asymptomatic subjects (Lyons et al., 1983; McFadyen & Winter, 1988). Data from the current study supported these findings as all subjects had greater GM activation for the loading response relative to single leg stance and preswing. Moreover, PFPS subjects had significantly higher GM amplitudes and less hip strength compared to controls. As discussed above, GM weakness would reflect the need for greater EMG
activity. To date, no other study has examined GM activation in PFPS during stair descent and precluded comparison with this study. Further studies are needed to determine the importance of the hip musculature during stair descent.

**EMG Onset Timing Differences**

**VM – VL onset timing differences.** Results from this study showed simultaneous activation of the VM and VL at the onset of stair descent, which were in agreement with previous reports (Brindle et al., 2003; Karst & Willett, 1995; Powers et al., 1996; Sheehy et al., 1998). However, findings from this study contradicted those reported by Cowan et al. (2001), who reported a 19.39 ms delay in VMO activation during stair descent. Although the current study determined onsets identical to the Cowan et al. study, variations in the sample population might have contributed to differing results. PFPS subjects in the current study were younger (age = 24.5 ± 3.2 yr vs. 27.0 ± 8.1 yr) in comparison to their study. Cowan et al. included subjects who reported pain at a minimum of 3 on a 10-cm visual analog scale but did not report average pain ratings. They also did not report the duration of symptoms for the PFPS subjects. For the current study, PFPS subjects reported pain of 4.4 ± 1.5 cm and had more chronic symptoms (duration = 14.4 ± 12.8 mos). It is not known if subjects with more acute symptoms would demonstrate different neuromuscular patterns.

**GM and vastii muscle onset timing differences.** All subjects in the current study demonstrated delayed GM activation relative to the VM and VL; however, there were no significant between group differences. Brindle et al. (2003) examined hip and knee temporal characteristics. Control subjects in their study had a GM delay of 33 ms and 52 ms for the VM and VL; PFPS subjects had a GM delay of 61 ms and 112 ms for the VM and VL. Based on these significant differences, Brindle et al. concluded that changes in GM neuromuscular activity patterns may contribute to PFPS pathology.

Subjects in the current study had delayed GM activation of approximately 80 ms for both the VM and VL. These findings contradicted those from Brindle et al. (2003) because of greater GM delay and between group similarities. The current study also collected and analyzed data in a different manner from Brindle et al. As stated above, variations in methodology and signal processing compromise study comparisons.
Therefore, additional studies are needed to better understand timing characteristics between the GM, VM, and VL.

Conclusion and Future Direction

The purpose of this study was to determine differences in hip and knee function, strength, and neuromuscular patterns in females diagnosed with and without PFPS. PFPS subjects demonstrated lower LSI for functional performance tests. Overall, PFPS subjects had less hip and knee strength than controls. Although previous works have reported quadriceps weakness as a prime etiological factor, the results of this study suggested that PFPS subjects had greater percent differences in hip strength. Additionally, this study demonstrated that hip external rotator strength may play a more significant role in PFPS than originally thought. However, caution should be taken in interpreting this finding, since it is not known if hip weakness was the cause of or a result of PFPS. Finally, EMG amplitudes showed that PFPS required greater muscle activity, possibly resulting from hip and knee weakness. No timing differences existed for the GM and vastii muscles.

These results support a relationship between the hip musculature and PFPS. Researchers have stated that hip weakness can affect lower extremity kinematics and hypothesize that excessive femoral adduction and internal rotation can adversely affect knee function (Mascal et al., 2003; Powers et al., 2003; Simoneau, 2002). In order to determine this relationship, Chapter 4 examined hip and knee kinematics during the stair stepping task.
<table>
<thead>
<tr>
<th>Muscle</th>
<th>Origin</th>
<th>Insertion</th>
</tr>
</thead>
<tbody>
<tr>
<td>Piriformis</td>
<td>Pelvic surface of the sacrum</td>
<td>Greater trochanter</td>
</tr>
<tr>
<td>Quadratus femoris</td>
<td>Proximal part of the ischial tuberosity</td>
<td>Femoral intertrochanteric crest</td>
</tr>
<tr>
<td>Obturator internus</td>
<td>Pelvic surface of obturator membrane and margin of obturator foramen</td>
<td>Greater trochanter</td>
</tr>
<tr>
<td>Obturator externus</td>
<td>Pubic and ischial rami</td>
<td>Femoral trochanteric fossa</td>
</tr>
<tr>
<td>Gemellus superior</td>
<td>Ischial spine</td>
<td>Greater trochanter</td>
</tr>
<tr>
<td>Gemellus internus</td>
<td>Ischial tuberosity</td>
<td>Greater trochanter</td>
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Table 3.2  
Comparison of Means δ (± Standard Deviation) for Electromyographic Onset Timing Differences

<table>
<thead>
<tr>
<th></th>
<th>Controls</th>
<th>PFPS</th>
</tr>
</thead>
<tbody>
<tr>
<td>VM – GM</td>
<td>-79 ± 64§</td>
<td>-73 ± 65§</td>
</tr>
<tr>
<td>VL - GM</td>
<td>-83 ± 62§</td>
<td>-75 ± 67§</td>
</tr>
<tr>
<td>VL – VM</td>
<td>-1.28 ± 8*</td>
<td>-3.83 ± 9†</td>
</tr>
</tbody>
</table>

PFPS = patellofemoral pain syndrome
GM = gluteus medius
VM = vastus medialis
VL = vastus lateralis
δ Expressed in milliseconds
§ p > .55
* Not significantly different from 0 (p = .073)
† Not significantly different from 0 (p = .530)
Figure 3.1

LSI = limb symmetry index
PFPS = patellofemoral pain syndrome
Significant overall main effect ($p < .002$)
Figure 3.2
Descriptive Statistics for Strength Measures*.

* Expressed as a unit of strength normalized to subject weight and height
PFPS = patellofemoral pain syndrome
Significant overall main effect ($p < .006$)
Figure 3.3
Comparison of Electromyographic Amplitudes for the Gluteus Medius.

% MVIC = percent maximum voluntary isometric contraction
GM = gluteus medius
Load = loading response
SLS = single leg stance
PFPS = patellofemoral pain syndrome

* $p = .003$ (PFPS significantly different from controls)
† $p = .049$ (PFPS not significantly different from controls)
‡ $p = .602$ (PFPS not significantly different from controls)
Figure 3.4
Comparison of Electromyographic Amplitudes for the Vastus Medius.

% MVIC = percent maximum voluntary isometric contraction
VM = vastus medius
Load = loading response
SLS = single leg stance
PFPS = patellofemoral pain syndrome
* $p = .001$ (PFPS significantly different from controls)
† $p = .018$ (PFPS not significantly different from controls)
‡ $p = .413$ (PFPS not significantly different from controls)
Figure 3.5
Comparison of Electromyographic Amplitudes for the Vastus Lateralis.

% MVIC = percent maximum voluntary isometric contraction
VL = vastus lateralis
Load = loading response
SLS = single leg stance
PFPS = patellofemoral pain syndrome
* $p = .066$
† $p = .215$
‡ $p = .837$
CHAPTER FOUR

Lower Extremity Kinematics and Patellofemoral Pain Syndrome

PFPS has been one of the most common, but challenging, orthopaedic problems that clinicians face because of a lack of consensus regarding its true etiology (Witvrouw et al., 2005). Possible etiologic factors have included quadriceps weakness, delayed VMO activation, tight lateral retinacular tissues, trauma, overuse, patellar instability, osteochondritis dissecans, neurologic disorders, and biomechanical dysfunction (Brody & Thein, 1998; Wilk et al., 1998). Regarding biomechanical dysfunction, much attention has focused on the quadriceps angle (Q angle).

The Q angle (the angle formed by drawing a line from the anterior superior iliac spine to the midpoint of the patella and another drawn from the midpoint of the patella to the tibial tubercle) represents the resultant quadriceps pull (Herrington & Nester, 2004; Livingston, 1998) and is related to normal knee valgus. The distal femur’s medial orientation forms a natural 170 degree knee valgus angle (Neumann, 2002b). This angulation explains why the quadriceps normally pull the patella laterally during terminal knee extension, a pattern described as the “law of valgus” (Fulkerson, 1997). Based on this relationship, a higher Q angle may cause the quadriceps to exert a greater lateral force vector and predispose the patella to excessive lateral tracking (Messier, Davis, Curl, Lowery, & Pack, 1991; Powers et al., 2002).

Typically, clinicians have defined an increased Q angle as one exceeding 15 to 20 degrees (Livingston, 1998). However, a review of the literature has not supported the relationship between an increased Q angle and PFPS (Caylor, Fites, & Worrell, 1993; Fulkerson, 1997; Livingston, 1998). Powers (2003) explained that most studies have taken static Q angle measurements, which may preclude detection of the Q angle’s influence during dynamic activities.

Regarding anterior cruciate ligament (ACL) injury, researchers have examined knee valgus during dynamic activities (Hewett, Myer, & Ford, 2004; Hewett et al., 2005; Lephart et al., 2002; Malinzak, Colby, Kirkendall, Yu, & Garrett, 2001). Moreover, others have shown that knee valgus can increase lateral patellofemoral joint compressive forces (Lee, Anzel, Bennett, Pang, & Kim, 1994; Lee et al., 2003). Since knee valgus can
influence the magnitude of the Q angle, researchers should evaluate its influence on PFPS during dynamic activities (Powers, 2003).

Many researchers have examined sagittal plane knee kinematics (Andriacchi et al., 1980; Brechter & Powers, 2002; Crossley, Cowan et al., 2004; Salsich, Brechter, Farwell, & Powers, 2002). The Q angle and knee valgus are affected by lower extremity frontal and transverse planes of motion (Powers, Maffucci, & Hampton, 1995); however, few authors have examined these influences in people diagnosed with PFPS. The following review of the related literature explains how faulty frontal and transverse planes of motion can lead to patellofemoral joint dysfunction and contribute to PFPS etiology.

Review of the Related Literature
Theoretical Overview of the Lower Extremity Kinetic Chain

Tiberio (1987) theorized that PFPS could result from extrinsic factors (e.g., hip, foot, and ankle influences) and described how excessive subtalar pronation could adversely affect knee function. Specifically, excessive pronation is coupled with tibial internal rotation. During normal gait, the knee must extend from approximately 12% to 40% of the normal gait cycle (Perry, 1992). Knee extension occurs when the tibia is in an externally rotated position relative to the femur (the screw-home mechanism) (Norkin & Levangie, 2001). Based on this relationship, the femur must compensate for increased tibial internal rotation through even greater femoral internal rotation. In other words, greater femoral internal rotation relative to tibial rotation will enable knee extension (Tennant et al., 2001).

It has been shown that excessive femoral internal rotation can facilitate lateral patella tracking and increase patellofemoral joint contact pressures (Lee et al., 1994; Lee et al., 2003; Powers et al., 2003). Tiberio (1987) assumed that these rotational influences originated from the distal aspect of the lower extremity kinetic chain. However, it was not known if excessive femoral internal rotation may initially occur proximally and if excessive subtalar pronation actually contributed to PFPS.
Subtalar Pronation, Tibial Rotation, and PFPS

Researchers have used both static and dynamic methods to assess relationships between subtalar pronation, tibial rotation, and PFPS. Powers et al. (1995) initially measured static rearfoot postures in subjects diagnosed with and without PFPS in a non-weight bearing (prone) position. They hypothesized that PFPS subjects would demonstrate greater rearfoot varus and require excessive and prolonged pronation to achieve medial rearfoot and forefoot contact during gait. Therefore, excessive pronation, coupled with tibial internal rotation, would lead to greater femoral internal rotation and contribute to PFPS pathology (Tiberio, 1987).

Results from this study partially supported this theory. PFPS subjects had an average of 8.9 degrees rearfoot varus compared to 6.8 degrees for controls. Although these amounts varied significantly, it was unclear if differences were clinically relevant. Also, Powers et al. (1995) did not determine if PFPS subjects actually demonstrated excessive pronation and tibial internal rotation during gait.

Livingston and Mandigo (2003) examined the magnitude of right and left rearfoot angles under a static, weight bearing condition but found no significant differences between asymptomatic, unilateral, and bilateral PFPS subjects. Interestingly, although not statistically significant, asymptomatic controls demonstrated greater rearfoot valgus angles for both limbs. Livingston and Mandigo concluded that the magnitude of rearfoot valgus may not predict PFPS etiology.

Other researchers have examined the relationship between rearfoot motion and PFPS during dynamic activities. Messier et al. (1991) compared rearfoot kinematics, kinetics, isokinetic strength, and Q angles in runners diagnosed with and without PFPS. They found that the magnitude of pronation did not discriminate between groups. However, regression analysis showed that the Q angle was the most predictive factor for PFPS pathology. It should be noted that changes in the Q angle can result from other lower extremity rotations, like the femur (Powers, 2003). Therefore, PFPS subjects may have greater hip internal rotation not assessed in the study. Although these findings are in contrast with others (who did not find an association between PFPS and the Q angle), they suggested that the Q angle might have greater importance when examining subjects during dynamic activities.
More recently, Powers et al. (2002) investigated the relationship between pronation and lower extremity rotations (tibial and femoral) in females diagnosed with and without PFPS during gait. They hypothesized that symptomatic subjects would exhibit larger degrees of pronation, tibial internal rotation, and femoral internal rotation, as theorized by Tiberio (1987). Subjects underwent motion analysis while ambulating at a self-selected pace and demonstrated similar magnitudes and timing for peak pronation and tibial rotation, which were in agreement with Messier et al. (1991).

Powers et al. (2002) did find significant group differences in femoral movement. PFPS subjects demonstrated 2.1 degrees of femoral external rotation compared to 1.6 degrees of internal rotation for the control group. Peak femoral rotation for the PFPS subjects also occurred later in the gait cycle compared to control subjects. Powers et al. concluded that subjects with PFPS might have used decreased femoral internal rotation as a compensatory strategy for decreasing the Q-angle. They also cautioned that pelvic rotation might have influenced femoral position. Therefore, future research should investigate the influence of pelvic position on femoral position and PFPS.

Femoral Rotation and PFPS

Historically, researchers have described PFPS etiology as abnormal movement of the patella on the femur (typically described during open kinetic chain activities), even though PFPS patients typically complain of pain during activities involving a flexed knee in a loaded position. Powers et al. (2003) examined femoral and patella movement during non-weight bearing and weight bearing extension using kinematic magnetic resonance imaging. Six females with PFPS and a history of lateral patellar subluxation participated. They obtained axial images as subjects extended their knee from 45 degrees to 0 degrees flexion with a load equal to 5% body weight donned on the ankle during the non-weight bearing exercise. For the weight bearing exercise, subjects performed a single leg squat from 0 degrees to 45 degrees knee flexion. The researchers then calculated bisect offset index (to determine medial/lateral displacement), patellar tilt angle, femoral rotation, and patella rotation.

Powers et al. (2003) reported greater lateral patellar displacement and lateral patellar tilt during the non-weight bearing exercise but increased femoral internal rotation during the weight bearing exercise. They also identified differences between patellar
rotation during non-weight bearing and weight bearing exercise. Lateral patella rotation occurred more during non-weight bearing extension and changed very little during weight bearing extension. Powers et al. characterized non-weight bearing extension as the patella rotating on the femur and weight bearing extension as the femur rotating beneath the patella. Results suggested that pelvic and femoral position may have a significant influence on patella tracking.

**Dynamic Knee Valgus and Hip Motion**

As discussed above, many studies have not supported the relationship between an excessive Q angle and PFPS, possibly because of the static nature of this measure (Herrington & Nester, 2004). However, knee valgus during dynamic activities may explain better the relationship between the Q angle and PFPS etiology. Knee valgus represents a frontal plane motion that may result from femoral adduction. Because the hip abductors provide frontal plane stabilization, weakness can lead to excessive hip adduction and increased knee valgus (McFadyen & Winter, 1988; Neumann, 2002a; Neumann & Hase, 1994; Perry, 1992; Sahrmann, 2002; Simoneau, 2002). Together, these motions can increase the Q angle and adversely affect patellofemoral joint function (Powers, 2003).

Only a single case study has specifically examined hip frontal, as well as transverse, plane motion for subjects diagnosed with PFPS (Mascal et al., 2003). Two females diagnosed with PFPS underwent strength testing using a HHD, while a single subject completed a three-dimensional motion analysis (it was not disclosed why only one subject underwent motion analysis). For this purpose, the subject descended a 20-cm high step, over a 3-second period, for a total of 3 repetitions. The investigators then calculated average hip adduction and hip rotation angles during the entire stance phase of stair descent. Following the initial evaluation, subjects completed a 14-week intervention that focused on hip, pelvis, and trunk musculature function and underwent a post-intervention evaluation in the same manner.

At the end of the intervention, both subjects demonstrated improved knee (20% and 10% increase) and hip (increase range 42% - 317%) strength. The subject who underwent motion analysis had average hip adduction of 8.7 degrees prior to and 2.3 degrees immediately following the intervention. She also improved hip internal rotation
from 1.4 degrees to 2.6 degrees external rotation. Results suggested that a hip, pelvis, and trunk muscle strengthening program positively affected lower extremity kinematics during stair-stepping in a subject diagnosed with PFPS. Mascal et al. (2003) concluded that a decrease in both hip adduction and internal rotation would move the patella lateral relative to the ASIS and decrease the dynamic Q angle. Although the authors believed that this was a clinically relevant finding, additional studies are needed to generalize these findings to a broader PFPS patient population.

**Lateral Patellofemoral Contact Pressures**

Although PFPS can result from a chronic overloading of the patellofemoral joint, stress may also develop from abnormal mechanics. Researchers have demonstrated how faulty lower extremity transverse and frontal plane movements can increase patellofemoral contact pressures. The following section explains how femoral and tibial rotation (transverse plane) and a higher Q angle (frontal plane) can affect patellofemoral contact pressures.

**Rotational influences.** Lee et al. (1994) examined patellofemoral contact pressures during different amounts of fixed femoral rotational deformities. Using a cadaveric model, they compared patellofemoral contact pressures at varying amounts of femoral internal and external rotation. They found that smaller amounts (0 to 20 degrees) of either internal or external rotation had little effect on patellofemoral contact pressures. However, significant increases occurred during greater amounts of rotation. Specifically, femoral external rotation caused more stress to the patella’s medial facets whereas internal rotation caused more stress to the lateral facets. Based on these findings, increased femoral internal rotation, which may occur during dynamic activities, could lead to patellofemoral joint irritation.

Others have examined influences from tibial rotation. Lee, Yang, Sandusky, & McMahon (2001) used a cadaveric model to determine the relationship between tibial rotation and patellofemoral joint contact pressures. They found higher contact pressure readings during external tibial rotation, compared to tibial internal rotation, which occurred primarily at the lateral patellar articular facets. This finding differed from the effect during femoral rotation. Lateral patellar contact pressures resulted from greater femoral internal rotation whereas these same contact pressures increased with higher
tibial external rotation. Together, femoral internal rotation and tibial external rotation increase the Q angle and the resultant lateral (valgus) quadriceps pull.

Csintalan, Schultz, Woo, McMahon, & Lee (2002) also examined the effect of tibial rotation on patellofemoral joint contact pressures in cadaveric knees. Additionally, their investigation compared differences between male and female specimens. Like Lee et al (2001), tibial external rotation increased lateral patellar facet contact pressures, especially in positions close to terminal knee extension. They also found that tibial internal rotation reduced lateral patella contact pressure with a minimal increase to medial patellar facet pressures. More important, female specimens had greater contact pressures compared to males at lower knee flexion angles. This finding was clinically relevant because it may explain why females are at greater risk for developing PFPS.

Frontal plane influences. Researchers have also investigated the influence of the Q angle (knee valgus) on patellofemoral joint contact pressures. Using 6 cadaver knees, Mizuno et al. (2001) found that a higher Q angle shifted the patella laterally and increased lateral patellofemoral joint contact pressures. However, decreasing the Q angle did not shift the patella medially. These findings were clinically relevant because they showed a relationship between a greater Q angle (knee valgus) and patellofemoral joint stress.

Elias, Cech, Weinstein, & Cosgrea (2004) examined the influence of the Q angle using a computer simulation model. This model, developed to characterize how patellofemoral joint loading influences contact pressures, incorporated quadriceps forces similar to those generated during 40 to 90 degrees of knee motion. Like Mizuno et al. (2001), Elias et al. found that a 25 degree Q angle applied greater lateral forces and contact pressures to the patella. They also reported that increasing the vastus medialis force-generating capabilities had minimal effect on decreasing contact pressures. Most important, Elias et al. found that medialization of the tibial tubercle was the most effective alteration for reducing lateral forces applied to the patella. Medialization of the tibial tubercle effectively decreased the Q angle and reduced the magnitude of valgus forces transmitted to the patella.
Researchers believe that females are at greater risk of developing PFPS because of anatomical differences that result in higher Q angles as compared to males (Almeida et al., 1999; Hutchinson & Ireland, 1995). Horton and Hall (1989) measured Q angles, pelvic width, and femoral length in a group of male and female subjects. Overall, males had wider pelvises and longer femurs. However, females had a greater average Q angle (15.8 degrees) compared to males (11.2 degrees).

Horton and Hall (1989) reported a significant correlation between gender and Q angle ($r = -0.517$) when controlling the effects of femoral and pelvic width. Alternatively, there was no significant correlation between Q angle and the anatomic measures when eliminating the effect of gender. They concluded that females have larger Q angles but were unable to provide an anatomical explanation or a new predictor of the Q angle.

Livingston and Gahagan (2001) further examined the relationship between gender, pelvic width, and femoral length. Like the Horton and Hall study, males demonstrated greater average pelvic widths and femoral lengths. Livingston and Gahagan also calculated a pelvic width to femoral length ratio for each group and found that females had greater ratios than males. They concluded that a shorter femoral length, relative to pelvic width, may affect the magnitude of hip adduction that females might require to position their feet under the body’s center of mass during functional activities. Therefore, excessive hip adduction could increase knee valgus and facilitate lateral patella tracking in females.

Researchers have examined hip and knee kinematics in females during dynamic activities (Ferber, Davis, & Williams, 2003; Hewett et al., 2005; Lephart et al., 2002; Malinzak et al., 2001; Noyes, Barber-Westin, Fleckenstein, Walsh, & West, 2005). They have shown that females perform running, cutting, and jumping activities with increased femoral adduction, femoral internal rotation, knee valgus, and tibial external rotation. Although designed to investigate ACL injury, findings from these studies are relevant to PFPS because these combined motions can increase both lateral patella tracking and patellofemoral joint compressive forces (Csintalan et al., 2002; Lee et al., 1994; Lee et al., 2001; Mizuno et al., 2001; Powers, 2003).
In summary, PFPS was originally thought to result from an excessive Q angle. More recent studies have suggested that dynamic knee valgus angles might be more indicative of patellofemoral joint dysfunction. It has also been shown that females perform many dynamic activities with the knee in a more valgus position. Based on these findings, the current study was designed to further investigate the relationship between hip adduction, hip internal rotation, and knee valgus in subjects diagnosed with and without PFPS. A female subject population was chosen because females are more likely to demonstrate these movement patterns (Csintalan et al., 2002; Mascal et al., 2003).

**Purpose and Research Hypotheses**

The purpose of this study was to compare hip and knee kinematics for females diagnosed with and without PFPS. It was hypothesized that female subjects diagnosed with PFPS would demonstrate the following:

1. PFPS subjects would demonstrate greater average hip adduction, hip internal rotation, and knee valgus angles during the stance phase of stair descent compared to control subjects.
2. PFPS subjects would demonstrate greater peak hip adduction, hip internal rotation, and knee valgus angles during the stance phase of stair descent compared to control subjects.
3. PFPS subjects would achieve peak hip adduction, hip internal rotation, and knee valgus angles later in the stair descent cycle compared to control subjects. This hypothesis is clinically relevant because PFPS subjects may demonstrate motions that apply a valgus force to the patella over a longer period of time.

**Methodology**

**Subjects**

Eighteen females diagnosed with PFPS (age = 24.5 ± 3.2 years, height = 1.68 ± 0.1 m, body mass = 618.0 ± 89.3 N, pain = 4.4 ± 1.5 cm, duration of symptoms = 14.4 ± 12.8 months) and 18 asymptomatic females (age = 23.9 ± 2.8 years, height = 1.67 ± 0.1 m, body mass = 608.2 ± 83.4 N) participated in this study. All subjects met the inclusion criteria, as summarized in Chapter 1, and signed an informed consent approved by the University of Kentucky Institutional Review Board prior to participation.
Procedures

First, subjects completed a 10-cm visual analog scale reflecting usual pain during the past week (Crossley, Bennell et al., 2004). They then rode a stationary bike for 3 minutes in a pain-free range of motion at a submaximal speed. Next, retroreflective markers, with a diameter of 20 mm, were meticulously placed on subjects using a standard Cleveland Clinic marker setup. After collecting an anatomic calibration file, subjects were shown the stair stepping task and allowed 5 practice trials. They were instructed to ascend and descend two 20-cm high steps, ensuring that the test extremity lifted and lowered the body on the first and third steps, respectively. Subjects also took a minimum of 3 strides prior to and immediately following stair stepping in order to maintain a continuous movement pattern. All subjects performed the task at a standardized rate of 96 beats per minute (Cowan et al., 2000; Gilleard et al., 1998).

After demonstrating proficiency with the test, subjects performed 10 test trials. During this time, video data were sampled at 60 Hz. Data from the last 5 trials were analyzed because of potential learning effects that might have been associated with earlier trials, even with subjects having performed 5 practice trials. Refer to Appendix C for unit specifications of the motion analysis equipment used in this study.

Data Processing

Video data were processed in the manner described in Appendix D. Hip transverse plane, hip frontal plane, and knee frontal plane angles for the last 5 individual trials were calculated using OrthoTrak 5.0 software (Motion Analysis Corporation, Santa Rosa, CA) using methods described by Grood and Suntay (1983) (Refer to Table 2.1). The individual trials were then normalized to 100% of the gait cycle and ensemble averaged. Average joint angles, peak joint angles, and time to peak joint angle (expressed as the percentage of the gait cycle in which it occurred) from the normalized data during the stance phase of stair descent were used for statistical analysis.

Statistical Analysis

Independent \( t \)-tests were used to determine group differences in age, height, and weight. Separate 2 X 3 (group X angle) ANOVAs for repeated measures on angle were used to determine differences in average and peak joint angles during stance. A 2 X 3 (group X time) ANOVA for repeated measures on time was used to determine
differences in time to peak angle during stance. Effect sizes were calculated for all measures as described by Cohen (1988). All statistical analyses were performed using SPSS version 12.0 (SPSS, Inc., Chicago, IL). Level of significance was established at the 0.05 level; the sequentially rejective Bonferroni (Bonferroni-Holm) post hoc test (Holm, 1979) was used to determine the significance of interactions for the two-factor ANOVAs.

**Results**

Independent *t*-tests for subject demographics revealed similar age, height, and weight (*p* > .44) characteristics for both groups. Results from separate ANOVAs for average joint angles and peak joint angles showed neither a significant main effect nor an interaction effect (*p* > .05). The ANOVA for time to peak angle had a significant interaction effect (*p* = .004). PFPS subjects demonstrated a greater time to peak angle for knee valgus. Tables 4.1 through 4.3 summarize all descriptive data and effect size calculations for each motion.

**Discussion**

Recently, researchers have focused much attention on the association between excessive hip motion and PFPS. They have theorized that excessive hip transverse and frontal plane motion may increase knee valgus. Knee valgus, in turn, can result in greater lateral patella tracking and higher patellofemoral joint compressive forces (Fulkerson, 1997; Lee et al., 1994). To date, no researchers have simultaneously examined hip kinematics and knee valgus to corroborate this theory.

To better understand this association, the current study evaluated hip adduction, hip internal rotation, and knee valgus in subjects diagnosed with and without PFPS during stair descent. It was hypothesized that PFPS subjects would demonstrate greater hip adduction, hip internal rotation, and knee valgus compared to controls. However, results did not support the initial premise as PFPS and control subjects demonstrated similar average motion and similar peak angles during stair descent.

**Average Hip Internal Rotation**

Only a single case study (Mascal et al., 2003) has examined hip transverse plane kinematics in a subject diagnosed with PFPS during the stance phase of stair descent. In this study, the subject demonstrated average hip internal rotation of 1.4 degrees, which was comparable to 2.06 degrees for the current study. However, control subjects in the
current study exhibited similar average hip internal rotation (mean = 0.99 degrees; \( p = 0.60 \)). This finding implied that PFPS subjects in both studies had values similar to asymptomatic controls.

It should be noted that subjects in the current study demonstrated much variability, which would preclude the ability to attain statistically significant differences. As discussed in Chapter 2, researchers have had great difficulty measuring transverse plane motion, especially for the hip (Kadaba et al., 1989). Therefore, a limitation of these findings could be ongoing problems associated with capturing hip rotation movement. This might also explain why other researchers (Powers et al., 2003; Tennant et al., 2001) have utilized kinematic magnetic resonance imaging techniques to refine this measurement.

**Average Hip Adduction**

Control subjects demonstrated 2.07 degrees more hip adduction than PFPS subjects. It was interesting to note that the \( p \)-value of 0.15 might have inferred a trend toward statistical significance. As noted above, subjects demonstrated high variability that would preclude attaining statistical significance. Post hoc power analysis (\( \beta = .80 \)), based on the current study’s mean hip adduction motion and subject variability, showed that a minimum of 27 subjects per group would be required. Therefore, differences between PFPS and control subjects might have existed, but were not detected, because of the relatively low sample size (Portney & Watkins, 2000b).

Effect size calculations are another way to assess group differences. Effect sizes are important because they may identify clinically relevant differences not found with statistical inference (Portney & Watkins, 2000c). The current study’s effect size for average hip adduction was 0.55, which represented a medium-to-large effect (Cohen, 1988). This finding suggested greater between group differences that could be clinically relevant for the following reason.

Subjects in the current study had chronic symptoms and reduced hip adduction might have represented a compensatory strategy. By limiting hip adduction, PFPS subjects could decrease the Q angle (valgus angle) and minimize the lateral force vector applied to the knee (Fulkerson, 1997). Powers et al. (2002) found a similar pattern with respect to femoral rotation for subjects with and without PFPS during normal gait. PFPS
subjects maintained an average of 2.1 degrees femoral external rotation, compared to 1.6 degrees of femoral internal rotation for control subjects. They concluded that PFPS subjects could reduce the Q angle by maintaining greater femoral external rotation. Therefore, the medium-to-large effect size (0.55) could have highlighted a clinically relevant compensatory strategy exhibited by PFPS subjects.

The subject in the Mascal et al. study (2003) demonstrated greater hip adduction (8.7 degrees) than the average for all subjects in the current study. One reason for this variation may result from methodological differences. In the Mascal et al. study, the subject lowered her center of mass slowly over a 3-second period. Descending a single step at a slower pace most likely represented a more difficult maneuver. It would require greater hip control and might represent a better way for identifying between group differences in hip adduction.

The current study used a standardized rate (96 beats per minute) for stair descent because it provided a reliable measure and represented an activity of daily living that has typically provoked patellofemoral joint pain. It was a relatively easier task, compared to the Mascal study, that subjects completed using smaller amounts of hip adduction. Therefore, future researchers may wish to compare hip adduction between subjects during a more demanding task.

**Average Knee Valgus**

It was originally hypothesized that PFPS subjects would descend stairs with greater knee valgus. Surprisingly, the results did not support this hypothesis as all subjects, on average, maintained a varus position. Average knee varus was 2.89 degrees for control and 5.70 degrees for PFPS subjects.

To my knowledge, no studies have reported knee frontal plane kinematic values for normal or PFPS subjects during stair descent. Yu et al. (1997) examined knee valgus during stair-stepping for healthy subjects but only reported intratrial reliability, and not descriptive, data. However, Zeller et al. (2003) examined knee frontal plane motion during a single-legged squat, a maneuver similar to stair descent. During the actual squat movement, healthy female subjects demonstrated knee varus similar to those for the current study’s control subjects.
PFPS subjects in the current study, on average, maintained a greater knee varus position compared to controls. Like hip internal rotation, high variability probably accounted for lack of statistical significance found between groups. A medium effect size (0.41) was calculated and might have highlighted a clinically relevant finding. PFPS subjects might have used knee varus as another compensatory strategy to reduce the Q angle and lateral valgus force applied to the knee.

**Peak Angles and Time to Peak Angles**

Other researchers have also examined peak motion and time to peak motion to differentiate movement patterns between subjects diagnosed with and without PFPS (Messier et al., 1991; Powers et al., 2002). They believed that greater and prolonged subtalar, tibial, and femoral rotations could adversely affect knee function. Based on these studies, it was hypothesized that PFPS subjects would move toward greater amounts of hip adduction, hip internal rotation, and knee valgus during stair descent. Results from the current study did not support this premise as PFPS and control subjects demonstrated similar peak values for hip and knee kinematics. Effect sizes for all measures were also minimal (< 0.35).

Subjects also demonstrated similar time to peak motion for hip kinematics. They achieved maximum hip internal rotation and hip adduction at approximately 42% of the stair descent cycle. Conversely, PFPS subjects achieved maximum knee valgus at 32% of the stair descent cycle while controls reached maximum knee valgus at 17% of the cycle. These values were significantly different and inferred that PFPS subjects moved toward a valgus position over a longer period of time compared to controls. Therefore, prolonged valgus might apply a lateral force vector over a longer duration that could stress patellofemoral joint structures.

**Conclusion and Future Direction**

Caution should be taken when interpreting kinematic data from the current study. As noted earlier, data for subjects within both groups was highly variable. The stair-stepping task used would be less demanding than others that incorporate jumping and landing. Subjects within each group might have utilized different hip and knee strategies, which would make it more difficult to identify between group differences. Therefore,
additional studies should continue to investigate frontal and transverse plane hip and knee kinematics but using more demanding activities (Powers, 2003).

It has also been reported that PFPS subjects can reduce forces applied to the knee by walking at slower velocities (Brechter & Powers, 2002; Powers et al., 2002; Powers et al., 1996). As discussed in Chapter 2, a standardized rate was used to facilitate between day reliability. Subjects in the current study might have utilized a different cadence, if given the choice, for purposes of reducing ground reaction forces that might be transmitted through the lower extremity kinetic chain (Powers et al., 1999). Future studies should incorporate kinetics to identify differences between subjects diagnosed with and without PFPS.

In summary, PFPS and control subjects demonstrated similar hip and knee kinematics. PFPS subjects had chronic symptoms and may have compensated for faulty movement patterns that might have contributed to PFPS pathology. It is unknown if subjects with more acute symptoms might have exhibited different movement patterns. Finally, researchers have proposed different classifications of PFPS etiology (Wilk et al., 1998; Witvrouw et al., 2005), and the current study used inclusion criteria based on clinical practice (pain during provocative activities like stair-stepping, prolonged sitting, and squatting). Therefore, future studies should refine inclusion criteria to assess PFPS subjects who exhibit hip weakness that may result in excessive hip adduction, hip internal rotation, and knee valgus.
Table 4.1
Summary of Hip Internal Rotation Angles and Time to Peak Hip Internal Rotation Angle

<table>
<thead>
<tr>
<th></th>
<th>Controls</th>
<th>PFPS</th>
<th>( p )-value</th>
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<tbody>
<tr>
<td>Mean Hip Internal (External) Rotation Angle</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mean (degrees)</td>
<td>0.99</td>
<td>2.06</td>
<td>0.60</td>
</tr>
<tr>
<td>SD</td>
<td>8.02</td>
<td>7.19</td>
<td></td>
</tr>
<tr>
<td>Cohen’s ( d )</td>
<td></td>
<td></td>
<td>0.14( \delta )</td>
</tr>
<tr>
<td>Peak Hip Internal (External) Rotation Angle</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mean (degrees)</td>
<td>5.64</td>
<td>6.62</td>
<td>0.76</td>
</tr>
<tr>
<td>SD</td>
<td>7.72</td>
<td>7.94</td>
<td></td>
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<tr>
<td>Cohen’s ( d )</td>
<td></td>
<td></td>
<td>0.13( \delta )</td>
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<tr>
<td>Time to Peak Hip Internal (External) Rotation Angle</td>
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<td></td>
<td></td>
</tr>
<tr>
<td>Mean (percent of stair descent cycle)</td>
<td>42.28</td>
<td>42.72</td>
<td>0.86</td>
</tr>
<tr>
<td>SD</td>
<td>7.04</td>
<td>6.16</td>
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<tr>
<td>Cohen’s ( d )</td>
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<td>0.07( \delta )</td>
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SD = standard deviation
PFPS = patellofemoral pain syndrome
\( \delta \) Small effect (Cohen, 1988)
Table 4.2
Summary of Hip Adduction Angles and Time to Peak Hip Adduction Angle

<table>
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<th>Controls</th>
<th>PFPS</th>
<th>p-value</th>
</tr>
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<tbody>
<tr>
<td><strong>Mean Hip Adduction (Abduction) Angle</strong></td>
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<td></td>
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<tr>
<td>Mean (degrees)</td>
<td>2.61</td>
<td>0.54</td>
<td>0.15</td>
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<tr>
<td>SD</td>
<td>3.87</td>
<td>3.72</td>
<td></td>
</tr>
<tr>
<td>Cohen’s d</td>
<td></td>
<td></td>
<td>0.55§</td>
</tr>
<tr>
<td><strong>Peak Hip Adduction (Abduction) Angle</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mean (degrees)</td>
<td>5.43</td>
<td>3.99</td>
<td>0.31</td>
</tr>
<tr>
<td>SD</td>
<td>3.51</td>
<td>4.57</td>
<td></td>
</tr>
<tr>
<td>Cohen’s d</td>
<td></td>
<td></td>
<td>0.35β</td>
</tr>
<tr>
<td><strong>Time to Peak Hip Adduction (Abduction) Angle</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mean (percent of stair descent cycle)</td>
<td>43.11</td>
<td>39.78</td>
<td>0.42</td>
</tr>
<tr>
<td>SD</td>
<td>9.19</td>
<td>13.94</td>
<td></td>
</tr>
<tr>
<td>Cohen’s d</td>
<td></td>
<td></td>
<td>0.28‡</td>
</tr>
</tbody>
</table>

SD = standard deviation
PFPS = patellofemoral pain syndrome
§ Medium to large effect (Cohen, 1988)
β Medium effect (Cohen, 1988)
‡ Small to medium effect (Cohen, 1988)
Table 4.3
Summary of Knee Valgus Angles and Time to Peak Knee Valgus Angle

<table>
<thead>
<tr>
<th></th>
<th>Controls</th>
<th>PFPS</th>
<th>( p )-value</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Mean Knee Varus (Valgus) Angle</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mean (degrees)</td>
<td>2.89</td>
<td>5.70</td>
<td>0.28</td>
</tr>
<tr>
<td>SD</td>
<td>8.05</td>
<td>5.42</td>
<td></td>
</tr>
<tr>
<td>Cohen’s ( d )</td>
<td></td>
<td></td>
<td>0.41( \beta )</td>
</tr>
<tr>
<td><strong>Peak Knee Varus (Valgus) Angle</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mean (degrees)</td>
<td>(0.64)</td>
<td>1.20</td>
<td>0.38</td>
</tr>
<tr>
<td>SD</td>
<td>7.72</td>
<td>4.48</td>
<td></td>
</tr>
<tr>
<td>Cohen’s ( d )</td>
<td></td>
<td></td>
<td>0.29( \dagger )</td>
</tr>
<tr>
<td><strong>Time to Peak Knee Valgus Angle</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mean (percent of stair descent cycle)</td>
<td>17.28</td>
<td>32.11</td>
<td>0.004( \alpha )</td>
</tr>
<tr>
<td>SD</td>
<td>14.22</td>
<td>15.79</td>
<td></td>
</tr>
<tr>
<td>Cohen’s ( d )</td>
<td></td>
<td></td>
<td>0.99( \pm )</td>
</tr>
</tbody>
</table>

SD = standard deviation
PFPS = patellofemoral pain syndrome
\( \beta \) Medium effect (Cohen, 1988)
\( \dagger \) Small to medium effect (Cohen, 1988)
\( \dagger \) Medium effect (Cohen, 1988)
\( \alpha \) PFPS significantly different from control subjects
\( \pm \) Large effect (Cohen, 1988)
CHAPTER FIVE

Etiology of Patellofemoral Pain Syndrome. A proximal link to a distal problem

Witvrouw et al. (2005) recently stated that PFPS continues to be one of the most challenging musculoskeletal pathologies clinicians face. It remains a multifactorial problem, one not even having a consensus regarding the terminology for pain. Although often referred to as anterior knee pain, PFPS has encompassed other diagnoses such as chondromalacia patellae, runner’s knee, jumper’s knee, and patellar arthralgia. The common bond among diagnoses has been the thought that PFPS was solely a “knee” disorder. Based on this premise, interventions have focused primarily on knee articular structures and musculature. Recently, though, researchers have shown that subjects diagnosed with knee pain report significant pain reduction after completing a rehabilitation program that focused on hip strengthening (Cornbleet, Sahrmann, & Norton, 2005; Crossley et al., 2002; Fredericson et al., 2000; Mascal et al., 2003; Pettitt & Dolski, 2000). Although the hip may contribute to pathology, few have specifically examined hip function and its influence on PFPS.

Past and Present Findings

The primary purpose of this dissertation was to determine the association, if any, between hip impairments and PFPS. As summarized in Chapter 3, subjects diagnosed with PFPS had knee extensor weakness but even greater hip abductor and external rotator weakness, findings in agreement with Ireland et al. (2003). Weakness was also apparent during functional performance testing, as evidenced by PFPS subjects having lower limb symmetry indexes (Loudon et al., 2002). Regarding EMG measures, researchers have reported higher quadriceps activity during stair-stepping and knee extension exercise for PFPS subjects (Mohr et al., 2003; Powers, 2000). They inferred that greater motor recruitment was necessary to compensate for quadriceps weakness, although strength was not measured. Results from the current study corroborated their premise as PFPS subjects had greater muscle activity, especially during the loading response and single leg stance intervals of stair descent.

Others have hypothesized that quadriceps and gluteus medius activation onsets may be altered in subjects diagnosed with PFPS. Some have found delayed VM activation relative to the VL (Cowan, Bennell, Crossley et al., 2002; Cowan et al., 2001;
Voight & Wieder, 1991); whereas others have reported delayed GM onset relative to the quadriceps (Brindle et al., 2003). Results from the current study did not support these findings and agreed with others who were unable to substantiate this theory (Karst & Willett, 1995; Powers et al., 1996; Sheehy et al., 1998). Therefore, it remains elusive if delayed muscle activations are present and significant, if in fact, any exist.

The data from Chapter 4 summarized hip and knee kinematics. Although it was hypothesized that PFPS subjects would demonstrate excessive hip adduction, hip internal rotation, and knee valgus, results from this study did not support this premise. An ongoing problem was significant subject variability and inherent difficulties with measuring frontal and transverse plane motion (McFadyen & Winter, 1988). Another reason for similar movement patterns among subjects might have been compensatory strategies that PFPS subjects developed (Powers et al., 2002). Excessive hip and knee motions might have preceded the development of PFPS. Therefore, prospective studies are needed to further understand these biomechanical influences on PFPS etiology.

Enhancing the Evaluation Process for PFPS

Nonoperative treatment is the gold standard treatment for PFPS, with physical rehabilitation being the most recommended intervention (Fulkerson, 2002). An important aspect of rehabilitation is the evaluation process. However, difficulty arises with pain as the primary complaint, since it is hard to objectively measure and quantify. Based on this subjectivity, a secondary purpose of this dissertation was to identify reliable measurement tools that may enhance the evaluation process.

Measurement reliability is critical because clinicians must use tools capable of identifying not only initial impairments but also changes throughout the rehabilitation process. Unfortunately, few researchers have addressed measurement reliability, with most only examining reliability specific to asymptomatic subjects. It is not known if subjects with pathology would respond similarly with repeat testing.

This study examined measurement reliability for evaluation tools commonly used to assess PFPS impairments: 1) functional performance tests, 2) hand-held dynamometry, 3) surface electromyography, and 4) motion analysis. Functional performance tests had acceptable ICCs, with exception of the anteromedial lunge test for PFPS subjects. For this test, PFPS subjects improved significantly on the second day of testing (See Table
PFPS subjects might have anticipated increased discomfort with the lunge activity during initial testing. However, they reported similar pain ratings (a rating \( \pm .05 \) cm of the original score) prior to the second day of testing. Therefore, PFPS subjects might have performed repeat testing more aggressively due to a lower pain expectation.

HHD was conducted using a belt-resisted method and the “make” test. ICCs reflected good reproducibility but some variability in hip measures for PFPS subjects. ICCs for PFPS subjects also had low standard errors of measurement (SEM). The small SEM indicated high measurement precision and limited variability between measures, findings that would support the reliability of these measures.

Overall, ICCs for EMG amplitudes and muscle onset were acceptable. Electrode placement was meticulously documented, and subjects completed the stair-stepping task in a standardized manner. Together, these procedures likely enhanced reproducibility. Finally, ICCs for kinematic measures exceeded .70 for all motions, except for the hip transverse plane in PFPS subjects. This finding suggested greater biological variability in the PFPS subjects.

**Associations between Pain and Clinical Measures**

PFPS is a diagnosis based primarily on a patient’s complaint of pain and perceived loss of function. Clinicians routinely assess these parameters using a 10-cm visual analog scale (VAS) and Anterior Knee Pain Scale (Kujala et al., 1993), tools capable of providing reliable and valid outcome measures (Crossley, Bennell et al., 2004). As discussed above, clinicians measure other parameters, such as functional performance, strength, neuromuscular activity, and kinematics. Although these measures are routinely evaluated, limited data exist regarding associations between these impairments and patellofemoral joint pain.

Pain does not necessarily affect muscle or joint mechanical function; however, identifying parameters that have stronger associations with pain may assist clinicians with the evaluation process. For example, if the step-down functional performance test has a stronger association with pain, compared to the anteromedial lunge, then it may represent a better evaluation tool specific for patients diagnosed with PFPS.
To better understand associations between impairments and pain, correlation analyses were conducted. As described in previous chapters, all subjects documented their usual pain over the previous week using a 10-cm VAS (See Appendix E). This measure was chosen because of its reliability, validity, and responsiveness for measuring outcomes specific to PFPS subjects (Crossley, Bennell et al., 2004). The Spearman’s rho ($\rho$) coefficient was calculated for most measures since these data (control and PFPS treated as a single sample) did not meet the assumption of normality (Shapiro-Wilks (1965) W statistic $p < .05$). Data for VM-VL timing differences and peak hip kinematics were normally distributed (Shapiro-Wilks W statistic $p > .05$) and therefore evaluated using the Pearson product moment coefficient ($r$).

Results from Correlation Analyses

The step-down functional performance test had a moderate, and significant, inverse correlation with pain ($\rho = -0.66$) while the anteromedial lunge had a weak association ($\rho = -0.25$). For strength, hip abduction ($\rho = -0.36$) and external rotator ($\rho = -0.60$), but not knee extensor ($\rho = -0.27$), measures were significantly correlated with pain. EMG amplitudes during the loading response and single leg stance intervals of stair descent had a moderate and significant correlation with pain ($\rho$ range $= 0.41 - 0.65$; $p < .05$). Only GM activity during the preswing interval showed a moderate association with pain ($\rho = 0.42$). Significant associations were not identified for either pain and EMG onsets or pain and kinematics. Tables 5.1 through 5.5 summarize correlation coefficients for all measures.

Interpretation of Correlation Analyses

Functional Performance Tests

Loudon et al. (2002) were the only researchers who have performed a correlation analysis on the functional performance tests used in this study. The step-down and anteromedial lunge tests were chosen because they reported higher ICCs and higher correlation coefficients for these tests. They calculated correlation values of 0.57 and 0.73 for the step-down and anteromedial lunge test, respectively.

Results from the current study were only in partial agreement with Loudon et al. (2002). The correlation value for the step-down test ($\rho = -0.66$) was significant ($p = 0.01$) and showed a moderate inverse association between pain and limb symmetry index.
Correlation values between studies appeared similar, although it was unclear why the Loudon study’s coefficients showed a positive relationship. It would have seemed more logical that PFPS subjects, having higher pain levels, would have lower levels of performance, implying an inverse relationship between pain and function.

The anteromedial lunge test correlation value (ρ = -0.25) was much lower than that reported by Loudon et al. (2002), even though subjects in the current study performed this test in an identical manner. One possible explanation may be differences in reported pain. PFPS subjects in the current study had an average pain rating of 4.4 on a 10-cm VAS; Loudon et al. did not report this parameter. Therefore, subjects in the Loudon et al. study might have experienced greater pain during the anteromedial lunge test, compared to controls, which could have accounted for differing associations with pain.

**Hip and Knee Strength**

Researchers have shown that subjects diagnosed with PFPS experience quadriceps weakness (Powers et al., 1997; Roush et al., 2000; Stiene et al., 1996). Recently, researchers have also identified hip weaknesses in this patient population (Ireland et al., 2003; Niemuth et al., 2005). These findings have provided preliminary evidence regarding relationships between the hip musculature and PFPS.

Results from the current study supported previous works since subjects with PFPS demonstrated hip and knee weakness. However, the degree of correlation between pain and strength varied by structure. The association between knee extensor strength and pain was both weak (ρ = -0.27) and non-significant, findings that agreed with Powers et al. (1997). Like the current study, Powers et al. measured quadriceps strength with the knee flexed to 60 degrees, since females can generate greater torque in this position (Lieb & Perry, 1971). This position also maximized patella contact within the trochlear groove and possibly minimized pain during strength testing (Steinkamp, Dillingham, Markel, Hill, & Kaufmen, 1993). It was not known if PFPS subjects might have generated lower torque values in test positions that reproduced their pain. For example, testing knee strength close to full extension (one that would decrease overall patella contact) might have elicited greater pain, resulted in smaller torque values, and highlighted a stronger association between pain and knee extensor strength.
Alternatively, hip abductor strength showed a weak ($\rho = -0.36$), but significant, inverse correlation with pain. Hip external rotator strength showed a greater ($\rho = -0.60$), and significant, inverse association. As discussed in Chapter 3, the hip abductor and external rotators provide a stabilizing effect on the entire lower extremity and contribute to normal knee function (Simoneau, 2002). Even though knee extensor strength deserves consideration (Natri et al., 1998), the current findings showed that the hip musculature may have an even greater influence on PFPS than originally thought.

**Gluteus Medius and Vastii EMG Amplitudes and Kinematics**

Although data showed a stronger association between pain and hip strength, EMG activity may reflect better the muscle demands required for functional activities. Results from this study showed moderate and significant correlations between pain and EMG activity for all muscles during loading response and single leg stance. Results also showed a moderate and significant association between pain and GM activity during preswing.

**Loading response.** McFadyen and Winter (1988) have described the loading response interval as the most demanding throughout the stair descent cycle. During this interval, the hip and knee muscles decelerated and controlled forward and downward motion of the body onto the step. Moreover, researchers have reported relatively higher hip and knee muscle EMG activity during this interval for asymptomatic subjects (Kadaba et al., 1989; Lyons et al., 1983; McFadyen & Winter, 1988). These findings identified the stabilizing effects provided by the hip and knee musculature (eccentric muscle action) during the initial phase of stair descent.

Correlation coefficients for the GM ($\rho = 0.55$), VM ($\rho = 0.65$), and VL ($\rho = 0.41$) revealed moderate to high associations with pain and support results from strength testing (See Chapter 3). PFPS subjects had significantly less hip and knee strength compared to controls and would require greater EMG activity during the more demanding intervals of stair descent. Moreover, it is interesting to note that the correlation between knee extensor strength and pain was both weak and non-significant. This finding suggested that EMG activity may reflect better the relationship between muscular demands and pain during a functional activity.
Single leg stance. Like the loading response, moderate associations between EMG activity and pain were found for the GM (ρ = 0.46), VM (ρ = 0.46), and VL (ρ = 0.42). These findings suggested a stronger association between pain and higher VM and VL activation than that between pain and quadriceps strength. Higher EMG activity for the VM and VL, and not only the VM, might reflect an overall decrease in quadriceps function (Malone et al., 2002).

Preswing. During preswing, subjects lowered their body to contact the contralateral foot onto the next step. It was thought that greater muscle activation would be required to control this forward movement; yet this premise was not corroborated. According to McFadyen and Winter (1988), the body was likely positioned with the center of mass located more centrally over the foot, which would provide a stable base. Therefore, increased stability could have accounted for less EMG activity required during the preswing interval.

Correlation coefficients for the VM (ρ = 0.11) and VL (ρ = -0.03) with pain were weak. Conversely, the GM correlation (ρ = 0.42) showed a moderate and significant association with pain. Although unclear for this finding, this association showed that PFPS subjects had greater GM activation throughout the entire stance phase of stair descent. It is not known if such activation may stabilize the knee (prevent valgus motion).

EMG onset timing differences and kinematics. Tables 5.4 and 5.5 showed weak and non-significant associations between EMG timing differences and kinematics and pain. This finding was not unexpected since data related to these parameters from Chapters 3 and 4 did not show significant group differences. Based on these group similarities, it was not expected that pain would have been strongly correlated to these variables.

Final Concluding Thoughts

The overall purpose of this dissertation was to gain additional information regarding the relationship between hip function and PFPS. Unlike previous studies, the hip and knee were examined simultaneously. Results from this study showed that PFPS subjects demonstrated hip and knee impairments, especially for functional performance, strength, and EMG activation. Conversely, data from this study did not identify
neuromuscular timing or kinematic differences. Therefore, additional studies are required to better understand these parameters.

Pain is a determining factor for the diagnosis of PFPS. Findings from this study have provided preliminary evidence regarding significant associations between pain and hip musculature function. More importantly, these data support previous studies that have reported improvements in patient impairments following an intervention that focused on hip musculature strengthening.

My primary purpose for choosing this dissertation topic was to collect pilot data for future intervention studies. Originally, it was my intent to measure functional performance, strength, EMG activity, and kinematics variables for PFPS subjects prior to and immediately following a 6-week intervention. However, important questions needed to be answered before conducting this type study. Will the evaluation tools provide reliable measures for both PFPS and control subjects? Do differences in these parameters actually exist between subjects diagnosed with and without PFPS? Are these measures correlated with pain and, therefore, meaningful for assessing changes in pain following an intervention?

Results from this study showed the clinical tools can provide reliable measures. It was also shown that subjects with PFPS demonstrated greater deficits in hip strength and required greater hip and knee muscle activation during stair-stepping as compared to controls. However, PFPS subjects exhibited similar EMG activation onsets and kinematics as controls. Finally, a moderate association existed between pain and LSI for the step-down test. Hip strength demonstrated a stronger correlation with pain compared to that for quadriceps strength. GM, VM, and VL EMG amplitudes during the more demanding intervals of stair descent had moderate associations with pain.

Data from this study support the hip as a proximal link to a distal problem. It remains elusive if pure hip weakness or perhaps decreased hip control during functional activities is responsible for PFPS. Although future studies should employ interventions that focus on the hip, it might be more meaningful to determine if a hip strengthening program or a functional rehabilitation program may benefit PFPS subjects the best.
Table 5.1
Summary of Spearman’s rho ($\rho$) Correlation Coefficients for Functional Performance Tests and Pain

<table>
<thead>
<tr>
<th>Functional Performance Test</th>
<th>$\rho$</th>
<th>$p$-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Step-down</td>
<td>-0.66</td>
<td>0.01</td>
</tr>
<tr>
<td>Anteromedial Lunge</td>
<td>-0.25</td>
<td>0.05</td>
</tr>
</tbody>
</table>

Table 5.2
Summary of Spearman’s rho ($\rho$) Correlation Coefficients for Strength Measures and Pain

<table>
<thead>
<tr>
<th>Strength Measure</th>
<th>$\rho$</th>
<th>$p$-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hip Abductors</td>
<td>-0.36</td>
<td>0.05</td>
</tr>
<tr>
<td>Hip External Rotators</td>
<td>-0.60</td>
<td>0.01</td>
</tr>
<tr>
<td>Knee Extensors</td>
<td>-0.27</td>
<td>&gt; 0.05</td>
</tr>
</tbody>
</table>
Table 5.3
Summary of Spearman’s rho (ρ) Correlation Coefficients for Gluteus Medius, Vastus Medialis, and Vastus Lateralis Electromyographic Amplitudes and Pain

<table>
<thead>
<tr>
<th>EMG Amplitude</th>
<th>ρ</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>GM Load</td>
<td>0.55</td>
<td>0.01</td>
</tr>
<tr>
<td>VM Load</td>
<td>0.65</td>
<td>0.01</td>
</tr>
<tr>
<td>VL Load</td>
<td>0.41</td>
<td>0.05</td>
</tr>
<tr>
<td>GM SLS</td>
<td>0.46</td>
<td>0.01</td>
</tr>
<tr>
<td>VM SLS</td>
<td>0.46</td>
<td>0.01</td>
</tr>
<tr>
<td>VL SLS</td>
<td>0.42</td>
<td>0.01</td>
</tr>
<tr>
<td>GM Preswing</td>
<td>0.42</td>
<td>0.05</td>
</tr>
<tr>
<td>VM Preswing</td>
<td>0.11</td>
<td>&gt; 0.05</td>
</tr>
<tr>
<td>VL Preswing</td>
<td>-0.03</td>
<td>&gt; 0.05</td>
</tr>
</tbody>
</table>

GM = gluteus medius
VM = vastus medialis
VL = vastus lateralis
Load = loading response
SLS = single leg stance
Table 5.4
Summary of Spearman’s rho ($\rho$) and Pearson’s Product ($r$) Correlation Coefficients for Gluteus Medius, Vastus Medialis, and Vastus Lateralis Electromyographic Onset Timing Differences and Pain

<table>
<thead>
<tr>
<th>EMG Timing Difference</th>
<th>Coefficient</th>
<th>$p$-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>GM - VM</td>
<td>$\rho = 0.03$</td>
<td>$&gt; 0.05$</td>
</tr>
<tr>
<td>GM - VL</td>
<td>$\rho = 0.02$</td>
<td>$&gt; 0.05$</td>
</tr>
<tr>
<td>VL - VM</td>
<td>$r = 0.24$</td>
<td>$&gt; 0.05$</td>
</tr>
</tbody>
</table>

GM = gluteus medius  
VM = vastus medialis  
VL = vastus lateralis
Table 5.5
Summary of Spearman’s rho (\( \rho \)) and Pearson’s Product (\( r \)) Correlation Coefficients for Hip and Knee Kinematics and Pain

<table>
<thead>
<tr>
<th>Motion</th>
<th>Coefficient</th>
<th>( p )-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Average Hip Internal Rotation</td>
<td>( \rho = 0.10 )</td>
<td>&gt; 0.05</td>
</tr>
<tr>
<td>Average Hip Adduction</td>
<td>( \rho = -0.20 )</td>
<td>&gt; 0.05</td>
</tr>
<tr>
<td>Average Knee Varus</td>
<td>( \rho = 0.12 )</td>
<td>&gt; 0.05</td>
</tr>
<tr>
<td>Peak Hip Internal Rotation</td>
<td>( r = 0.11 )</td>
<td>&gt; 0.05</td>
</tr>
<tr>
<td>Peak Hip Adduction</td>
<td>( r = -0.23 )</td>
<td>&gt; 0.05</td>
</tr>
<tr>
<td>Peak Knee Valgus</td>
<td>( \rho = 0.10 )</td>
<td>&gt; 0.05</td>
</tr>
<tr>
<td>Time to Peak Hip Internal Rotation</td>
<td>( \rho = 0.08 )</td>
<td>&gt; 0.05</td>
</tr>
<tr>
<td>Time to Peak Hip Adduction</td>
<td>( \rho = 0.04 )</td>
<td>&gt; 0.05</td>
</tr>
<tr>
<td>Time to Peak Knee Valgus</td>
<td>( \rho = 0.24 )</td>
<td>&gt; 0.05</td>
</tr>
</tbody>
</table>
APPENDICES

Appendix A

Consent to Participate in a Research Study

WHY ARE YOU BEING INVITED TO TAKE PART IN THIS RESEARCH?

You are being invited to take part in a research study that involves understanding how the hip joint can cause patellofemoral knee pain syndrome, commonly referred to as anterior knee pain. You are being invited to take part in this research study because you have been diagnosed with patellofemoral pain syndrome or you have been asked to participate because you do not have knee problems and will serve as a control subject. If you volunteer to take part in this study, you will be one of about 30 people to do so.

WHO IS DOING THE STUDY?

The person in charge of this study is Lori A. Bolgla, MS (PI) of the Rehabilitation Sciences Doctoral Program. She is being guided in this research by Terry Malone, EdD and Timothy L. Uhl, PhD. There may be other people on the research team assisting at different times during the study.

WHAT IS THE PURPOSE OF THIS STUDY?

The purpose of this study will be to better understand the effect that excessive hip motion may have on the development of patellofemoral pain syndrome. Another purpose will be to determine if evaluation techniques commonly used by physicians, physical therapists, and certified athletic trainers can provide reliable measures of hip motion, hip strength, and knee function.

WHERE IS THE STUDY GOING TO TAKE PLACE AND HOW LONG WILL IT LAST?

The research procedure will be conducted at the University of Kentucky Wenner-Gren Biomedical Laboratory. The study will require that you report to the laboratory for two sessions that should last approximately 90 minutes each.

WHAT WILL YOU BE ASKED TO DO?

We will ask you for a brief medical history so that we may determine if you can participate in the study. If you are a control subject, then you may participate as long as you do not meet any exclusion criteria. If you meet the inclusion criteria, then you will sign this consent inform to signify your willingness to participate. To facilitate taking accurate tests measurements, we will ask that you wear a pair of Lycra shorts and tank top
(which we will provide if necessary). You will be compensated for your time and parking expense associated with study participation.

Pain and Function Assessment:
You will also be asked to look at a scale in which you will rate the pain that you have had in your knee during the past week. The scale will be a 10-centimeter (cm) long line having increments numbered from 0 to 10. You will be asked to place a mark on the line that represents the amount of knee pain during the past week (0 means no pain; 10 means the worst pain imaginable). You will also complete this scale at the time of your second testing session. If your knee rating is significantly different on the second test day, then you may not participate on the second testing date because this change in pain may affect the validity of results from the second test. Next, you will ride a stationary bike for 3 minutes in a manner that does not cause pain to your knee. You will then practice two functional performance tests commonly used to assess people having patellofemoral pain 3 to 5 times. You will perform the following functional performance tests:

1. **Step-Down Test**
   For the step-down test, you will stand on an 8” step using the test leg. You will lower your body enough to brush the heel of the opposite leg on the floor (in front of the step), and then raise your body upward by straightening your test knee.

2. **Anteromedial Lunge Test**
   For the anteromedial lunge test, you will stand behind a start line and then lunge (to a 90° knee angle) forward and slightly across your body with the uninvolved leg three times. We will measure the distance from the start line to the back of your heel for each trial and take 80% of the longest measure. A piece of tape placed 80% the distance of the longest measure will give you a target that you must lunge past during actual testing.

After you demonstrate proper technique (past literature has recommended three to five practice trials), you will perform as many repetitions as you can properly in 30 seconds for each test. We will test both legs to determine a limb symmetry index to compare your measurements to that of other subjects.

**Strength Assessment**
We will measure strength using a hand-held dynamometer (a spring-like gauge that measures muscle strength). We will measure three muscles:

*Muscle #1 (Hip Abductors)*
You will lay on your side so that we may measure the muscle on the outside of your leg. You will lay on your side with both legs parallel to each other, with the test leg on top. We will place the dynamometer on the outside of your thigh just above your knee. You will push outward into the dynamometer for one practice trial and three test trials. You will hold the muscle contraction for 5 seconds and rest 1 minute between each trial. During this time, we will simultaneously gather surface electromyographic (EMG) data to provide a measure of the maximal amount of force you can produce. EMG data will
provide a representation of the electrical activity within your muscles when you muscles are working actively. This measurement is necessary so that we can compare your muscle activity to that of other subjects.

**Muscle #2 (Hip External Rotators)**
You will then sit in a position with the hips and knees bent 90 degrees. We will place the dynamometer on the inside of the test leg just above the ankle. You will push inward against the dynamometer for one practice trial and three test trials. You will hold the muscle contraction for 5 seconds and rest 1 minute between each trial.

**Muscle #3 (Knee Extensors)**
You will then sit in a position with the hips bent to 90 degrees and your test knee bent to 60 degrees. We will place the dynamometer on the front of your leg just above your ankle. You will push outward against the dynamometer for one practice trial and three test trials. You will hold the muscle contraction for 5 seconds and rest 1 minute between each trial. During this time, we will simultaneously gather EMG data to provide a measure of the maximal amount of force you can produce. This measurement is necessary so that we can compare your muscle activity to that of other subjects.

**Motion Analysis and EMG Preparation:**
Next, we will prepare you for motion analysis and electromyographic (EMG) data collection. Motion analysis will provide a means for evaluating motion of your hip and knee joints during a stair-stepping activity and EMG will measure electrical activity of your muscles as they contract while performing the task. You will have approximately 35 reflective markers placed on certain landmarks of your body to allow the motion analysis system to record hip and knee movement. You will also have five conductive (sticky) pads placed on your leg. The conductive pads will measure the amount of electrical activity in your muscles while you are going up and down the stair platform. Hair overlying the skin will be shaved, if necessary, and the skin will be cleaned thoroughly with an abrasive pad and alcohol swab prior to application of all conductive pads.

**Stair-stepping Task:**
You will perform five practice and five test trials of a stair-stepping that you will complete at your own walking pace. You will go up and down two 20-cm (8 inch) high steps, making sure that the test leg lifts your body on the first step and lowers your body on the third step. We will collect motion analysis and EMG data during this activity.

You will return to the laboratory within 5 to 7 days for repeat testing. The principal investigator will provide you an appointment time that will be convenient for you to return to the laboratory during this 5 to 7 day period. The principal investigator will also contact you by either telephone or e-mail (according to your preference of being reminded) to remind you of the second testing day. You will perform all tests in the same manner described above. You will complete another visual analog scale because differences in pain may affect your functional test performance. You may be excluded if you have a significant difference in the visual analog scale pain assessment on the second testing day.
ARE THERE REASONS WHY YOU SHOULD NOT TAKE PART IN THIS STUDY?

You should not participate in this study if you have had knee surgery, are participating in a lower extremity rehabilitation program at this time, have lower extremity injury/s other than patellofemoral pain syndrome, have an allergy to tape, and are under 18 years of age or over 35 years of age. If you are a control subject, then you should not participate if you have had any lower extremity injury or under 18 years of age or over 35 years of age.

WHAT ARE THE POSSIBLE RISKS AND DISCOMFORTS?

Risks are minimal in this study. You may experience a skin reaction from the adhesive pads or joint or muscle soreness from activities that you will perform. To reduce the possibility of muscle soreness and joint fatigue, you will perform a warm-up activity prior to testing. We will also ask that you refrain from physical activity, other than normal walking, for a 24-hour period following the end of the study.

There is always a chance that exercise may harm you. We will do everything we can to keep you from being harmed. Additionally, you may experience a previously unknown risk or side effect.

WILL YOU BENEFIT FROM TAKING PART IN THIS STUDY?

You will not get any personal benefit from taking part in this study.

DO YOU HAVE TO TAKE PART IN THE STUDY?

If you decide to take part in the study, it should be because you really want to volunteer. You can stop at any time during the study. If you decide not to take part in this study, your decision will have no effect on your grades or standing at the University of Kentucky.

IF YOU DON’T WANT TO TAKE PART IN THE STUDY, ARE THERE OTHER CHOICES?

If you do not want to be in the study, there are no other choices except not to take part in the study.

WHAT WILL IT COST YOU TO PARTICIPATE?

There are no costs associated with your participation other than the time committed for participation.

WHO WILL SEE THE INFORMATION THAT YOU GIVE?

We will keep private all research records that identify you to the extent allowed by law.
Your information will be combined with information from other people taking part in the study. When we write about the study to share it with other researchers, we will write about the combined information we have gathered. You will not be identified in these written materials. We may publish the results of this study; however, we will keep your name and other identifying information private.

We will make every effort to prevent anyone who is not on the research team from knowing that you gave us information, or what that information is. For example, your name will be kept separate from the information you give, and these two things will be stored in different places under lock and key. You should know, however, that there are some circumstances in which we may have to show your information to other people. For example, the law may require us to show your information to a court.

**CAN YOUR TAKING PART IN THE STUDY END EARLY?**

If you decide to take part in the study, you still have the right to decide at any time that you no longer want to continue. You will not be treated differently if you decide to stop taking part in the study.

The individuals conducting the study may need to withdraw you from the study. This may occur if you are not able to follow the directions they give you or if they find that your being in the study is more risk than benefit to you.

**WHAT HAPPENS IF YOU GET HURT OR SICK DURING THE STUDY?**

If you believe you are hurt or if you get sick because of something that is done during the study, you should call Lori A. Bolgla at 859-333-6356 immediately. It is important for you to understand that the University of Kentucky will not pay for the cost of any care or treatment that might be necessary because you get hurt or sick while taking part in this study. That cost will be your responsibility. Also, the University of Kentucky will not pay for any wages you may lose if you are harmed by this study.

Medical costs that result from research-related harm can not be included as regular medical costs. The University of Kentucky is not allowed to bill your insurance company. You should ask your insurer if you have any questions about your insurer’s willingness to pay under these circumstances.

**WILL YOU RECEIVE ANY REWARDS FOR TAKING PART IN THIS STUDY?**

You will receive $20 for each testing session (for a maximum of $40) to cover your time and parking expenses associated with participating in this study. If you are excluded from the second testing day (because of a significant change in pain), you will not receive the second $20 payment (you will only be paid for the first day of testing).
WHAT IF YOU HAVE QUESTIONS?

Before you decide whether to accept this invitation to take part in the study, please ask any questions that might come to mind now. Later, if you have questions about the study, you can contact the investigator, Lori A. Bolgla at 859-333-6356. If you have any questions about your rights as a volunteer in this research, contact the staff in the Office of Research Integrity at the University of Kentucky at 859-257-9428 or toll free at 1-866-400-9428. We will give you a copy of this consent form to take with you.

WHAT ELSE DO YOU NEED TO KNOW?

You will be told if any new information is learned which may affect your condition or influence your willingness to continue taking part in this study.

______________________________  _____________________________
Signature of person agreeing to take part in the study       Date

______________________________
Printed name of person agreeing to take part in the study

______________________________  _____________________________
Name of person providing information to subject       Date

______________________________
Signature of Investigator
Do you have Knee Pain?

Researchers at the University of Kentucky College of Health Sciences Department of Rehabilitation Sciences are conducting a clinical research study to investigate the influence of the hip on patellofemoral pain syndrome.

You may be able to participate if you:
- are a female between the ages of 18 and 35;
- are generally healthy;
- have no history of lower extremity injury or surgery;
- have patellofemoral pain; and
- complain of knee pain when walking up or down steps.

Persons who qualify will be compensated and will participate in two 90 minute sessions. For more information, contact Lori A. Bolgla, P.T., at (859) 333-6356 or by e-mail at labolg2@uky.edu.
Appendix C

Measurement Tool Instrumentation

Hand-held Dynamometer

All isometric strength testing was performed using the Commander PowerTrack II™ (JTech Medical, Salt Lake City, UT) hand-held dynamometer (HHD). This digital strain-gauge dynamometer has a maximum load cell capacity of 125.0 lb (556.3 N), with a manufacturer-reported accuracy of 99%. The dynamometer’s calibration was confirmed prior to the study by placing known weights on the HHD and comparing this to the HHD’s reported weight. Accuracy was verified after every tenth testing session.

Surface EMG

A 16-channel Myosystem 1400 EMG system (Noraxon USA, Inc., Scottsdale, AZ) recorded muscle activity. Unit specifications for this system included a common ratio rejection ratio exceeding 100 dB, an amplifier gain of 1000, and input impedance exceeding 10 Mohm. EMG data were sampled at 960 Hz and initially band pass filtered from 10 to 1000 Hz. They were then converted from analog to digital using a 12-bit A/D board (National Instruments, Austin, TX), synchronized with the video data, and stored on a personal computer.

Motion Analysis

Video data were recorded using 7 high-speed, high-resolution (320 X 240) video cameras (Motion Analysis Corporation, Santa Rosa, CA) operating at 60 Hz. A three-dimensional volume of approximately 2.0 m X 1.2 m X 1.8 m was calibrated in accordance with procedures recommended by Motion Analysis Corporation. According to the manufacturer’s manual, the calibration process calculates eleven calibration coefficients, which implicitly define the configuration of a particular view. The calibration coefficients can define the path of an optical ray from the target (marker) to the camera through the object-space. The 3-dimensional position of a target can be determined when rays from 2 cameras intersect simultaneously in space. The tracking process uses data from intersecting optical rays from different views of the same event. EVaRT employs a “best fit” tracking algorithm using only good camera views. The manufacturer-reported accuracy for detection of marker position has been reported as ± 1 mm. Video and analog data were collected using the EVaRT 4.2 hardware-software system (Motion Analysis Corp.) and stored on a personal computer.
Procedures for Processing EMG and Kinematic Data

Surface EMG Data
1. Muscle Activation Amplitudes

   EMG data were initially band pass filtered at 10 to 1000 Hz (during data collection using the Myosystem 1400 EMG system) and further band pass filtered at 20 to 480 Hz (during data processing) using Datapac Software (Run Technologies). For purposes of determining activation amplitudes, data were converted to root mean square (RMS) values using a 55 msec time constant (Sheehy et al., 1998). Resting and MVIC data were processed in an identical manner and used to express activation amplitudes as a percent MVIC. The MVIC for each muscle was determined by calculating the RMS amplitude recorded over a 500 millisecond (ms) window (Bamman, Ingram, Caruso, & Greenisen, 1997). This amount was assumed to represent 100% isometric muscle activity for each muscle.

2. Muscle Activation Onsets

   For purposes of determining activation onsets, data were full wave rectified and low pass filtered at 50 Hz (Cowan et al., 2000). A muscle onset was defined as the point in which the signal deviated by more than 3 standard deviations, for a minimum of 25 ms, over the baseline level taken 200 ms before the trial began.

Kinematic Data

   Video data were sampled at 60 Hz, tracked, and smoothed using a fourth order Butterworth zero phase-lag low-pass filter, with a cutoff frequency of 6 Hz, using EVaRT 4.2 software (Motion Analysis Corporation). Processed data were then analyzed using OrthoTrak 5.0 software (Motion Analysis Corporation). This software used a joint coordinate system based on work by Grood and Suntay (1983) to describe knee motion. Based on this convention, joint motion was described as follows:

<table>
<thead>
<tr>
<th>Motion</th>
<th>Positive</th>
<th>Negative</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hip transverse plane</td>
<td>Internal rotation</td>
<td>External rotation</td>
</tr>
<tr>
<td>Hip frontal plane</td>
<td>Adduction</td>
<td>Abduction</td>
</tr>
<tr>
<td>Knee frontal plane</td>
<td>Adduction</td>
<td>Abduction</td>
</tr>
<tr>
<td></td>
<td>(Varus)</td>
<td>(Valgus)</td>
</tr>
</tbody>
</table>
Appendix E

Summary of Pain, Functional Performance Test, and Strength Testing

Pain
Researchers (Chesworth, Culham, Tata, & Peat, 1989; Crossley, Bennell et al., 2004) have determined the reliability for using a 10-cm visual analog scale to measure subjective pain. The extreme left side of the visual analog scale stated “no pain” whereas the extreme right side stated “worse pain imaginable.” Subjects placed a mark on the scale that most likely describes their usual pain over the previous week. The measured distance from the extreme left side of the scale to the subject’s mark was used for statistical analysis.

Functional Performance Tests
Lower extremity function was assessed using the step-down and anteromedial lunge functional performance tests (Loudon et al., 2002). For the step-down test, subjects stood on an 8” step using the test extremity, lower their body enough to brush the foot of the opposite lower extremity on the floor (in front of the step), and returned to full knee extension. For the anteromedial lunge test, subjects stood behind a start line and lunged (to 90° knee flexion) forward and across midline with the uninjured lower extremity three times. I measured the distance from the start line to the back of the heel for each trial and took 80% of the longest measure. A piece of tape placed 80% the distance of the longest measure gave subjects a target they lunged past (the back of the test heel was placed in front of the target) during this test. For each test, the number of repetitions completed for each lower extremity in a 30-second time period was counted.

Strength Testing Protocol
1. Hip Abductor Strength
Subjects were positioned in sidelying with the test leg in a neutral position by placing pillows between the lower extremities. The HHD was placed over the lateral femoral condyle and secured with a Velcro strap (Ireland et al., 2003). Subjects produced maximal isometric contractions using the “make” test (Andrews et al., 1996; Bohannon, 1997). They generated maximum force over a 2-second period and maintained this force for an additional 5 seconds. Subjects performed one practice (Andrews et al., 1996;
Bohannon, 1997) and three test trials of a maximal isometric hip abductor contraction, with a 30-second rest period between trials.

2. Hip External Rotation Strength

Hip external rotator isometric strength was measured using methods previously described in the literature (Ireland et al., 2003; Jaramillo, Worrell, & Ingersoll, 1994; Niemuth et al., 2005). Subjects sat with the hips and knees in 90 degrees of flexion. The HHD was placed just proximal to the medial malleolus and secured with a strap. Subjects were instructed to pull against the strap and the primary investigator ensured that subjects did not simultaneously flex or adduct the hip. As described above, they generated force using the “make” test (Andrews et al., 1996; Bohannon, 1997). Subjects performed one practice and three test trials of a maximal isometric hip external rotator contraction, with a 30-second rest period between trials.

3. Knee Extensor Strength

Knee extensor isometric strength was measured using methods described previously in the literature (Mohr et al., 2003; Powers, 2000). Subjects were positioned with the hip in 90 degrees of flexion and the knee in 60 degrees of flexion. The HHD was placed just proximal to the malleoli and secured with a strap. As described above, they generated force using the “make” tests (Andrews et al., 1996; Bohannon, 1997). Subjects performed one practice and three test trials of a maximal isometric quadriceps contraction, with a 30-second rest period between trials. I chose this position because asymptomatic females can generate maximum isometric force at 60 degrees knee flexion (Lieb & Perry, 1971).
A Priori Power Analysis

*A priori* power analysis using $\alpha = .05$ and $\beta = .20$ was used to determine the number of subjects required to protect against type I and II errors. For FPT, Loudon et al. (2002) found significant differences using 15 subjects per group. For isometric strength measures, 15 subjects per group would be adequate based on strength difference of 15% (Ireland et al., 2003) and previously reported variability (Bohannon, 1997). For EMG amplitudes, Mohr et al. (2003) reported significant differences using 13 PFPS and 11 control subjects in a study that examined EMG amplitudes during a similar stair-stepping task. For quadriceps timing differences, a similar study conducted at the UK Musculoskeletal Lab found significant VM to VL timing differences using 14 PFPS and 14 control subjects (Boling, Bolgla, Mattacola, et al., 2004, unpublished data). Brindle et al. (2003) reported significant GM to VM and VL timing differences in a group of 16 PFPS and 12 control subjects.

Limited information existed regarding kinematic parameters specific to subjects diagnosed with PFPS. From clinical experience, I believe that a 10 degree difference in hip abduction, hip internal rotation, and knee abduction would discriminate between subjects diagnosed with and without PFPS. Using this difference and a standard deviation of $\pm$ 10 degrees, a minimum of 16 subjects would be needed for each group. Based on all previous data, the current study included 18 subjects in each group.
Normalizing of Stance Phase of Stair Descent for EMG Data

The stance phase of stair descent was divided into the following intervals: 1) loading response, 2) single leg stance, and 3) preswing (Mohr et al., 2003). Loading response began at the initial point where any part of the ipsilateral foot contacted the step and ended as subjects lifted the contralateral foot off the previous step (e.g., initial double leg stance). Single leg stance occurred when the test extremity supported the entire body mass during stair descent. Preswing began when any part of the contralateral foot contacted the ground and ended as subjects lifted the test extremity’s foot off the stair (e.g., terminal double leg stance).

OrthoTrak software (Motion Analysis Corp.) ensemble averaged (5 trials for each subject) and normalized data to 100% of the stair descent cycle. The software also summarized the percent of the entire cycle spent during loading response, single leg stance, and total stance. Based on this information, preswing was determined by subtracting the percent for loading response and single leg stance from total stance [% preswing = % total stance – (% loading response + % single leg stance)]

The Table below summarizes the percent of the stair descent cycle associated with loading response, single leg stance, and preswing for control and PFPS subjects. Independent t-tests were then conducted to test for the presence of group differences in these intervals. All subjects descended the stairs in a similar manner and between-subject variability was low. Therefore, the average percent time that all subjects required during each interval (all data combined) was used to normalize EMG data.

Based on these average values, stance phase was divided and expressed as a percentage of total stair descent in the following intervals:

<table>
<thead>
<tr>
<th>Stance Phase</th>
<th>Percentage of Stair Descent</th>
</tr>
</thead>
<tbody>
<tr>
<td>Loading Response</td>
<td>0% - 7%</td>
</tr>
<tr>
<td>Single Leg Stance</td>
<td>8% - 46%</td>
</tr>
<tr>
<td>Preswing</td>
<td>47% - 58%</td>
</tr>
</tbody>
</table>
Table.
Summary of Average Time (expressed as a percent of the stair descent cycle) for Intervals of the Stance Phase of Stair Descent

<table>
<thead>
<tr>
<th>Interval</th>
<th>PFPS Mean</th>
<th>PFPS SD†</th>
<th>Control Mean</th>
<th>Control SD†</th>
<th>p – value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Load Response</td>
<td>6.6</td>
<td>1.1</td>
<td>7.5</td>
<td>1.6</td>
<td>.07</td>
</tr>
<tr>
<td>Single Leg Stance</td>
<td>39.8</td>
<td>1.7</td>
<td>38.7</td>
<td>1.7</td>
<td>.09</td>
</tr>
<tr>
<td>Preswing</td>
<td>11.6</td>
<td>1.7</td>
<td>12.8</td>
<td>2.5</td>
<td>.11</td>
</tr>
</tbody>
</table>

† SD = standard deviation
Appendix H

Data Collection Sheet

<table>
<thead>
<tr>
<th>Subject #</th>
<th>Age</th>
<th>Hgt.</th>
<th>Wgt.</th>
</tr>
</thead>
</table>

Lower extremity Left Right
Duration of symptoms

Do you have a history of significant lower extremity injury other than patellofemoral pain syndrome (except for control subjects)? Yes No

Shoe Size
Distance from greater trochanter to lateral femoral condyle
Distance from lateral knee joint line to lateral malleolus

Visual Analog Scale Measurement
Day 1 Day 2

Functional Performance Tests

<table>
<thead>
<tr>
<th>Test Order</th>
<th>Test</th>
<th>Day 1</th>
<th>Day 2</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Step-down</td>
<td>Left</td>
<td>Right</td>
</tr>
<tr>
<td></td>
<td>Lunge</td>
<td>Left</td>
<td>Right</td>
</tr>
</tbody>
</table>

Distance used for Lunge test
## Strength Measurements

<table>
<thead>
<tr>
<th>Test Order</th>
<th>Muscle</th>
<th>Day 1</th>
<th>Day 2</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Hip abductors</td>
<td>Trial 1</td>
<td>Trial 2</td>
</tr>
<tr>
<td></td>
<td>Hip external rotators</td>
<td>Trial 1</td>
<td>Trial 2</td>
</tr>
<tr>
<td></td>
<td>Knee extensors</td>
<td>Trial 1</td>
<td>Trial 2</td>
</tr>
</tbody>
</table>

### Surface Electrode Placement (in cm):
- **Gluteus Medius**: 1/3 the distance from iliac crest and greater trochanter
- **Vastus Medialis Oblique**: Distance superior to patella, Distance medial
- **Vastus Lateralis**: Distance superior to patella, Distance lateral

### Muscle Onset Timings (conversion to time):

<table>
<thead>
<tr>
<th>Day</th>
<th>Trial 1</th>
<th>Trial 2</th>
<th>Trial 3</th>
<th>Trial 4</th>
<th>Trial 5</th>
</tr>
</thead>
<tbody>
<tr>
<td>Day 1</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Day 2</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

### Muscle Onset Timings Differences for Day 1:

<table>
<thead>
<tr>
<th></th>
<th>VMO-GM</th>
<th>VL-GM</th>
<th>VL-VMO</th>
</tr>
</thead>
<tbody>
<tr>
<td>Trial 1</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Trial 2</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Trial 3</td>
<td></td>
<td></td>
<td></td>
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<tr>
<td>Trial 4</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Trial 5</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
Muscle Onset Timings Differences for Day 2:

<table>
<thead>
<tr>
<th></th>
<th>VMO-GM</th>
<th>VL-GM</th>
<th>VL-VMO</th>
</tr>
</thead>
<tbody>
<tr>
<td>Trial 1</td>
<td>________</td>
<td>________</td>
<td>________</td>
</tr>
<tr>
<td>Trial 2</td>
<td>________</td>
<td>________</td>
<td>________</td>
</tr>
<tr>
<td>Trial 3</td>
<td>________</td>
<td>________</td>
<td>________</td>
</tr>
<tr>
<td>Trial 4</td>
<td>________</td>
<td>________</td>
<td>________</td>
</tr>
<tr>
<td>Trial 5</td>
<td>________</td>
<td>________</td>
<td>________</td>
</tr>
</tbody>
</table>

Muscle Amplitude (frame number):

<table>
<thead>
<tr>
<th></th>
<th>Begin</th>
<th>End</th>
<th>Duration</th>
</tr>
</thead>
<tbody>
<tr>
<td>Day 1</td>
<td>Day 2</td>
<td>Day 1</td>
<td>Day 2</td>
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<tr>
<td>Day 1</td>
<td>Day 2</td>
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<td>Day 1</td>
<td>Day 2</td>
<td>Day 1</td>
<td>Day 2</td>
</tr>
</tbody>
</table>

Muscle Amplitude (conversion to time):

<table>
<thead>
<tr>
<th></th>
<th>Begin</th>
<th>End</th>
<th>Duration</th>
</tr>
</thead>
<tbody>
<tr>
<td>Day 1</td>
<td>Day 2</td>
<td>Day 1</td>
<td>Day 2</td>
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<tr>
<td>Day 1</td>
<td>Day 2</td>
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<td>Day 2</td>
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<td>Day 2</td>
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<tr>
<td>Day 1</td>
<td>Day 2</td>
<td>Day 1</td>
<td>Day 2</td>
</tr>
</tbody>
</table>
Calibration:

<table>
<thead>
<tr>
<th>Muscle</th>
<th>Zero Off-Set (rest)</th>
<th>1-volt Scale (maximum)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Day 1</td>
<td>Day 2</td>
</tr>
<tr>
<td>Gluteus Medius</td>
<td>______</td>
<td>______</td>
</tr>
<tr>
<td>Vastus Medialis Oblique</td>
<td>______</td>
<td>______</td>
</tr>
<tr>
<td>Vastus Lateralis</td>
<td>______</td>
<td>______</td>
</tr>
</tbody>
</table>

Muscle Amplitudes (% MVIC):

<table>
<thead>
<tr>
<th></th>
<th>Load Response</th>
<th>Midstance</th>
<th>Pre-swing</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>GM</td>
<td>VMO</td>
<td>VL</td>
</tr>
<tr>
<td>Day 1</td>
<td>______</td>
<td>______</td>
<td>______</td>
</tr>
<tr>
<td>Day 2</td>
<td>______</td>
<td>______</td>
<td>______</td>
</tr>
</tbody>
</table>
Subject # _______

Testing Day 1 2

Indicate your greatest level of knee discomfort during the past week

No pain at all | Worse pain imaginable
Appendix J

Summary of Procedures

**Warm-up Procedures**
1. Explain the procedures and obtain informed consent
2. Complete 10-cm VAS
3. Ride a stationary bike for 3 minutes at a submaximal speed
4. Obtain the following demographic information:
   - Age
   - Duration of symptoms
   - Height
   - Weight
   - Thigh length (greater trochanter to distal femur at the lateral knee joint line)
   - Tibia length (proximal tibia at the lateral knee joint line to lateral malleolus)

**Functional Performance Tests**

**Step-Down Test**
1. Subjects stand on an 8” step
2. Subjects step forward and down toward the floor
3. The down limb only brushes the floor with the heel and then returns to full knee extension (counts as 1 repetition)
4. Count the number of repetitions subjects can perform in 30 seconds
Anteromedial Lunge
1. Line the subject behind a start line
2. Subject lunges forward with the uninvolved leg so that the front knee flexes to 90° and crosses midline
3. Record the distance from the start line to the back of the heel of the lead leg
4. Subject performs this task 3 times
5. Calculate 80% of the maximal distance and mark with a piece of tape to provide a target for testing purposes
6. Count the number of lunges that a subject can perform in 30 seconds

Manual Muscle Testing and EMG Normalization
1. Don EMG electrodes to GM, VMO, and VL
2. Take a 3-second resting file
3. Take MVIC simultaneously during manual muscle testing

Hip Abductors
1. Place subjects in 10° abduction with pillows between thighs
2. Secure the dynamometer just proximal to the lateral condyle
3. Allow subjects 1 practice using the “make test” gradually generate maximal contraction over a 2-second period and hold for 5 seconds
4. Repeat the process for 3 trials
5. Rest 1 minute between trials

Hip External Rotation
1. Subjects sit with the hip and knees in 90° flexion with the hips and trunk stabilized with straps
2. Place the dynamometer 2-cm proximal to the medial malleolus
3. Allow subjects 1 practice using the “make test” gradually generate maximal contraction over a 2-second period and hold for 5 seconds
4. Repeat the process for 3 trials
5. Rest 1 minute between trials
Knee Extension
1. Subjects sit with the hip in 90° flexion and knees in 60° flexion with the hips and trunk stabilized with straps
2. Place the dynamometer 2-cm proximal to the malleoli
3. Allow subjects 1 practice using the “make test” gradually generate maximal contraction over a 2-second period and hold for 5 seconds
4. Repeat the process for 3 trials
5. Rest 1 minute between trials

Kinematic and EMG Data Collection
1. Don reflective markers
2. Allow subjects to practice the task 5 times to a metronome set at 96 bpm
3. Place a piece of tape on the walkway where subjects will use the test extremity to hit the second and third steps
4. Confirm that cameras read all markers
5. Take a 1-second static trial
6. Remove all knee and ankle markers
7. Collect a minimum of 10 walking trials
Appendix K

### Participant Sign-up Sheet

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REFERENCES


Lori Ann Bolgla, PT, MS, ATC

Date of birth: December 12, 1962

Place of birth: Augusta, Georgia

Education


Medical College of Georgia, Augusta, GA. Graduated summa cum laude with a Bachelor of Science- Physical Therapy, September 1991- June 1993.

University of Georgia, Athens, GA. Master of Accounting, June 1984- June 1985.

University of Georgia, Athens, GA. Graduated cum laude with a Bachelor of Business Administration- Accounting, September 1980- June 1984.

Professional Positions

University of Kentucky/ Drayer Physical Therapy Institute, Research Assistant, January 2005-present

University of Kentucky, Rehabilitation Sciences Doctoral Program, Graduate Assistant, August 2002-December 2004.

Medical College of Georgia Center for Sports Medicine, Augusta, GA. Senior physical therapist, July 1997- July 2002.

Medical College of Georgia Hospital and Clinics, Augusta, GA. Senior physical therapist, November 1996- July 1997.


Hitchcock Rehabilitation Center, Aiken, SC. Staff physical therapist, June 1993- November 1996.
Scholastic and Professional Honors

2006 Sports Physical Therapy Section “Excellence in Research” Award
2004 Recipient of Dean’s Special Award
2003-2004 University of Kentucky Academic Excellence Scholarship
Alpha Eta Honor Society
Who’s Who Among Students in American Universities and Colleges
Beta Alpha Psi Accounting Honor Society
Golden Key Honor Society
Beta Gamma Sigma Business Honor Society

Publications - Manuscripts


Bolgla LA, Keskula DR: “Intrarater Reliability of Functional Performance Tests,”

Publications – Book Chapters

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Lori A. Bolgla