Knee Joint Biomechanics in People with Medial Compartment Knee Osteoarthritis

By

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Abstract

**Background:** Sagittal plane external flexion moment in early stance has been reported to be both higher and lower in people with medial knee OA compared to control subjects.

**Purpose:** Sagittal plane knee joint kinetics and kinematics were compared between people with medial compartment OA and healthy subjects. Subjects with OA and low pain scores were also compared to those with OA and moderate pain scores. **Subjects:** Forty people with medial knee OA and 40 age and sex matched control subjects.

**Method:** Knee alignment and OA severity were measured from radiographs. Frontal and sagittal plane moments and knee flexion angle during gait were measured using a three dimensional motion analysis system and force plates. The Western Ontario McMaster Universities Osteoarthritis Index (WOMAC) was used to measure pain; this score was used to divide the OA group into low pain and moderate pain OA groups. The relationship between knee flexion moment in early stance and gait speed, knee flexion angle and pain was also determined. **Results:** Subjects with OA had lower gait speed and cadence, and higher double limb support time. Peak knee adduction moment (first 50% of the gait cycle) was higher in the OA group. The knee flexion moment in early stance did not differ between groups; however the knee extension moment in late stance was lower in the knee OA group. The moderate pain group walked more slowly than the low pain group. The knee adduction moment, sagittal plane moments and knee flexion angle in stance did not differ between OA groups. A positive correlation was found between knee flexion moment and knee flexion angle in early stance in the knee OA group; the correlations between knee flexion moment and pain and gait speed were not significant.

**Conclusion:** No differences were found in the knee flexion moment or stance phase knee flexion angle between the groups compared in this study, and therefore the results do not
contribute to resolution of the controversy in the literature. Participants with knee OA in this study had relatively mild to moderate disease severity, which may have contributed to the non-significant findings.
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Chapter 1

1.1 Introduction

Osteoarthritis (OA) is a chronic degenerative disease affecting synovial joints. Joints most commonly affected include the knee, hip, and joints of the spine and hand, although any synovial joint can be involved. Joint osteoarthritic changes may result from excessive wear and tear of the articular cartilage alone.\textsuperscript{1} However, the etiology of OA is more commonly viewed as a multifactorial process resulting from both mechanical (joint loading), and biochemical (metabolic) events that destabilize the normal coupling of degradation and synthesis of articular cartilage chondrocytes, extracellular matrix and subchondral bone.\textsuperscript{2-4} These alterations lead to softening, fibrillation, ulceration, and loss of joint articular cartilage. Bony changes include sclerosis and eburnation of subchondral bone, and osteophyte and subchondral cyst formation.\textsuperscript{1,5} Progression of joint damage can lead to incongruity of the joint articular surfaces, loss of ligament stability, joint axis deviations, and ultimately joint deformation.\textsuperscript{5,6} OA is considered a chronic process that develops over years.

Osteoarthritis can be diagnosed by radiographic changes and/or by typical clinical symptoms.\textsuperscript{7} Radiographic findings which identify OA include osteophyte formation along the joint margins, joint space narrowing, subchondral sclerosis and cyst formation.\textsuperscript{8} Presence of osteophytes is the most specific radiographic marker of OA, although osteophytes are not present in early disease. The classification criteria for symptomatic OA established by The American College of Rheumatology include pain on
activity and/or at rest, joint tenderness, limitation of movement, occasional effusion, and variable degrees of inflammation.9

Two major risk factors for OA disease occurrence are age and sex.2,6 OA is the most prevalent disorder among adults 65 years and older. However, OA has also been reported in younger adults with a steep rise in prevalence after age 40.6 Sex specific differences are also evident in OA distribution. Before 50 years of age, OA prevalence is higher in men than in women and the opposite is true after 50 years of age.2 Joint involvement also varies according to the sex of an individual.2 Hand and knee OA are particularly prevalent among older women, while hip OA is more common in older men.2,10,11

In 1990, it was reported that OA was the eighth leading non-fatal burden of disease in the world accounting for 2.8% of total years with disability.12 Disability from OA has been largely attributed to OA of the knee or hip joints. In older adults, OA of lower extremity joints causes greater dependency in walking, stair climbing, and other lower extremity tasks than any other disease. The risk of disability due to knee OA is equivalent to the risk caused by cardiac disease and is greater than the risk due to other medical conditions in older adults.13 Coexistence of knee OA with other health disorders increases the disability risk.14 In 1997, the World Health Organization reported that knee OA was the fourth leading cause of disability in older women and the eighth most important cause of disability in older men.12 In 2001, it was estimated that knee OA affected 13% of adults who were 60 years of age and older. A quarter of this group were severely disabled.15
Loading through the knee joint during walking and other activity is believed to be a factor contributing to the pathomechanics of knee OA.\textsuperscript{16, 17} The knee joint is a complex structure with three joint compartments, including the medial and lateral tibio-femoral compartments and patella-femoral joint. During walking, forces across the knee joint are not transmitted equally between the medial and lateral compartments. The load on the medial compartment is approximately 2.5 times greater than that on the lateral compartment.\textsuperscript{18} Consistently higher loads on the medial compartment have been shown to result in degenerative changes of the articular cartilage.\textsuperscript{2-4} This imbalance in the load distribution may explain the higher prevalence of medial compartment involvement (75\%) reported in subjects with knee OA relative to the lateral compartment (25\%).\textsuperscript{19} Furthermore, with disease progression, increased mechanical load and damage to the medial side of the knee joint has been associated with increasing knee varus alignment, further loading the medial compartment.\textsuperscript{20, 21}

Knee OA pathomechanics can be better understood by analyzing the dynamic joint load in the frontal and sagittal planes using three dimensional gait analyses. The knee adduction moment in the frontal plane is a reliable measure of the load on the medial and lateral compartments of the knee joint during gait.\textsuperscript{16, 17, 22} In the gait cycle of healthy adults, there is an initial brief knee abduction moment at initial contact; this is followed by a knee adduction moment through the remainder of stance phase. There is an initial peak in the first 50\% of stance phase and a second peak in the latter half of stance. The magnitude of the adduction moment is lower in the period between these peaks.\textsuperscript{23} During walking, individuals with medial compartment knee OA have higher knee adduction moments throughout stance compared to healthy control subjects.\textsuperscript{16, 22}
Furthermore, a high knee adduction moment has been reported to be a risk factor for both knee OA onset and progression.\textsuperscript{16, 17}

Sagittal plane knee joint moment profiles have also been studied in people with knee osteoarthritis. In the normal gait cycle, there is an initial external extension moment at the knee following initial contact.\textsuperscript{24} This is followed by an external knee flexion moment in early stance phase as the knee flexes and the limb accepts weight. An external extension moment occurs in late stance as the knee extends and the body moves forward over the fixed foot.\textsuperscript{24} Studies investigating sagittal plane moments during gait in people with knee osteoarthritis have reported conflicting findings. Several authors have reported that individuals with medial knee OA have lower external knee flexion moments in early stance compared to healthy control subjects.\textsuperscript{25, 26, 31} Other authors have reported higher knee flexion moments in early stance compared to healthy control subjects\textsuperscript{18, 27, 28} Some studies reported no differences in the early knee flexion moment between knee OA and control groups.\textsuperscript{22, 29, 30}

Findings related to the knee extension moments are more consistent. There is some evidence that individuals with knee OA have lower knee extension moments in the initial stance phase than healthy control subjects.\textsuperscript{29, 31} In late stance, authors have reported either lower knee extension moments in people with medial compartment knee OA compared to healthy older adults or no difference between groups.\textsuperscript{18, 26, 30, 31}

The conflicting findings regarding the external flexion moment may be attributed to differences in knee flexion angle during early stance. Lower knee flexion moments may be a consequence of reducing the knee flexion angle during the stance
phase of a gait cycle.\textsuperscript{31} Astephen et al\textsuperscript{31} reported a lower knee flexion angle for individuals with knee OA compared to healthy control subjects during early stance phase of gait (p<0.05). Studies that reported high knee flexion moments in individuals with medial knee OA found that the stance phase knee flexion angle was similar to that of the healthy control subjects (p>0.05).\textsuperscript{18,28}

Knee OA severity may be a factor in determining the magnitude of flexion and extension moments during the stance phase of a gait cycle.\textsuperscript{29,32,33} Researchers have suggested that individuals with moderate medial knee OA would have similar flexion and extension moments to that of the healthy control subjects because of low joint pain levels and a normal knee range of motion.\textsuperscript{29,32,33} However, individuals with severe knee OA may walk more slowly and minimize flexion and extension moment magnitude in an attempt to reduce joint pain.\textsuperscript{29,32,33}

Based on the conflicting findings, it is unclear whether people with medial compartment knee OA have higher or lower sagittal plane external knee flexion moments during the early stance phase of a gait cycle compared with the healthy control subjects. It is also unclear whether the flexion moment is related to severity of OA, to the knee flexion angle or gait speed in early stance phase. Therefore, the primary purpose of the study was to investigate sagittal plane moments and knee kinematics to determine if there are differences between people with knee OA and healthy control subjects. Secondly, the study determined whether there were differences in sagittal plane moments and knee kinematics between OA subjects with low pain scores compared to those with moderate pain scores. The groups were divided based on knee pain severity.
Specific objectives were:

1. To determine if stance phase external flexion and extension moments differ between people with knee OA and healthy controls and whether these moments differ between people with low versus moderate pain scores in knee OA subjects. It was hypothesized that external knee flexion and extension moments would be lower in people with knee OA compared to healthy controls and lower in the OA group with low pain than in the OA group with moderate pain.

2. To determine if knee flexion angle in early stance differs between people with knee OA and healthy controls and between those with low versus moderate pain knee OA groups. It was hypothesized that knee flexion angles would be lower in those with OA compared to healthy controls and in those with low versus moderate pain knee OA groups.

3. To investigate the relationship between the knee flexion moment in early stance and the stance phase knee flexion angle in subjects with OA. It was hypothesized that the knee flexion moment would be positively correlated with stance phase flexion angle.

4. To investigate the relationship between knee OA severity as determined by pain scores and knee flexion moment in early stance phase in subjects with knee OA. It was hypothesized that there would be a significant negative correlation between these variables.

5. To investigate the relationship between knee flexion moment in early stance phase and gait speed in subjects with knee OA. It was hypothesized that there would be a significant positive correlation between these variables.
Chapter 2

Literature Review

2.1 Etiology of Knee Osteoarthritis

The following review will provide information regarding the risk factors associated with onset and progression of osteoarthritis. According to epidemiological studies, disease development is attributed to the interaction of both systemic and biomechanical factors. Systemic factors that increase susceptibility to development of knee OA include hormonal status, bone mineral density, nutritional factors, and genetic predisposition. Biomechanical factors such as joint malalignment, high knee joint loads and quadriceps muscle weakness, are also risk factors for onset of disease and act as catalysts that speed up the osteoarthritic process.

2.1.1 Systemic factors

2.1.1.1 Hormonal Status

Estrogen deficiency may contribute to the etiology of osteoarthritis (OA) in women after menopause.\textsuperscript{34, 35} In a cross-sectional study, women who had used oral estrogen for 10 years or more had lower risk of developing OA of the hip [odds ratio (OR): 0.57, 95% confidence interval (95% CI) 0.40 – 0.82] compared to women who used estrogen for less than 10 years.\textsuperscript{34} A follow-up study over a period of 8 years revealed that current users of estrogen replacement therapy (ERT) had a lower relative risk of incident radiographic knee OA in comparison with those who never used estrogen [Relative risk (RR): 0.4, 95% CI, 0.1-3.0, 60% chance of no incident radiographic knee OA compared with never users].\textsuperscript{35} However, the difference was non-significant.\textsuperscript{35}
who used ERT also had a lower risk of progression of knee OA compared with less users of ERT [OR: 0.5, 95% CI, 0.1-2.9: p>0.05]. These results suggest that use of ERT may have moderate protective effect against radiographic knee OA in women after menopause.

2.1.1.2 Bone Density

Researchers have found that high bone density is associated with an increased prevalence of hip and knee OA. For example, it has been reported that women with radiographic hip osteoarthritis had 8% -12% greater bone density compared to women without osteoarthritis (p<0.001). Similar findings have been reported when comparing women with and without knee osteoarthritis. According to this Framingham study, bone mineral density (BMD) was 9% higher for the knee OA group than the group without knee OA (p<0.0001). A report from another Framingham study revealed that over an 8-year follow-up period, the risk for incident radiographic knee OA was 5.6% and 11.8% in women in the lowest and highest quartiles for BMD respectively.

However, evidence suggests that with higher levels of BMD, there is a decrease in the risk for progression of knee OA. According to the Framingham study, the risk of progressive knee OA was decreased significantly from 34.4% to 18.9% between the lowest and highest BMD quartiles (p<0.001). Furthermore, women who gained BMD over a period of 8 years, when compared with women who had lost BMD at a rate of more than 0.4 gm /cm², were at a decreased risk of progression of knee OA (p<0.001). These findings suggest that higher BMD may increase the risk of onset of osteoarthritis in women. However, greater bone density may have a protective effect against disease
progression by slowing down the subchondral bone changes that are associated with the disease progression.  

2.1.1.3 Nutritional factors

Continuous exposure to oxidants may contribute to osteoarthritis. Oxidants may affect the joint cartilage and synovial fluid matrix by damaging cartilage collagen, synovial fluid hyaluronate and a macromolecule which accounts for the synovial fluid viscosity. Dietary intake of anti-oxidants such as Vitamin C may help in reducing the risk of the joint cartilage damage. It has been reported that radiographic osteoarthritis progression and knee pain were reduced by threefold in people with high intake of vitamin C when compared to those with a low intake of vitamin C [OR: 0.3, 95% CI, 0.1 to 0.8]. However, Vitamin C intake did not affect the onset of knee osteoarthritis.

High levels of vitamin D may protect against both onset and progression of osteoarthritis. Parfitt et al suggested that vitamin D may contribute to normal bone metabolism by regulating the levels of chondrocytes that produce and maintain the cartilaginous matrix. Low levels of vitamin D may impair the ability of bone to respond optimally and may increase the risk of disease incidence. Lane et al reported that the risk of incident hip OA (definite joint space narrowing) for subjects who were in the middle [OR: 3.21, 95% CI, 1.06-9.68] and lowest tertiles [OR: 3.34, 95% CI, 1.13-9.86] of vitamin D serum levels was threefold higher when compared with subjects in the highest tertile. Also, the Framingham study found that individuals with low levels of vitamin D intake increased the risk of radiographic knee osteoarthritis progression by
fourfold when compared to people with high vitamin D intake [OR: 4.0, 95% CI, 1.4 to 11.6].

2.1.1.4 Genetic predisposition

Genetic predisposition may contribute to the onset of osteoarthritis. Disease occurrence may be higher in families that have genes affecting osteoarthritis, such as the vitamin D receptor gene that influences bone metabolism and cartilage protein genes affecting the production of cartilaginous matrix. Neame et al reported that siblings (n = 737, age >40 years) of people with knee OA were at greater risk of developing knee OA by three fold in comparison with the general population (n = 1729, age >40 years). An association was reported between disease occurrence and the presence of loci on chromosome 2q when analysing families with OA disease and their affected sibling pairs (p<0.05).

2.1.1.5 Age and Sex

The prevalence and incidence of knee and hip OA increases with age. The relationship between age and risk of OA is mediated by age related changes of the joint articular cartilage resulting in abnormal joint loading and instability. However, evidence suggests that ageing is not the primary determinant of knee OA incidence. It has been reported that older persons were at high risk of developing knee OA only if they were women, obese or were physically active. In the Framingham study, 15.6% of older individuals developed radiographic knee OA (Kellgren and Lawrence (K/L) severity score of ≥ 2, age: 70.5 years) after an 8-year follow-up. After adjustment for multiple risk factors, it was found that women had a higher risk of knee OA incidence than men [odds
ratio: 1.8, 95% CI, 1.1-3.1]. Higher body mass index (BMI) [OR: 1.6 per five unit increase in BMI, 95% CI, 1.2 -2.2] and high levels of physical activity [OR: 3.3, 95% CI, 1.4-7.5] also increased the knee OA risk in older individuals.45

Similar findings were reported in another Framingham study.10 Sex determined progression rates of knee OA rather than age in this particular cohort. Rates of incident disease were 1.7 times higher in women than in men (age: 70.8 years, OR: 1.7, 95% CI, 1.0 – 2.7), and progressive disease occurred slightly more often in women than in men (p>0.05). During an 8-year follow up, it was found that 2% women in the study developed radiographic knee OA per year, and approximately 1% developed symptomatic knee OA per year. Eighteen percent of women who did not have OA at the beginning of the 8 year period developed radiographic OA and 8 % of women developed symptomatic OA. In women with knee OA at study onset, the severity increased approximately 4% per year. The authors reported that at the end of eight years follow-up, 31.8% of women had progressive radiographic OA (K/L severity grade 2 at baseline to K/L grade 3 at follow-up).10

2.1.2 Biomechanical factors

Alterations to the local mechanical environment of the knee joint may also lead to OA development and progression. Biomechanical factors such as joint load, joint alignment, and quadriceps strength, have been found to contribute to disease onset and progression.
2.1.2.1 Joint load

High and/or repetitive loads on the knee joint during walking and other activities are believed to be the major factor contributing to knee OA pathomechanics.\textsuperscript{16, 17} The knee joint is a complex structure with three joint compartments including the medial and lateral tibio-femoral compartments and the patello-femoral joint. During walking, forces across the knee joint are not transmitted equally between the medial and lateral compartments. The load on the medial compartment is approximately 2.5 times greater than that on the lateral compartment.\textsuperscript{18} Consequently, there are consistently higher loads on the medial compartment which could contribute to degenerative changes of the articular cartilage.\textsuperscript{18} This imbalance in the load distribution may explain the higher prevalence of medial compartment involvement (75\% of knee OA) reported in subjects with knee OA relative to the lateral compartment (25\% of knee OA).\textsuperscript{19}

Several factors contribute to high and/or repetitive loads on the knee joint.\textsuperscript{45, 47-49} Primary contributors include obesity, occupation, and participation in sport at elite levels. High body weight increases the load on the knee during standing and walking.\textsuperscript{45, 47-49} There is evidence that the risk for onset and progression of radiographic knee OA increases with a higher BMI.\textsuperscript{45, 47-49} According to a Framingham study, a higher baseline body mass index increased the risk of knee OA incidence [odds ratio $= 1.4$ per 10 lb change in weight, 95\% CI, 1.1 - 1.8].\textsuperscript{45} Manninen et al\textsuperscript{47} reported similar findings. According to their report, obesity was a strong risk factor for knee OA incidence and a linear relationship existed between body mass index and OA incidence; for every increase of BMI by 3.8 kg/m\textsuperscript{2} the relative risk was 1.4 [95\% CI, 1.2 - 1.5]. Furthermore, other studies have reported that a high BMI increases the risk of OA disease progression.\textsuperscript{48, 49}
Evidence also suggests that knee OA risk increases with jobs that require heavy lifting and/or kneeling and therefore place high and repetitive loads on the knee.\textsuperscript{50-52} According to the United States first National Health and Nutrition Survey 1971-1975, 7\% of participants (age: 55-64 years) were diagnosed with radiological knee osteoarthritis.\textsuperscript{50} Involvement in heavy work (lifting or kneeling) increased the knee OA risk by 2.5 times in men (OR: 2.45, 95\% CI, 1.21-4.97) and by 3.5 times in women (OR: 3.45, 95\% CI, 1.22 – 10.5).\textsuperscript{50} Another study reported that knee OA risk was increased in both men (age: 47-93 years, OR: 1.9, 95\% CI, 1.0 to 3.3) and women (age: 47-93 years, OR: 1.5, 95\% CI, 1.0 -2.3) by lifting weights of more than 10 kg for a period of 10 years with a frequency of ten times per week.\textsuperscript{52} With a combination of heavy workload and kneeling tasks, Felson et al\textsuperscript{51} reported an OR for development of knee OA of 2.22 (95\% CI, 1.38 – 3.58). Similar findings were reported in other studies.\textsuperscript{53-56} No evidence was found that work involving heavy lifting and kneeling is a risk factor for knee OA progression.

Literature also suggests that professional athletes who engage in sports involving high impact repetitive forces directly on the knee joint are at an increased risk for injury and subsequent development of OA.\textsuperscript{57,58} Kujala et al\textsuperscript{57} compared athletes in different sports, including soccer players, weight lifters, shooters and runners, and reported that knee OA risk was increased by five times in soccer players (OR: 5.21, 95\% CI, 1.14-23.8) when compared to shooters and runners. They also reported that soccer players (29\%) and weight lifters (31\%) had higher prevalence of knee OA when compared to athletes such as shooters (3\%) and runners (14\%), (p<0.05). Similar findings of increased knee OA risk in women who were involved in medium and long distance
running, compared to non-runners (OR: 3.57, 95% CI, 1.89-6.71) were reported in another study.58 However, it is unclear whether the increased risk of subsequent development of knee OA is primarily due to repetitive loading on the knee or is secondary to trauma to the joint due to involvement in sport.59

2.1.2.2 Joint Alignment

The load bearing axis (LBA) is represented by a line drawn from the centre of the femoral head to the centre of the ankle (Figure 2.1). In healthy subjects, this line passes through or just medial to the knee joint centre.62 Knee joint alignment is measured as the angle formed by the intersection of the anatomical axes of the femur (the line from the centre of the femoral head to the centre of femoral intercondylar notch) and the tibia (the line from ankle talus center to the center of the tibial spine tips) (Figure 1).5, 60-62 This angle is called the hip knee ankle angle (HKA). Under normal conditions, this angle ranges from 0 to 3 degrees of varus.62 (Figure 2.1). An HKA angle in excess of this normal range (0° to 3°), is considered a varus malalignment.62 In a valgus deformed knee (valgus malalignment), the load-bearing axis passes lateral to the knee center increasing forces across the lateral compartment.
Figure 2.1: Common patterns of frontal plane lower limb alignment, modified from Cook et al.\textsuperscript{63}

A. Varus alignment: LBA passes medial to the knee and knee centre is displaced laterally, HKA = $-ve$

B. Neutral alignment: knee center is located on the LBA, HKA = $0^\circ$

C. Valgus alignment: LBA is lateral to the knee and knee centre is displaced medially, HKA = $+ve$

LBA = Load bearing axis; FM = Femoral mechanical axis; HKA = Hip-Knee-Ankle angle; TM = Tibial mechanical axis.
Varus and valgus alignment may not be an independent risk factor for knee OA incidence.\textsuperscript{64} Only one study was found that reported an association between both varus (OR = 2.06, 95\% CI, 1.28-3.32) and valgus alignment (OR = 1.54, 95\% CI, 0.97-2.44) with medial and lateral knee OA incidence respectively\textsuperscript{64}. Further analyses, based on BMI, revealed a higher association between joint mal-alignment and the disease incidence only for individuals who were obese (BMI >30 kg/m\textsuperscript{2}).\textsuperscript{64}

However, evidence suggests that joint mal-alignment is an independent risk factor for disease progression.\textsuperscript{65} According to Sharma et al\textsuperscript{65}, both varus (OR = 4.01, 95\% CI, 2.19 – 7.62) and valgus alignment (OR = 4.78, 95\% CI, 2.08 – 11.02) increased the risk for medial or lateral knee OA progression by four times respectively. Other studies have reported similar findings.\textsuperscript{17, 66-68}

\textbf{2.1.2.4 Muscle weakness}

Quadriceps muscle weakness is common amongst individuals with knee OA.\textsuperscript{69, 70} Quadriceps weakness has been identified as one of the markers in detecting radiographic knee OA in people with or without pain.\textsuperscript{69, 70} According to Slemenda et al\textsuperscript{69} for every 13.56 Nm loss of knee extensor strength, the odds ratio for prevalence of radiographic knee OA was 0.8 [95\% CI, 0.71 - 0.90] and symptomatic knee OA was 0.71 [95\% CI, 0.51 – 0.87]. Women who developed knee OA had lower baseline extensor strength (40.82 Nm) than women who did not develop knee OA (47.20 Nm, p <0.001). Another study reported similar findings.\textsuperscript{70} After adjusting for the body weight, knee extensor strength was 18\% lower at baseline among women who developed knee OA than those women without knee OA.\textsuperscript{70} These findings indicate that women with low levels of
knee extensor strength had higher risk of developing knee OA compared to those with higher strength values.\textsuperscript{69, 70}

\subsection*{2.1.2.5 Joint injury}

Evidence suggests that individuals who had experienced knee joint injuries such as meniscal and cruciate ligament tears, fractures and dislocations were at a high risk for later development of knee OA.\textsuperscript{71, 72} According to a 36-year follow-up study, 13.9\% of participants who reported knee injuries during their younger days (22 years of age on average) had knee OA at the age of 65 years.\textsuperscript{71} The knee OA risk was increased by threefold in people with knee injuries earlier in life (RR: 2.95, 95\%CI, 1.35-6.45, p = 0.005).\textsuperscript{71} Roos et al\textsuperscript{72} reported similar findings. They found that the risk of knee OA was increased by 14 times in people who had undergone menisectomy after a 21 year follow-up (RR = 14.0, 95\%CI, 3.5 – 121.2). Knee injuries might disrupt normal knee joint mechanics leading to altered/abnormal load distribution within the damaged knee joint contributing to the increased incidence of later OA.

\subsection*{2.2 Gait Analysis}

Gait analyses are essential to describe the changes in knee joint function brought about by the OA disease process.\textsuperscript{73} Human walking is a tri-planar motion and to quantify objective physical findings in all three planes, a system for recording gait in three dimensions is required. Gait analyses are performed using a motion-analysis system. This includes a walkway with force plates embedded in it, markers, and cameras to track joint position during walking.\textsuperscript{24} Three-dimensional motion analysis software is used to process the gathered raw motion (temporal and distance parameters, and kinematic data)
and force plate data (gravitational and ground reaction forces). The information obtained can be used to provide objective data related to motion (kinematics) and for calculating joint forces and moments concerned with this motion (kinetics).\textsuperscript{74}

\textbf{2.2.1 Gait cycle}

A gait cycle is defined as the sequence of events, which occurs from initial contact of one foot to the successive initial contact of the same foot. In healthy adults, 60\% of the gait cycle is represented by stance phase (foot is in contact with the ground) and the remaining 40\% of the cycle is represented by swing phase (when the foot is not in contact with the ground). Stance phase is further divided into four sub-phases: loading response, mid-stance, terminal stance, and pre-swing.\textsuperscript{24} Swing phase is also sub-divided into early swing, mid-swing, and late-swing phases. In a gait cycle, there are two periods of double limb support (both feet contact the ground) and two periods of single limb support (body supported by a single limb).\textsuperscript{24}

\textbf{2.2.2 Temporal and distance parameters}

Common temporal and distance measures in a gait cycle are gait speed (m/s), cadence (steps/minute), and stride length (m).\textsuperscript{23, 24} There is evidence that individuals with medial compartment knee OA have lower gait speed, shorter stride length, and lower cadence when compared to healthy control subjects of similar age.\textsuperscript{26} According to Deluzio and Astephen\textsuperscript{26} people with knee OA walk slower (0.76 ± 0.08 m/s) than those without knee OA (0.95 ± 0.14 m/s, p<0.05). Similar findings have been reported in other studies of people with knee OA.\textsuperscript{29, 31, 73, 75-77} Conversely, other studies reported no differences in gait speed between knee OA and healthy control groups.\textsuperscript{28, 78}
There is evidence that people with medial compartment knee OA have lower stride length and cadence than healthy control subjects.\textsuperscript{27, 31} According to Astephen et al\textsuperscript{31} subjects with severe knee OA had significantly lower stride length compared to control subjects (knee OA: 1.16 m ± 0.19, healthy controls: 1.44 m ± 0.13, \( p < 0.0001 \)). In a similar study, Gok et al\textsuperscript{27} reported that the group with medial compartment knee OA walked with fewer steps per minute than the healthy control group (knee OA: 102 ± 10 steps per minute, Control: 112 ± 14 steps per minute, \( p < 0.05 \)). Similar findings were reported in other studies.\textsuperscript{73, 76}

\textbf{2.2.3 Kinematic Analysis}

Kinematics is a branch of biomechanics that is concerned with human movement without considering the forces that cause the motion.\textsuperscript{23} Kinematic gait variables include linear and angular displacements that describe the range of motion of a body segment in relation to an adjacent segment.

\textbf{2.2.3.1 Knee joint range of motion}

When the foot first contacts the floor the knee joint is in approximately 0° of flexion (Figure 2.2). In early stance phase, as the limb starts accepting weight, the knee joint reaches 15° of flexion.\textsuperscript{24} As the limb completes accepting the entire body weight, the knee joint angle reverses its pattern from 15° of flexion to 5° and 0° at mid-stance and heel-off, respectively.\textsuperscript{24} The knee starts to flex at heel off and reaches 30° of knee flexion at toe-off. In the swing phase, the limb flexes further and reaches approximately 70° of knee flexion before extending the knee for the heel-strike.\textsuperscript{24}
Figure 2.2: Schematic representation of the knee joint angle during the gait cycle.
Several studies have compared total knee joint range of motion in people with medial compartment knee OA and healthy controls.\textsuperscript{22,25,26,31,79} Conflicting findings have been reported. According to Astephen et al\textsuperscript{31} the group with severe knee OA had a lower knee range of motion than the healthy control subjects during the gait cycle (Severe knee OA: 49.9° ± 16.1, Control: 68.5° ± 6.01, $p<0.0001$). However, total range of motion in the group with moderate knee OA did not differ from the healthy subjects. Baliunas et al\textsuperscript{22} reported similar findings. They found that the group with end stage knee OA had a lower range of motion (58°± 7) than the healthy control group (64° ± 5, $p<0.001$).\textsuperscript{22} Several studies reported no differences in the knee range of motion between subjects with mild-moderate knee OA and healthy subjects during the gait cycle ($p>0.05$).\textsuperscript{25,26,79}

Authors have also reported differences in the knee flexion angle during early stance phase between people with knee OA and control subjects.\textsuperscript{31} Astephen et al\textsuperscript{31} reported differences in the knee flexion angle between OA groups of varying severity and healthy subjects (severe knee OA: 8.04° ± 6.22, moderate knee OA: 14.02° ± 7.12, control: 18.72° ± 7.28, $p<0.0001$). In addition, the flexion angle differed between the two OA groups ($p<0.0001$). However, Baliunas et al\textsuperscript{22} did not find differences in the stance phase flexion angle between control subjects and those with knee OA ($p = 0.625$). Other authors also did not report any difference in knee flexion angle in stance between healthy control subjects and those with knee OA ($p>0.05$).\textsuperscript{18,29}

Conflicting findings reported in the studies described above might be attributed to the severity of the knee OA disease. The study that showed differences in the stance phase knee flexion angle compared individuals with end-stage or severe knee OA with healthy control subjects\textsuperscript{31} whereas studies that reported no differences between
groups compared individuals with moderate knee OA with that of the healthy control subjects. Studies have also suggested that individuals with severe/end-stage knee OA reduced either their walking speed or stride length or cadence and differences in the knee range of motion might be attributed to these alterations in their temporal-distance parameters.

2.2.4 Kinetic Analysis

Kinetics is the branch of biomechanics that deals with the joint forces that cause motion. Data obtained regarding external forces, kinematic data, and anthropometric measures (segment mass, centre of mass and inertia) allow for the calculation of joint moments using an inverse dynamic approach. Joint moments are classified as external or internal. External moments are produced by external forces including gravitational (ground reaction forces) and inertial forces. The internal moments are equal and opposite to the external moments and are generated by muscles, joint capsules, and ligaments, which counteract the external forces acting on the body. In this review, external moments are reported unless stated otherwise.

2.2.4.1 Frontal Plane Knee Moment

Much of the research on knee OA mechanics has been concerned with measuring the knee joint load in the frontal plane using 3-dimensional gait analysis. Dynamic knee joint load in the frontal plane can be estimated by calculating the external knee adduction moment. The external knee adduction moment is the product of the frontal plane component of the ground reaction force (GRF) and the lever arm or the perpendicular distance between the knee joint centre of rotation and the GRF vector as it
passes from the centre of pressure (COP) under the foot to the vicinity of the centre of mass (COM) of the body (Figure 2.3a).\textsuperscript{18} The normal pattern of frontal plane moments in gait starts with an initial brief knee abduction moment followed by an adduction moment through the remainder of stance phase. There are two peaks in the first and second half of stance with an intervening lower moment (Figure 2.3b). Studies have reported higher knee adduction moments in subjects with medial knee OA compared to healthy knees.\textsuperscript{16, 17, 21, 22, 27} Both peaks have been reported to be higher in knee OA compared to healthy control subjects. Astephen et al\textsuperscript{31} also found that the adduction moment magnitude was greater in mid stance in the OA subjects.

It has also been found that a high peak knee adduction moment during gait is a risk factor for the presence, severity, and progression of medial compartment knee OA.\textsuperscript{18} Weidow et al\textsuperscript{80} reported that individuals with medial compartment knee OA (n=15) had a higher external knee adduction moment than that of the healthy control subjects (n=15, p<0.05, 52% higher than the controls). Furthermore, individuals with lateral compartment knee OA (n=15) had a lower average external knee adduction moment than that of the healthy control subjects (n=15, p<0.05, 63% lower than the controls).
Figure 2.3a: The external knee adduction moment, modified from Specogna et al.\textsuperscript{81}

Figure 2.3b: Profile of knee adduction moment during the stance phase in subjects with knee OA and healthy controls.
There is also evidence that a relationship exists between the knee adduction moment and disease progression.\textsuperscript{17} Miyazaki et al\textsuperscript{17} reported that the knee adduction moment correlated positively with radiographic disease progression, defined as more than one grade of joint space narrowing of the medial compartment over a period of 6 years ($r = 0.23$, $p<0.05$). They reported that the knee adduction moment was significantly higher for the OA group with progression than the group with no progression of disease ($p<0.0001$). The average loss of joint space width during the six years was 1.4 mm. With every 1% increase in the knee adduction moment, the risk of medial compartment OA progression was increased by 6.46 times.\textsuperscript{17}

\textbf{2.2.4.2 Sagittal Plane Knee Moments}

Sagittal plane kinetics may also be important in determining the knee joint reaction to dynamic joint loading. Knee joint reactions to dynamic loading in the sagittal plane can be assessed by analysing the external sagittal plane moments.\textsuperscript{23}

The typical sagittal plane knee moment waveform during the stance phase of gait begins with an external knee extension moment at initial contact, progressing to an external knee flexion moment in early stance phase and an external knee extension moment in late stance phase.\textsuperscript{23, 24} (Figure 2.4 & 2.5). At initial contact, the knee is extended and the ground reaction force vector is anterior to the knee joint, creating a small external extension moment. The hamstring muscles (knee flexors) are active to counteract this external moment. Following initial contact, the knee flexes approximately 15\textdegree{} and the GRF vector falls posterior to the knee, creating an external flexor moment. This moment is opposed by the quadriceps muscles (knee extensors) which contract at
this point preventing the knee from flexing further during load transfer to the stance limb. From heel-off to toe-off, the knee flexes to approximately 30°. The GRF vector falls anterior to the knee joint at this point in the gait cycle creating a late stance external extension moment. The knee flexor muscles counteract this moment.23,24
Figure 2.4: External knee joint sagittal moments during the stance phase of a gait cycle with centre of pressure (COP) and ground reaction force vectors (GRFV).
Figure 2.5: Profile of external knee sagittal moment during the stance phase.
Several studies have investigated sagittal plane lower extremity kinetics in people with medial compartment knee OA and compared them to those of healthy older adults.\textsuperscript{18, 22, 25-31, 79} There is little research comparing the initial knee extension moment between knee OA and healthy control groups. However, there is some evidence that individuals with knee OA have a lower knee extension moment in the initial stance phase than healthy control subjects.\textsuperscript{29, 31} According to Astepehn et al\textsuperscript{31}, individuals with knee OA of varying severity (moderate and severe) differed significantly from the healthy control subjects. The group with severe OA had a lower initial knee extension moment compared to the moderate OA group. Furthermore, both groups had a lower knee extension moment than the healthy control subjects in the initial stance phase (severe OA: \(-0.14 \pm 0.08\) Nm/kg; moderate OA: \(-0.26 \pm 0.10\) Nm/kg; healthy subjects: \(-0.31 \pm 0.14\) Nm/kg, \(p<0.05\)). Another study reported similar findings between knee OA and healthy control groups.\textsuperscript{29}

Conflicting findings have been reported concerning the early stance phase flexion moment. Several researchers have found that individuals with medial compartment knee OA had lower external knee flexion moments compared to healthy controls.\textsuperscript{25, 26, 31, 79} For example, Astephen et al\textsuperscript{31} found that people with knee OA (moderate OA, \(n = 60\); severe OA, \(n = 60\)) had a significantly lower early stance flexion moment compared to controls (\(n=60\)) [moderate OA group: \(0.40 \pm 0.22\) Nm/kg, severe OA group: \(0.33 \pm 0.20\) Nm/kg, healthy controls: \(0.52 \pm 0.29\) Nm/kg, \(p<0.002\)]. Another study by Deluzio and Astephen\textsuperscript{26} reported that the knee OA group had a lower external knee flexion moment than the healthy control group [knee OA group: \(-0.28 \pm 1.17\)
Nm/kg, healthy control: 0.22 ± 0.79 Nm/kg, p<0.05]. Other studies have reported similar findings.25, 79

Conversely, other researchers have reported higher external knee flexion moments for those with knee OA compared to healthy controls.18, 27, 28 For example, Schipplein and Andriacchi18 reported knee flexion moments of 3.32 ± 1.86 %Bw*Ht for the knee OA group and 1.81 ± 0.65 %Bw*Ht for the healthy control group (p<0.05). Similar findings were reported by Al-Zahrani and Bakheit28 [knee OA: 0.33 ± 0.33Nm/kg, healthy control: 0.10 ± 0.13Nm/kg, p<0.05] and Gok et al27 [knee OA: 0.62 ± 0.25Nm/kg, healthy control: 0.42 ± 0.20Nm/kg, p<0.05]. Finally, there have been reports of no significant differences in the external knee flexion moment in early stance between OA and control groups.22, 29, 30

Peak knee flexion angle during the stance phase may affect the magnitude of the knee flexion moment. Lower knee flexion angle in early stance may result in a lower knee flexion moment. Astephen et al31 found that the OA groups differed amongst themselves and from the healthy control group in the early stance phase knee flexion angle (moderate OA: 14.02° ± 7.12, severe OA: 8.04° ± 6.22, healthy control: 18.72° ± 7.28, p<0.002). Other authors have also found that individuals with knee OA walk with a reduced knee flexion angle (a straighter knee) in early stance phase compared to healthy subjects.26, 27, 29

Conversely, Schipplein and Andriachhi18 reported that people with knee OA had a similar stance phase peak knee flexion angle to that of healthy control subjects (p> 0.05). They argued that subjects with knee OA adapt their gait to produce higher
external flexion moments and higher muscle forces across the joint to stabilize the knee.\textsuperscript{18} They further argue that a positive relationship may exist between external knee adduction and flexion moments in early stance.\textsuperscript{18} They reported that participants in the OA group had higher mean knee adduction moments and higher knee flexion moments than those of the healthy control group (p<0.05).\textsuperscript{18} The authors speculated that high moments in both planes might provide dynamic joint stability in subjects with moderate knee OA.\textsuperscript{18}

Findings regarding the late stance extension moment are more consistent. Authors have reported either lower knee extension moments in people with medial compartment knee OA compared to healthy older adults or no difference between groups.\textsuperscript{18, 26, 30, 31} Astephen et al\textsuperscript{31} reported that the moderate and severe knee OA groups had lower external extension moments in late stance (moderate OA: 0.35 ± 0.21 Nm/kg, severe OA: 0.10 ± 0.19 Nm/kg) than the healthy controls (0.42 ± 0.13 Nm/kg, p<0.05). Other studies reported no differences in the late stance extension moment between knee OA and control groups (p<0.05).\textsuperscript{18, 26, 30}

2.3 Conclusion

This review highlighted the role played by biological and biomechanical risk factors in knee OA incidence and progression. Biological factors increase susceptibility for OA development and later, with the alterations to the local environment, the disease process speeds up leading to progression of knee OA. Based on the above review it is evident that the knee adduction moment can be used to measure the knee joint load during walking. However, the forces across the knee joint due to the dynamic load are also associated with the sagittal plane mechanics. There are conflicting reports
regarding sagittal plane knee joint moments and kinematics in people with medial
compartment knee OA. Therefore, this study proposes to further investigate sagittal plane
moments and kinematics of individuals with medial compartment knee OA and healthy
subjects.
Chapter 3
Methods

3.1 Study Design

A cross sectional group comparison (group with medial compartment knee OA compared to a healthy control group matched for age and gender) was used in this study. The knee OA group was sub-divided into low and moderate OA groups based on pain scores from the Western Ontario McMaster Universities Osteoarthritis Index (WOMAC). This study utilized baseline data from a completed study investigating the effect of an exercise intervention on frontal plane knee joint loading in people with medial compartment knee OA (Appendix 1).

3.2 Participants

Forty individuals with OA of the medial compartment of the knee joint and 40 healthy participants without OA participated in this study after giving their written informed consent (Appendix 1). The University Health Sciences Research Ethics Board at Queen’s University, Kingston, Ontario, Canada, approved the study.

3.2.1 Knee OA Group

Individuals with medial compartment knee OA were recruited from the community through newspaper advertisements and from the practices of orthopaedic surgeons in Kingston, Ontario. They were selected for the study based on the following criteria: age $\geq$ 40 years, self-reported pain in the knee for most days of the month, radiographic evidence of medial compartment knee OA or documented evidence of cartilage loss in the medial compartment by arthroscopy or magnetic resonance imaging.
For those individuals with bilateral medial compartment knee OA, the affected knee was defined as the most painful knee.

Exclusion criteria included other disorders which might affect gait such as rheumatoid arthritis, heart disease, or any neurological conditions including stroke or Parkinson’s disease. Prior periarticular fractures of the knee joint were also exclusion criteria. If the subjects with knee OA had received an intra-articular corticosteroid or visco-supplementation injection into either knee within the previous 3 months, had a diagnosis of hip OA, had previous trauma affecting one or both hips, or had previous replacement of any joint in the lower extremities they were also excluded from participation in the study. Exclusion criteria included non-English speakers, cognitive impairment that would preclude obtaining informed consent and inability to ambulate without a gait aid. Potential participants were also excluded if they were receiving any rehabilitation for knee OA or were participating in an exercise program designed to strengthen the hip musculature at the time of recruitment.

3.2.2 Control group

A group of 40 healthy individuals matched initially to the OA group by sex, age (± 5 years), height (± 5 cm), and mass (± 5 kg) formed the control group. However, as the study progressed, it became difficult to match groups based on height and body weight. Therefore, sex and age became the primary criteria for matching between groups. Participants from this group were also recruited through newspaper advertisements and posters displayed in churches and seniors’ centers in the Kingston area. Exclusion criteria included musculoskeletal disorders of the lower extremity,
cardiovascular disease, or neurological conditions such as stroke or Parkinson’s disease or any other disorders that might have affected ambulation or ability to undergo the required testing.

3.3 Sample Size

This study utilized baseline data from a completed study investigating the effect of an exercise intervention on frontal plane knee joint loading in people with medial compartment knee OA. There were 40 participants with knee OA and 40 control subjects matched for age and gender.

3.4 Data Collection

3.4.1 Setting

For all participants, the majority of testing was conducted in the Motor Performance Laboratory at Queen’s University, Kingston, Ontario. Radiographs of the knee were taken in the Radiology Department at Kingston General Hospital.

3.4.2 Alignment and Knee OA Grading

A bilateral anterior-posterior radiograph of the knees in weight-bearing was obtained in accordance with hospital’s standardized protocol. If participants had weight-bearing knee radiographs taken within 6 months of the date of testing, permission was requested to obtain digital images. New radiographs were not obtained in these cases.

A computer software program (Horizon Image Viewer, version 1.5, OAISYS Medical Inc.), was used to define femoral and tibial bone landmarks on the digital images in order to measure frontal plane knee alignment.82-86 Assessment of all
digital images and alignment measurements was carried out by a single study investigator who had been trained in the application of the software program. The most commonly used measure of knee alignment is the hip knee ankle (HKA) angle. Full limb radiographs are needed to obtain this angle, defined as the angle formed by the intersection of the mechanical axes of the femur (the line from the centre of the femoral head to the centre of the femoral intercondylar notch) and the tibia (the line from ankle talus center to the center of the tibial spine tips). As the radiographs obtained for this study were not full-limb radiographs, the HKA angle was estimated using the anatomic axis angle: the angle formed at the centre of the knee joint by the intersection of the femoral and tibial anatomic (shaft) axes. The femoral anatomic axis was defined as the axis that passes through the centre of the femoral shaft to the mid-condylar point of the distal femur and the tibial anatomic axis as the axis that passes through the centre of the tibial shaft to the centre of the tibial plateau (interspinous midpoint). Since the femoral mechanical axis has been shown to deviate from the femoral anatomic axis by 4-5°, the mechanical axis alignment (the HKA angle) was estimated by subtracting 5° from the anatomic axis angle.

Radiologic severity of knee OA was classified using the system described by Cooke et al for both knees. Severity scores for each participant were determined by the Orthopaedic Consultant who developed the grading system. In this classification scheme, the medial compartment was scored for joint space narrowing (0-3), femoral osteophytes (0-3), tibial erosion (0-4), and subluxation (0-3). Tibial erosion was graded by means of progressive loss of bone, from dishing to marginal destruction, and subsequent fragmentation leading to gross bone damage. Subluxation was identified as a
shift medially or laterally between the tibial spines and the femoral sulcus. Scores range from 1 to 13; a participant with maximum knee damage would score a maximum of 13 on the grading scheme. This grading scheme has been shown to have excellent inter-reliability scores for the knee OA population. Severity scores have been found to be significantly correlated with alignment measures.90

3.4.3 Knee Pain and Function

The Western Ontario McMaster Universities Osteoarthritis Index (WOMAC) was used to obtain a self-reported measure of joint pain, stiffness, and difficulty in function in the participants with knee OA. WOMAC is a self-administered questionnaire with 24 questions pertaining to joint pain (5 questions), stiffness (2 questions) and physical function (17 questions). Each question has a sub-scale ranging from 0 to 4; with 4 representing extreme pain, stiffness and functional difficulty. The scores were then summed to produce total scores for each of the three measures as well as a total score. The WOMAC is a recommended tool for the evaluation of knee OA populations, and the reliability and validity of the WOMAC have been well established in this particular population.91-94

Either radiographic score or pain could be used to divide the people with OA into two groups based on severity. Radiographic changes do not correlate highly with pain severity.2 Pain scores as measured on the WOMAC were used in this study to separate the participants with OA into two equal groups; a low pain score group with scores below 7/20 and a moderate group with scores of equal to or greater than seven.
The score of seven was selected because that is the score that divided the OA subjects such that there were equal numbers in two groups.

### 3.4.4 Gait Analysis

The Optotrak 3020 optoelectronic motion analysis system and two AMTI force plates (Advanced Mechanical Technology Inc., Newton, MA) were used for gait analysis. The two force plates were mounted within an eight-metre walkway in the Motor Performance Laboratory at Queens University, Canada. The Optotrak motion analysis system included two infrared cameras that were placed on either side of the walkway. The cameras detect the position of infrared emitting diodes (IRED’s) placed on the subject. The position of these markers is then transmitted to a central processing computer for determining the three dimensional position of the markers relative to the cameras and the force plates. This enables tracking the motion as per a pre-determined coordinate system.

Participants were dressed in shorts and a loose fitting shirt for the testing. Rigid marker clusters containing infrared light emitting diodes (IREDs) were positioned on the dorsum of the foot, lateral shank, lateral thigh, and sacrum, and over the spinous processes of the seventh cervical/first thoracic vertebrae. The clusters were secured with Velcro straps and/or tape to avoid movement of the markers during the walking trials. Although markers were attached to both legs, only data from the test leg were analyzed. For healthy controls, the test leg corresponded with the affected leg of the matched knee OA participants.

Participants walked at a self-selected comfortable pace on the eight metre walkway in their comfortable walking shoes. Five good walking trials were obtained for
each participant; trials were included only if the participants landed with one foot on each force plate and all IRED markers were visible by the cameras over the full course of the gait cycle to be analyzed. Ground reaction force data from the two force plates were collected at a sampling frequency of 200 Hz.

Following the gait trials, participants stood in view of the cameras and a series of reference trials were captured using a pointed probe fitted with four IRED markers. Using the probe, specific bone landmarks were touched with the probe to identify the location of the landmarks in relation to the marker clusters. Based on the location of these landmarks and the marker clusters, joint centres could then be approximated. The bone landmarks selected for the reference trials included the first and fifth metatarsal heads, medial and lateral malleoli, medial and lateral femoral epicondyles, greater trochanters, a point directly vertical to the greater trochanter at the level of the mid-iliac crest bilaterally and the acromion processes of the scapula.

Visual three-dimensional motion analysis software (C-motion Inc., Rockville, MD) was used to process the gathered raw motion (temporo-spatial parameters) and force plate data (gravitational and ground reaction forces). An inverse dynamics procedure was applied to the gathered data to generate external knee joint moments during stance phase.

3.5 Outcome Measures from Gait Analysis

3.5.1 Knee Joint External Moments

To calculate and compare peak external knee joint frontal and sagittal plane moments between the groups, the stance phase of the gait cycle for the test leg was
divided into 100 points representing 100% of the stance phase. Peak external knee adduction, flexion, and extension moments were calculated from the average moment waveforms of five gait trials. For external knee adduction and flexion moments, the highest peak in the first 50% of the stance phase was used in the analysis, while the second 50% of the stance phase was used to calculate the external knee extension moment. The average peak extension moment in early stance was also obtained. The peaks of these moments were then normalized to body weight and height (expressed in the units of %Bw*Ht), to allow comparison between groups. The average point in the gait cycle where these moments occurred was also compared between groups.

3.5.2 Knee flexion angle in early stance phase

Maximum knee flexion angle (°) in the early stance phase of the gait cycle was calculated in the first 50% of stance phase. The average knee flexion angle obtained from five gait trials was used for the analyses.

3.5.3 Temporal-Spatial Parameters

Data gathered from five walking trials were used to obtain the average of the following temporal-spatial parameters: gait speed (m/s), stride length (m), double limb support time (secs), and cadence (steps/min).

3.6 Procedure

Potential participants either self-identified as having knee OA after reading advertisements or contacted investigators after receiving a letter from the orthopaedic surgeon. Preliminary screening was conducted by phone. Participants who appeared to
meet the inclusion criteria on initial screening were given an appointment in the gait laboratory. Study objectives were explained in detail and questions were answered about the study. All participants then signed the informed consent (Appendix 1). Testing was conducted in the Radiology Department at Kingston General Hospital and in the Motor Performance Laboratory at the school of Rehabilitation Therapy. The testing session lasted approximately 2 – 2.5 hours.

A medical history was obtained for participants with knee OA and control subjects. Those who had exclusion criteria identified during this process were thanked for their participation and participation in the study was concluded. Prior to the gait testing, OA participants were asked to complete the WOMAC questionnaire and subjects in both groups completed the Physical Activity Scale for the Elderly (PASE). PASE data were not used in this study. Participants’ weight and height were measured using a regular scale and tape measure. Participants were then asked to change into shorts for the walking tests. Surface markers were positioned on their skin at the foot, ankle, hip, lower back and base of the neck and straps were used to hold the markers in place. They were asked to walk along an 8-metre indoor walkway at a comfortable speed in their normal footwear as the two cameras detected the movement of the surface markers. Participants were provided with rest breaks in between walking trials. Five good trials were collected from each participant and the average of these trials was used for final analyses. If participants from either group had not received a recent knee X-ray, they were accompanied to the X-ray Department at Kingston General Hospital. In some cases, a separate appointment was made for X-rays.
3.7 Statistical Analyses

The first analyses involved a comparison between the group with knee OA and the control group. Distribution of the data collected was determined using the Kolmogorov-Smirnov Statistic. Independent sample t-tests were used to determine whether age, height, weight, and body mass index differed between the groups. Independent t-tests were also used to determine differences in temporal distance parameters of gait (gait speed, stride length, double limb support time, and cadence) and knee alignment. Finally, independent t-test were used to determine if the knee adduction moment in the first 50% of stance, the three external knee sagittal moments, the percent of the gait cycle at which these peaks occurred and the stance phase knee flexion angle differed between the OA and control group. The alpha level (p-level) was set at 0.05 for significance testing. Statistical analysis was performed using SPSS software (version 16.0.1. SPSS Incorporated, Chicago, Illinois, 2006).

The second analyses compared the two groups with knee osteoarthritis. The procedures for this comparison were as described above. Pearson correlation coefficients were used to determine associations between the stance phase flexion moment and knee flexion angle in early stance, the stance phase flexion moment and severity as determined by WOMAC pain scores and knee flexion moment in early stance and gait speed in the knee OA subjects. Correlation between knee flexion moment and knee flexion angle was also determined in the control group.
Chapter 4

Results

4.1 Knee Osteoarthritis and Control Group Comparison

4.1.1 Characteristics of the study participants

Forty participants with knee OA (mean age 62.98 ± 9.73 years, 23 women) and forty matched control participants (64.12 ± 9.04 years) completed the study. The data were examined for outliers, identified as mean values for an individual participant that were three standard deviations or more outside the group mean. Only one outlier was identified in the control group for BMI and this subject’s value was excluded from analysis. With this exclusion, all data were normally distributed as verified using Kilmgorov-Smirnov statistic.

Demographic characteristics for the 40 participants in each group are displayed in table 4.1. The OA group had higher values for weight and BMI compared to the control group (p<0.05). There were no significant differences in age or height between groups. The average grade of disease severity (Cooke’s scale) for the OA subjects was 3.7 ± 2.1 (maximum 13). The average pain score as recorded by the WOMAC was 5.5 ± 2.9, indicating an overall mild to moderate level of severity. The knee was in significantly greater varus alignment in the OA group (p< 0.05). Knee alignment ranged from -12.7° to 2.9° with an average of -4.1° ± 4.3 in the OA group. The corresponding values for the control group were -2.2° ± 1.9 (range: -8.9° to 1.7°).
Table 4.1 Demographic Characteristics and Temporal and Distance Parameters of Gait

<table>
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<tr>
<th></th>
<th>Knee OA (N = 40)</th>
<th>Control (N = 40)</th>
<th>p-value</th>
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</thead>
<tbody>
<tr>
<td>Age (yr)</td>
<td>62.98 (9.73)</td>
<td>64.12 (9.04)</td>
<td>p = 0.586</td>
</tr>
<tr>
<td>Height (m)</td>
<td>1.73 (0.11)</td>
<td>1.70 (0.08)</td>
<td>p = 0.228</td>
</tr>
<tr>
<td>Weight (kg)</td>
<td>82.31 (20.00)</td>
<td>69.71 (11.03)</td>
<td>p = 0.001*</td>
</tr>
<tr>
<td>BMI (kgm(^{-1}))</td>
<td>27.40 (5.5)</td>
<td>23.74 (2.66)</td>
<td>p = 0.000*</td>
</tr>
<tr>
<td>Stride length (m)</td>
<td>1.20 (0.16)</td>
<td>1.26 (0.15)</td>
<td>p = 0.083</td>
</tr>
<tr>
<td>Cadence (Steps/minute)</td>
<td>97.36 (10.5)</td>
<td>104.60 (9.3)</td>
<td>p = 0.003*</td>
</tr>
<tr>
<td>DLST (s)</td>
<td>0.40 (0.08)</td>
<td>0.33 (0.05)</td>
<td>p = 0.001*</td>
</tr>
<tr>
<td>Gait speed (ms(^{-1}))</td>
<td>1.00 (0.20)</td>
<td>1.12 (0.20)</td>
<td>p = 0.006*</td>
</tr>
</tbody>
</table>

All values are the mean values with standard deviations in the parentheses.

(*) Significant differences between groups; p<0.05

DLST = Double limb support time

BMI = Body Mass Index
4.1.2 Temporal and Distance Parameters of Gait

Temporal and distance parameters of gait are reported in Table 4.1. Temporal and distance data were not available for all subjects. Obtaining these data requires that the markers on the test foot are visible through one complete gait cycle. In cases where two consecutive heel contacts of the test leg were not recorded, it was not possible to obtain these data. This occurred in subjects with a long stride length where the camera range was insufficient to record consecutive foot contacts of the test limb. Stride length, cadence, and DLST data were not available for five subjects in the control group and three subjects in the OA group. Gait speed data were not available for two subjects in the control group and one subject in the OA group.

No outliers were identified in the temporal and distance data and data for each variable were normally distributed. Independent t-tests confirmed that the OA group had a slower gait speed (m/s), lower cadence (steps/min) and longer double limb support time (DLST) (s). There was no difference in the stride length between groups (p>0.05).

4.1.3 Knee Joint Moments

Data were reviewed to identify outliers and individual subjects data were excluded from the analyses if the mean value was beyond three standard deviations from the group mean. The number of outliers excluded was three for initial knee extension moment (OA group: 1, Control group: 2) and two for the early knee flexion moment (Control group: 2). For the remaining variables (knee adduction moment, and late knee extension moment), there were no outliers identified (Table 4.2). Normal distribution of all variables was verified using Kilmgorov Statistic following removal of outlying data.
Table 4.2: Final number of participants included for the analyses

<table>
<thead>
<tr>
<th>Joint moment</th>
<th>Knee OA</th>
<th>Control</th>
<th>Outliers</th>
<th>Final Number (N)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Knee adduction</td>
<td>40</td>
<td>40</td>
<td>0</td>
<td>80</td>
</tr>
<tr>
<td>Initial knee extension</td>
<td>39</td>
<td>38</td>
<td>3</td>
<td>77</td>
</tr>
<tr>
<td>Early knee flexion</td>
<td>40</td>
<td>38</td>
<td>2</td>
<td>78</td>
</tr>
<tr>
<td>Late knee extension</td>
<td>40</td>
<td>40</td>
<td>0</td>
<td>80</td>
</tr>
</tbody>
</table>

Outliers: Cases excluded if they were beyond the range of three standard deviations from the mean.
Average curves through stance phase for the frontal and sagittal plane moments are displayed in Figures 4.1a and b. The statistical analyses revealed that the average peak knee adduction moments in the first 50% of stance phase was higher in participants with knee OA (2.97 ± 0.90 % Bw*Ht) compared with the control group (2.46 ± 0.62 % Bw*Ht, p<0.05) (Table 4.3). The OA group also had a significantly lower knee extension moment in the late stance phase of the gait cycle (OA group: -0.59 ± 1.04 %Bw*Ht, control group: -1.11 ± 1.0 % Bw*Ht, p<0.05) (Table 4.3). There were no differences in mean peak knee flexion moment or the initial stance knee extension moment between the groups (p>0.05). The findings did not differ when the data from all the outliers was included in a second analysis.

The average peak knee flexion moment in early stance occurred at approximately the same time in both groups (p>0.05) (Table 4.4 and Figure 4.1b). Similar observations were found for the two extension moments in early and late stance phases (p>0.05) (Table 4.4). However, the occurrence of the peak knee adduction moment in the first 50% of stance phase occurred later in stance phase in the OA group (OA group: 29 ± 6.0 % stance phase, Control group: 24.9 ± 2.61 % stance phase, p<0.05).

### 4.1.4 Knee Joint Angle

Data were available for all subjects and there were no outliers. There was no violation of normality as determined by the Kilmogorov statistic. The graph of knee flexion angle during stance is displayed in Figure 4.2. Participants in the two groups had similar knee flexion angle profiles during early stance phase of a gait cycle. Knee flexion
angle in early stance was 14.4°± 4.24 in the OA group and 13.62°± 6.03 in the control group (p>0.05).
Figure 4.1 a: Frontal plane moments through stance phase. The peak adduction moment in the first 50% of stance phase was significantly higher in the OA group (p<0.05).

Figure 4.1 b: Sagittal plane external moments through stance phase. The late peak knee extension moment was significantly lower in the knee OA group. (p<0.05).
Table 4.3 External Frontal and Sagittal Plane Moments and Knee Kinematics

<table>
<thead>
<tr>
<th>Joint moment</th>
<th>Knee OA (n = 40)</th>
<th>Control (n = 40)</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Knee adduction</td>
<td>2.97 (0.90)</td>
<td>2.46 (0.62)</td>
<td>p = 0.004*</td>
</tr>
<tr>
<td>Initial knee extension</td>
<td>-1.14 (0.40)</td>
<td>-1.30 (0.42)</td>
<td>p = 0.11</td>
</tr>
<tr>
<td>Early stance knee flexion</td>
<td>2.40 (1.13)</td>
<td>2.73 (1.40)</td>
<td>p = 0.20</td>
</tr>
<tr>
<td>Late stance knee extension</td>
<td>-0.59 (1.04)</td>
<td>-1.11 (1.0)</td>
<td>p = 0.02*</td>
</tr>
</tbody>
</table>

Mean external moments were reported in %BW*HT with standard deviations in the parentheses, (*) denotes significant differences between groups with p<0.05.

Table 4.4: Joint moment occurrence in Stance Phase

<table>
<thead>
<tr>
<th>Joint moment</th>
<th>Knee OA</th>
<th>Control</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Knee adduction</td>
<td>29% (6.00)</td>
<td>24.9% (2.61)</td>
<td>p = 0.033*</td>
</tr>
<tr>
<td>Initial knee extension</td>
<td>3.2% (2.90)</td>
<td>2.45% (1.80)</td>
<td>p = 0.166</td>
</tr>
<tr>
<td>Early stance knee flexion</td>
<td>24.16% (3.23)</td>
<td>23.8% (3.60)</td>
<td>p = 0.714</td>
</tr>
<tr>
<td>Late knee extension</td>
<td>68.70% (7.33)</td>
<td>68.20% (6.00)</td>
<td>p = 0.599</td>
</tr>
</tbody>
</table>

Reported values were time of occurrence in stance phase with standard deviations in the parentheses, (*) denotes significant differences between groups with p<0.05.
Figure 4.2: Pattern of stance phase knee angle. There were no significant differences between groups in early stance average flexion angle (p > 0.05).
4.2 Low versus Moderate Pain OA Groups

4.2.1 Characteristics of participants with OA

WOMAC pain scores were used in this study to classify participants with OA into two equal groups; a low pain score group with scores below 7/20 and a moderate group with scores equal to or greater than 7/20. Baseline demographic characteristics for the 20 participants in each group are displayed in Table 4.5. The data were examined for outliers, identified as mean values for an individual participant that were three standard deviations or more outside the group mean. There were no outliers in height, weight or BMI and there was no violation of normality as determined by the Kilmogorov statistic. There were no significant differences in age, height, weight, or BMI between groups (p>0.05). Disease severity, as measured by the Cooke scale, and knee alignment also did not differ between groups (p>0.05).

4.2.2 Temporal and Distance Parameters of Gait

Temporal and distance parameters of gait are reported in Table 4.5. Temporal and distance data were not available for all subjects for the reasons provided previously. Stride length, cadence, and DLST data were not available for two subjects in the OA group with low pain scores and one subject in the OA group with moderate pain scores. Gait speed data were not available for one subject in the OA group with low pain scores. No outliers were identified in the temporal and distance data and data for each variable were normally distributed. Independent t-tests confirmed that the OA group with moderate pain scores had a slower gait speed (m/s) and longer double limb support time
(DLST) (p<0.05). There was no difference in the stride length or cadence between groups (p>0.05).
Table 4.5 Demographic characteristics, Knee Alignment, OA Severity, and Temporal and Distance Parameters of Gait of OA groups

<table>
<thead>
<tr>
<th></th>
<th>Low pain score (N =20)</th>
<th>Moderate pain score (N =20)</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (yr)</td>
<td>60.70 (8.52)</td>
<td>65.25 (10.53)</td>
<td>p = 0.141</td>
</tr>
<tr>
<td>Height (m)</td>
<td>1.72 (0.10)</td>
<td>1.74 (0.12)</td>
<td>p = 0.601</td>
</tr>
<tr>
<td>Weight (kg)</td>
<td>78.47 (19.02)</td>
<td>86.15 (20.07)</td>
<td>p = 0.230</td>
</tr>
<tr>
<td>BMI (kg m⁻¹)</td>
<td>26.36 (5.05)</td>
<td>28.40 (5.80)</td>
<td>p = 0.244</td>
</tr>
<tr>
<td>Knee alignment (°)</td>
<td>-3.80 (4.31)</td>
<td>-4.40 (4.39)</td>
<td>p = 0.657</td>
</tr>
<tr>
<td>X-ray grade severity</td>
<td>3.45 (2.11)</td>
<td>4.00 (2.15)</td>
<td>p = 0.420</td>
</tr>
<tr>
<td>WOMAC pain</td>
<td>3.15 (1.78)</td>
<td>7.95 (1.27)</td>
<td>p = 0.000*</td>
</tr>
<tr>
<td>Stride length (m)</td>
<td>1.23 (0.15)</td>
<td>1.15 (0.17)</td>
<td>p = 0.152</td>
</tr>
<tr>
<td>Cadence (Steps/minute)</td>
<td>99.62 (8.38)</td>
<td>95.22 (11.97)</td>
<td>p = 0.206</td>
</tr>
<tr>
<td>DLST (s)</td>
<td>0.35 (0.07)</td>
<td>0.42 (0.07)</td>
<td>p = 0.008*</td>
</tr>
<tr>
<td>Gait speed (m s⁻¹)</td>
<td>1.06 (0.16)</td>
<td>0.93 (0.22)</td>
<td>p = 0.047*</td>
</tr>
</tbody>
</table>

All values mentioned are the mean values with standard deviations in the parentheses.

(*) Significant differences between groups; p<0.05

DLST = Double limb support time

BMI = Body Mass Index

a Negative alignment value represents varus

b Radiographic grading scale from 0 -13
4.2.3 Knee Joint Moments

Data were reviewed for outliers for each of the four moments investigated and individual subject’s data were excluded from the analyses if they were beyond three standard deviations from the group mean. The number of outliers excluded for initial knee extension moment was one from the OA group with low pain scores and two for the knee adduction moment from the OA group with low pain scores. For the remaining variables (knee flexion moment and late knee extension moment) there were no outliers identified. Normal distribution of all variables was verified using Kilmgorov Statistic following removal of outliers. Average curves through stance phase for the frontal and sagittal plane moments are displayed in Figure 4.3. Statistical analyses revealed that there was no difference between groups for peak knee adduction moment in the first 50% of stance, knee flexion moment in early stance or knee extension moment in early or late stance phase. (p>0.05) (Table 4.6). There was no difference between groups in the occurrence of the initial extension or flexion sagittal plane moments (p>0.05) (Table 4.7). However, the late stance knee extension moment occurred later in the moderate pain group. The OA groups also did not differ in the occurrence of average peak knee adduction moment in the first 50% of stance phase (p>0.05) (Table 4.7). Observation of Figure 4.3a, reveals that the OA groups appeared to differ in the mid-stance knee adduction moment (at 50% of stance phase). However, analyses revealed that there were no differences between the OA groups at this point in the gait cycle (low pain score: 0.36 ± 0.12 Nm/kg, moderate pain score: 0.43 ± 0.14 Nm/kg, p>0.05). The findings did not differ when the data from all the outliers was included in second analysis.
Figure 4.3a: Frontal plane joint moment comparison between OA groups. There was no significant difference in the knee adduction moment in the first 50% of stance phase (p>0.05).

Figure 4.3b: Sagittal plane moments through the stance phase of gait in OA groups with low and moderate pain. There were no differences in the peak flexion moment in early stance or in the two extension moments between groups (P> 0.05).
Table 4.6 Comparisons between low and moderate pain score knee OA groups

<table>
<thead>
<tr>
<th>Joint moment</th>
<th>Low pain score (n = 20)</th>
<th>Moderate pain score (n = 20)</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Knee adduction</td>
<td>3.08 (0.60)</td>
<td>2.87 (0.77)</td>
<td>p = 0.37</td>
</tr>
<tr>
<td>Initial stance knee extension</td>
<td>-1.21 (0.45)</td>
<td>-1.11 (0.37)</td>
<td>p = 0.46</td>
</tr>
<tr>
<td>Early stance knee flexion</td>
<td>2.49 (1.00)</td>
<td>2.37 (1.14)</td>
<td>p = 0.70</td>
</tr>
<tr>
<td>Late stance knee extension</td>
<td>-0.75 (1.03)</td>
<td>-0.44 (1.10)</td>
<td>p = 0.36</td>
</tr>
</tbody>
</table>

Mean values were reported in % Bw*Ht with standard deviations in the parentheses.

Table 4.7: Joint moment occurrence in Stance Phase

<table>
<thead>
<tr>
<th>Joint moment</th>
<th>Low pain score</th>
<th>Moderate pain score</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Knee adduction</td>
<td>28.30% (5.50)</td>
<td>30.00% (6.30)</td>
<td>p = 0.378</td>
</tr>
<tr>
<td>Initial knee extension</td>
<td>3.10% (3.13)</td>
<td>3.30% (2.70)</td>
<td>p = 0.830</td>
</tr>
<tr>
<td>Early stance knee flexion</td>
<td>25.23% (3.06)</td>
<td>24.20% (4.70)</td>
<td>p = 0.416</td>
</tr>
<tr>
<td>Late knee extension</td>
<td>66.10% (6.70)</td>
<td>71.30% (7.18)</td>
<td>p = 0.023*</td>
</tr>
</tbody>
</table>

Reported values were time of occurrence in 100% stance phase with standard deviations in the parentheses, (*) represents significant differences between groups (p<0.05).
4.2.4 Knee Joint Angle

Data were available for all subjects and there were no outliers; there was no violation of normality as determined by the Kilmogorov statistic. The graph of knee flexion angle during stance is displayed in Figure 4.4. The average peak knee flexion angle in early stance did not differ between groups (OA with low pain: 14.5°± 4.12, OA with moderate pain: 14.30°± 4.46, p>0.05).
Figure 4.4: Stance phase knee flexion angle through the stance phase in OA groups with low and moderate pain. The average peak knee flexion angle did not differ between groups (p> 0.05).
4.3 Correlation Analyses

4.3.1 Association between external knee flexion moment and knee flexion angle

A Pearson correlation coefficient (r) was used to investigate the relationship between knee flexion angle and the external knee flexion moment in early stance in subjects with knee OA. According to Cohen’s guidelines, there was a strong positive correlation between these two variables \( r = 0.687, n = 40, p<0.05 \), with high levels of external knee flexion moment associated with higher levels of knee flexion angle (Figure 4.5). The correlation between knee flexion moment and knee flexion angle in the control group was \( r = 0.471, p = 0.002 \).

4.3.2 Association between external knee flexion moment and the pain severity

A Pearson correlation coefficient (r) was used to investigate the relationship between knee flexion moment and pain severity (WOMAC pain scores) in early stance in subjects with knee OA. According to Cohen’s guidelines, there was no significant correlation between these two variables \( r = -0.026, n = 40, p = 0.88 \).

4.3.3 Association between external knee flexion moment and gait speed

A Pearson correlation coefficient (r) was used to investigate the relationship between knee flexion moment and the gait speed (m/s) in early stance in subjects with knee OA. According to Cohen’s guidelines, there was no significant correlation between these two variables \( r = 0.021, n = 40, p = 0.20 \).
Figure 4.5: Scatter plot of knee flexion moment and knee flexion angle in early stance phase. A linear association between the knee flexion moment and the peak knee flexion angle in the early stance phase for the knee OA group was demonstrated ($r = .687$; $p<0.05$).
Chapter 5

Discussion

The first hypothesis, that the knee flexion moment in early stance would be lower in the OA group compared to healthy controls and lower in the OA group with moderate pain scores compared to the OA group with low pain scores was not supported in this study. The hypothesis that the knee extension moments would be lower in the OA group compared to controls was partially supported in that the extension moment in late stance was lower in the OA group compared to the healthy control subjects. There was no difference in knee flexion angle in stance between groups. There was no difference between groups in the early stance phase extension moment magnitude.

There was no difference in either extension moment or the knee flexion moment in early stance when comparing the two OA groups. The knee flexion angle in stance also did not differ between the two OA groups. However, there was a significant positive correlation between the knee flexion moment and knee flexion angle in early stance phase in participants with knee OA. There was no significant relationship between the knee flexion moment and gait speed or knee pain as measured by the WOMAC in participants with knee OA.

5.1 Subject Characteristics

It was initially planned that the osteoarthritis and control groups would be matched for age, sex, height, and weight. However, early in the recruitment process it was recognized that matching for height and weight would be very difficult and hence matching was based on only age and sex. The groups did not differ in mean age or height.
However, the osteoarthritis group had higher body weight and higher body mass index (BMI) compared to the control group. Previous researchers have also found that subjects with knee OA have higher BMI than the healthy control subjects.\textsuperscript{45, 47-49} Since high BMI is a risk factor for the development of knee OA\textsuperscript{45, 47-49}, it is not surprising that the OA group had a higher BMI on average compared to control subjects.

The knee OA group in the current study had moderate levels of OA severity as indicated by both pain scores and x-ray grade. The average WOMAC pain score for the OA group was $5.5 \pm 2.9$ and ranged from 1.0 to 11.0; the maximum pain score on the WOMAC is 20. Astephen et al\textsuperscript{31} reported a WOMAC pain score for their moderate OA group of $7.53 \pm 3.94$ and a score of $10.62 \pm 5.82$ for the more severe OA group, indicating that the participants of their moderate OA group might have higher levels of pain and disease severity compared to the OA group with low pain scores in this study. Previous studies used the Kellgren and Lawrence system (K/L system) to grade radiograph severity in subjects with knee OA,\textsuperscript{17, 31, 66, 79} whereas the Cooke et al\textsuperscript{82} scale (maximum of 13) was used for the current study. According to this scale\textsuperscript{82}, the OA group had moderate levels of severity ($3.7 \pm 2.1$).

Varus alignment scores for the OA group in the current study were significantly higher than for the control group. This finding is not surprising since varus alignment is a consequence of medial compartment knee OA. Scores in the OA group ranged from $-12.7^\circ$ to $2.9^\circ$ with a mean varus angle of $-4.1^\circ \pm 4.3$ degrees. This is similar to the varus alignment scores reported by previous investigators for subjects with medial knee OA. Miyazaki et al\textsuperscript{17} reported varus alignment scores for their OA group without disease progression of $-4.6^\circ \pm 3.8$ degrees. Cerejo et al\textsuperscript{66} reported a range of varus
alignment scores from -8.00° to -1.00° and a mean score of -3.04 ± 1.95 degrees for their moderate OA group (K/L grade 2).

There were no differences in age, height, weight, or BMI between the two groups with knee OA when divided based on WOMAC pain scores (p>0.05). Similarly, x-ray grade severity and knee joint varus alignment did not differ between groups (p>0.05). OA group classification into those with low and moderate pain score OA groups was somewhat arbitrary as it was based on the WOMAC pain score that divided the group into an equal number using a cut score of 7. It is not surprising that the division based on pain did not distinguish the groups based on x-ray severity, as it is known that pain scores do not correlate well with x-ray severity grade.2, 3

5.2 Gait Characteristics

Comparison between the knee OA group and the control group revealed that the OA group had a lower gait velocity (knee OA = 1.0 ± 0.20 m/s, control = 1.12 ± 0.20 m/s). Gait speed is a product of cadence (steps/minute) and stride length (m).23, 24

The average stride length did not differ between groups (p>0.05), however cadence was significantly lower for the OA group (knee OA = 97.36 ± 10.5 steps/min, control = 104.60 ± 9.3 steps/min) and likely contributed to the lower gait speed (p<0.05). The higher double limb support time found in the OA group (knee OA = 0.40 ± 0.08 sec, control = 0.33 ± 0.05 sec, p<0.05) can be explained by the lower gait speed in this group. The average gait speed in the control group was lower than reported for the healthy adults.23 This may related to the relatively short walkway in the laboratory, the attachment of IREDs at various locations and the fact that they are being tested in a strange environment.
These findings are consistent with the previous literature. Deluzio and Astephen\textsuperscript{26} reported gait speeds of $0.76 \pm 0.08$ m/s in subjects with knee OA compared to $0.95 \pm 0.14$ m/s, for the control group ($p<0.05$). In a similar study, Brinkmann and Perry\textsuperscript{75} found that their knee OA group walked slower than the control group (knee OA: $0.63 \pm 0.2$ m/s, control: $1.33 \pm 0.16$ m/s, $p<0.01$). Stauffer and associates\textsuperscript{73} also reported lower cadence in people with knee OA (70.8 steps per minute) compared to control subjects (94.5, steps per minute; $p<0.05$). They also reported longer stance time as a percentage of the gait cycle in the knee OA group (62.8%), compared to control subjects (56.6%; $p<0.05$) which they attributed to a longer double support period. According to these authors\textsuperscript{73}, reducing gait speed and increasing double limb support time are pain-relieving strategies that work to reduce the compressive load on the knee joint.

OA subjects with moderate pain scores had a slower gait speed than the OA subjects with low pain scores ($p<0.05$) in the current study. Although stride length and cadence were both lower in the OA groups neither measure was significantly different between groups ($p>0.05$). Double limb support time was longer in the OA group with moderate pain scores compared to the OA group with low pain scores reflecting a lower gait speed ($p<0.05$). Similarly, Astephen et al\textsuperscript{31} found that people with moderate OA walked more quickly than those with more severe knee OA ($1.25 \pm 0.22$ m/s and $0.92 \pm 0.24$ m/s respectively, $p<0.0001$). Astephen et al\textsuperscript{31} also reported greater stance time in the more severe OA group ($0.85 \pm 0.14$ sec, $p<0.0001$) compared to the moderate OA ($0.73 \pm 0.09$ sec).
Although the OA groups in the current study did not differ in demographic characteristics, x-ray grade, or knee alignment, OA subjects with moderate pain scores walked slower than the OA subjects with low pain scores. Decreased gait speed is a strategy to decrease knee joint load and hence pain. Since the groups were divided based on pain, it is not surprising that the OA group with moderate pain walked more slowly on average than the OA group with low pain.

5.3 Knee Adduction Moment

The comparison of the OA and healthy control groups revealed that the knee adduction moment in the first half of stance phase was higher on average in the OA group (knee OA: 2.97 ± 0.90 % Bw*Ht; control: 2.46 ± 0.62 % Bw*Ht, p<0.05) and occurred significantly later in the stance phase. The corresponding values in Nm/kg were determined to allow comparison to other studies and were 0.50 ± 0.15 Nm/kg and 0.40 ± 0.10 Nm/kg in the OA and control groups respectively. These findings were consistent with previous literature. Gok et al27 reported an adduction moment in a group with knee OA of 0.45 ± 0.11 Nm/kg, and a moment of 0.33 ± 0.05 Nm/kg, p<0.05 in the control group during the first half of the stance phase. In a similar study, Sharma and associates16 reported knee adduction moment values of 3.0 ± 1.1% Bw*Ht during the first half of the stance phase in people with knee OA with K/L grades from 0-2. Knee OA groups in both the Gok et al27 and Sharma et al16 had moderate levels of OA severity.

Differences in the knee adduction moment between the knee OA and control groups might be attributed to differences in the knee varus alignment between groups. During gait in neutrally aligned knee joints, load is disproportionally transmitted to the medial side of the knee joint. The knee adduction moment is a product of the
ground reaction force and the perpendicular distance of this force from the joint axis. Any increase in varus alignment has the potential to increase the moment arm and therefore the magnitude of the knee adduction moment. A strong association exists between varus alignment and knee adduction moment in both healthy and knee OA subjects. Andrews and associates reported a significant positive relationship between varus alignment (mechanical axis) and first peak knee adduction moment in normal subjects (r = 0.69, n = 11, p<0.05). Hsu et al reported similar findings in healthy subjects (r = 0.824, n = 120, p<0.05). Furthermore, Hurwitz et al reported a similar relationship between varus alignment (mechanical axis) and first peak knee adduction moment in subjects with medial knee OA (r = 0.74, n = 62, p<0.05). Varus alignment of more than 3° (joint malalignment) has been reported to further increase the load passing through the medial compartment of the knee joint.

The knee adduction moment in the first 50% of stance did not differ between the two knee OA groups. It appeared from Figure 4.3a that the OA group with moderate levels of pain had a higher mean adduction moment through the middle of stance phase. Results of a second analysis comparing the adduction moment average between groups at 50% of stance did not support this observation. The finding of no difference in adduction moment between OA groups is supported by Astephen et al who also reported no differences in the knee adduction moments between OA groups of varying severity. The fact that the varus alignment did not differ between the two OA groups in the current study may explain the similar knee adduction moment values in the two groups.
5.5 Sagittal Plane Moments

Both the OA and control group demonstrated an external knee extension moment immediately following foot contact. There was no difference in the magnitude of this moment between the knee OA and control groups (p>0.05). The values in the current study were -1.14 ± 0.40 % Bw*Ht for the OA group and -1.30 ± 0.42 % Bw*Ht in the control group (p>0.05). The corresponding values in Nm/kg were -0.19 ± 0.07 and -0.21 ± 0.07 in the OA and control groups respectively (p>0.05). Manetta and associates²⁹ also reported no difference in the initial extension moment between knee OA and healthy control subjects (p>0.05). However, Astephen and associates³¹ reported lower extension moments following foot contact for the group with more severe OA group compared to the healthy control group (more severe OA: -0.14 ± 0.08 Nm/kg, control: -0.31 ± 0.14 Nm/kg, p<0.002) and the group with moderate OA compared to healthy controls (moderate OA: -0.26 ± 0.10 Nm/kg, control: -0.31 ± 0.14 Nm/kg, p<0.002). A possible explanation for the non-significant findings in the current study is that the severity of knee OA was lower than both the moderate and severe groups in the study by Astephen et al³¹ as indicated by lower pain scores.

In the current study, there were no differences in knee extension moment following foot contact between the two OA groups (OA with low pain score: -1.21 ± 0.45 % Bw*Ht, OA with moderate pain score: -1.11 ± 0.37 % Bw*Ht, p>0.05). The corresponding values in Nm/kg were -0.20 ±0.08 for the OA group with low pain scores and -0.19 ± 0.06 Nm/kg in the OA group with moderate pain scores. Astephen and associates³¹ reported lower knee extension moments for the more severe knee OA group than the moderate knee OA group (moderate: -0.26 ± 0.10 Nm/kg, more severe: -0.14 ±
0.08 Nm/kg, p<0.002). The explanation for the differences between the studies may be that participants in the current study had less severe knee OA overall. The division into groups was arbitrarily based on a cut score of seven on the WOMAC. The average pain score of the moderate pain group was only 7.95 ± 1.27 compared to 3.15 ± 1.78 in the low pain group (p<0.05).

The knee flexion moment in stance also did not differ between the OA and healthy control group (knee OA: 2.40 ± 1.13 % Bw*Ht, control: 2.73 ± 1.40 % Bw*Ht, p>0.05) or between the two OA groups (OA with low pain score: 2.49 ± 1.00 % Bw*Ht, OA with moderate pain score: 2.37 ± 1.14 % Bw*Ht, p>0.05). The values in the current study (in Nm/kg) were 0.41 ± 0.19 for the knee OA group and 0.45 ± 0.23 in the control group (p>0.05). For the OA group with low pain scores the knee flexion moment in Nm/kg was 0.42 ± 0.17 and 0.40 ± 0.19 in the OA group with moderate pain scores (p>0.05).

This finding did not support the hypothesis that the knee OA group would have a lower flexion moment compared to controls. This was in agreement with previous literature that compared subjects with moderate OA to that of healthy control subjects. Manetta and associates reported a knee flexion moment of 0.25 ± 0.18 Nm/kg in subjects with knee OA and 0.23 ± 0.22 Nm/kg in healthy control subjects (p>0.05).

Previous authors have reported lower knee flexion moments in early stance in people with knee OA compared to healthy control subjects. Astephen et al reported that the group with more severe OA had a lower knee flexion moment in the early stance (0.33 ± 0.20 Nm/kg) than the healthy control group (0.52 ± 0.29 Nm/kg,
p<0.05). Furthermore, they also showed differences in the flexion moment between the moderate OA and healthy control groups (moderate OA: 0.40 ± 0.22 Nm/kg, control: 0.52 ± 0.29 Nm/kg, p<0.002). However, there were no differences in the flexion moment between the two OA groups in the Astephen et al’s study. The fact that the OA groups in the Astephen et al study had similar levels of joint pain (p> 0.0001), may have resulted in the similar flexion moments between the two severity groups. Further analyses revealed that their severe knee OA group also had a lower average peak flexion angle in early stance phase (8.04° ± 6.22 degrees) compared to the control group (18.72° ± 7.28, p<0.05). The lower knee flexion angle would likely reduce the moment arm and hence the knee flexion moment at this point in the gait cycle. In the current study, the knee OA group had a similar knee flexion angle in early stance (14.4° ± 4.24 degrees) to that of healthy control subjects (13.62° ± 6.03, p>0.05). Subjects in the current study also had lower severity as indicated by pain and radiographic scores and hence may have been less likely to need to adopt compensatory gait strategies to reduce pain. Reduction in gait speed may be sufficient compensation in people with lower severity of OA.

Correlation analyses revealed that there was a strong positive association between the knee flexion moment and the knee flexion angle in the early stance phase in the group with knee OA (r = 0.687, p<0.05). Although lower, a positive correlation between knee flexion moment and knee flexion angle was also found in the control group. These findings support the argument that the knee flexion moment can be reduced by keeping the knee in greater extension in the early stance phase of the gait cycle.

However, the knee flexion moment was not correlated with gait speed or pain scores in subjects with knee OA in the current study. Since the knee OA group had
similar knee flexion angles to that of healthy control subjects, joint pain and gait speed might not be confounding factors affecting the knee flexion moment in early stance.

In contrast, Schipplein and Andriachhi\textsuperscript{18} and Al-Zahrani and Bakheit\textsuperscript{28} reported higher flexion moments in early stance in OA subjects compared to healthy controls. Schipplein and Andriachhi\textsuperscript{18} compared 19 subjects with knee OA (mean age 55 years; mean varus deformity of 9°) to 15 healthy control subjects (mean age 62 years). The values for the knee OA group and control group were 3.32 ± 1.86 % Bw*Ht and 1.81 ± 0.65 % Bw*Ht respectively (p<0.01). The authors argued that the group with knee OA adapted their gait to produce a higher external flexion moment. The higher moment was suggested to increase muscle force and joint compressive forces, required by subjects with knee OA for knee joint stability. According to the authors, this style of gait, which demands more muscle forces, could be used as a compensatory mechanism to increase the joint stability. Eight of the subjects in this study had a mean knee flexion contracture of between 5 and 15 degrees and 11 subjects had between zero and 5 degrees of contracture. The knee flexion angle in stance phase was 25±19 degrees compared to 20.41± 5.28 degrees in the control group. It is likely that the higher knee angle in stance phase contributed to the higher flexion moment in stance due to the increase in moment arm.

Comparison of the OA and control group in the current study revealed a significantly lower knee extension moment in late stance phase in the OA group (-0.59 ± 1.04 % Bw*Ht) compared to the control group (-1.11 ± 1.00 % Bw*Ht, p<0.05). The corresponding values in Nm/kg were -0.01 ± 0.18 for the knee OA group and -0.18 ± 0.17 in the control group (p<0.05). Astephen et al\textsuperscript{31} also reported a lower extension moment for the more severe OA group (-0.10 ± 0.19 Nm/kg) compared to the control group (-0.42
± 0.13 Nm/kg, p<0.05). However, there were no differences in late stance knee extension moment between their moderate OA group (-0.35 ± 0.21 Nm/kg, p<0.05) and their control group. Messier and associates\textsuperscript{98} suggested that subjects with knee OA reduce the knee extension moment in late stance and hence knee compressive forces by decreasing their walking speed in response to pain. Thus the lower gait speed in the OA group than the control group (p<0.05) may have resulted in the lower knee extension moment in late stance phase. Statistical analyses revealed a positive correlation between gait speed and late stance knee extension moment in the combined control and OA subjects (r = 0.23, n =80, p<0.05) and in the OA group (r = 0.33, n = 40 p<0.05). This finding supports the argument that gait speed has some influence on the extension moment at this point in stance phase.

The knee extension moment in late stance did not differ between OA groups in the current study (OA with low pain scores: -0.75 ± 1.03 % Bw*Ht, OA with moderate pain scores: -0.44 ± 1.10 % Bw*Ht, p>0.05). The corresponding values in Nm/kg were -0.13 ± 0.17 for the OA group with low pain scores and -0.07 ± 0.18 in the OA group with moderate pain scores (p>0.05). Asteph\textsuperscript{31} en et al\textsuperscript{31} did report differences in the late stance phase knee extension moment between OA groups of varying severity (moderate OA: -0.35 ± 0.21 Nm/kg, more severe OA: -0.10 ± 0.19 Nm/kg, p<0.05). The OA group with moderate pain scores in the current study did walk more slowly than the OA group with low pain scores and therefore, if gait speed influences this moment as suggested above, a lower knee extension moment would be expected in the OA group with moderate pain scores. In looking at the mean values and Figure 4.3b, there does
appear to be a trend toward a lower knee extension moment in late stance in the OA group with moderate pain scores.

5.6 Limitations

Compensatory gait mechanisms have been previously reported in subjects with knee OA such as lateral trunk lean towards the affected stance limb and toe-out gait.\textsuperscript{30, 99, 100} These mechanisms were found to reduce knee adduction moment. However, we did not measure these parameters in our sample of participants.

A second limitation of this study was the relatively mild to moderate disease severity in the knee OA group and the relatively small sample size when the OA group was divided based on pain scores. Astephen et al\textsuperscript{31} compared OA groups of varying severity with healthy control subjects and found that both moderate and severe OA groups differed from each other and from the healthy control group in early stance knee flexion moment (p<0.05). The sample size was larger and the range of OA severity was greater with more subjects in the higher severity range. With higher levels of knee pain, subjects would be more likely to minimize the knee flexion angle in early stance, thereby reducing the moment arm and the knee flexion moment.

Another limitation of the current study is the arbitrary classification of our OA group into two equal groups based on a WOMAC pain score that divided the group in two equal subgroups in terms of number of subjects. Although the OA group with moderate pain scores walked more slowly than the OA group with low pain scores, this division did not result in any other significant differences between the two groups. It could be argued that both groups had relatively mild to moderate knee OA. The division
of the OA group based on WOMAC pain scores may not have resulted in heterogeneous
groups in terms of severity.

5.7 Conclusion

In conclusion, the initial peak extension moment and peak flexion moment
in early stance did not differ between subjects with knee OA and healthy control subjects.
However, the group with knee OA had a lower mean late stance knee extension moment
than the healthy control group. There was no difference in knee flexion angle in early
stance between the OA and control group.

Although the OA group with moderate pain scores walked more slowly
than the OA group with low pain scores, there were no differences in knee joint kinetics
or kinematics between the two groups. There was a positive relationship between knee
flexion moment and knee flexion angle in early stance in the knee OA group but no
significant relationship between knee flexion moment and pain score or gait speed in this
group.
References


Appendix 1

School of Rehabilitation Therapy

Queen’s University

Kingston, Ontario

CONSENT FORM

TITLE OF PROJECT: The influence of a home program of hip abductor exercises on gait parameters and muscle strength in persons with knee osteoarthritis

BACKGROUND INFORMATION

Overview of the Study: You are being invited to participate in a research study directed by Elizabeth Sled, PhD candidate, and Dr. Elsie Culham, Faculty Advisor, in the School of Rehabilitation Therapy. This study will examine the influence of a home program of hip exercises on walking patterns and hip muscle strength in people with knee osteoarthritis. The muscles of the hip and thigh may have an effect on the forces acting at the knee joint by controlling the position of the pelvis and/or by acting as lateral stabilizers for the knee. Research suggests that the function of the hip muscles during walking may be decreased in people with knee osteoarthritis. Therefore, strengthening the hip muscles may be an effective strategy for reducing stress on the arthritic knee. Elizabeth Sled will read through this consent form with you. She will describe procedures in detail and answer any questions you may have.
DETAILS OF THE STUDY

**Aim of the Study:** To determine the influence of an eight-week home program of exercises for the hip muscles on walking patterns and hip muscle strength in people with knee osteoarthritis.

We invite you to participate as part of the knee osteoarthritis group if you have been diagnosed with knee osteoarthritis by your doctor, are 40 years of age or older, have knee pain on most days of the month and have at least some difficulty in daily function due to your knee pain. You are invited to participate as part of the control group if you have no diagnosis of knee or hip osteoarthritis and no history of hip or knee injury or pain.

**Description of the Testing**

Initial Testing Session: Testing will be conducted in the Motor Performance Laboratory at the School of Rehabilitation Therapy and in the Radiology Department at Kingston General Hospital. The initial testing session will last approximately 2 – 2.5 hours and will involve the following measures:

1) *Questionnaires:* You will be asked to complete two brief questionnaires, which will obtain information about your physical activities and your knee pain during daily function.

2) *Baseline measurements:* Your weight and height will be measured using a regular weigh scale and tape measure.

3) *Walking Performance:* You will be asked to wear shorts for the walking tests. Surface markers will be positioned on your skin at the foot, ankle, knee, hip, lower back and base
of the neck, and straps will be used to hold the markers in place. You will be asked to walk along an 8-metre indoor walkway at a comfortable speed wearing your normal footwear. Two large camera systems will detect the movement of the markers as you walk across the floor. You will be provided with rest breaks in between walking trials. We will collect five good walking trials from each side of the body.

4) **Hip Muscle Strength Testing:** You will stand with your back supported against the muscle strength testing device. A padded cuff will be positioned around your lower thigh just above your knee. You will be asked to keep your knee straight and to take your leg out to the side and back to the midline in a small range of movement while providing maximum effort against the resistance of the machine. Testing will be performed at a comfortable speed and 5 repetitions will be completed. You will also be asked to push against the cuff without any movement of your leg, holding for 5 seconds 3 times. The tests will then be repeated for the opposite leg. You will be given a 2-minute rest between tests.

5) **Physical Performance Measures:** You will be asked to rise from a regular chair and then sit again as fast as you can for 5 repetitions.

**Exercise Program:** If you are a participant in the group with knee osteoarthritis, you will be taught a home exercise program. An additional 30 minutes will be required after the testing for one of the researchers, a physical therapist, to teach you several simple exercises for the hip muscles. You will be instructed in how to contract your hip muscles during walking, stepping up on a step and standing on one leg. You will also be taught a side-lying leg lift exercise using elastic tubing around your lower thigh to provide
resistance. You will be asked to perform these exercises four times per week (on alternate days) for 8 weeks.

For those participating in the exercise program, two follow-up visits with the physical therapist will occur in the laboratory or in your home (your preference) during the 8-week program. These visits will last about 30 minutes. The physical therapist will review your exercise program with you and teach you how to progress the exercises. You are also encouraged to contact the physical therapist by phone if you have any questions or concerns during the 8-week exercise period. If you are part of the control group of participants without knee osteoarthritis you will be instructed to continue your daily activities, but not to begin any new exercise program, over the 8 weeks after the initial testing session.

*X-rays:* If you have not received a recent knee X-ray, you will be asked to have an x-ray taken on the date of your first visit or on a separate day at Kingston General Hospital. You will be required to stand on a turntable with your knees ahead and your feet positioned on markers. Your hips will be supported by adjustable pads to help maintain the position. A hand rest is available for support. You will be asked to distribute your weight evenly on both legs and to keep still during the sequence of x-rays. Front views of your knees will be taken. We will use these x-rays to grade the level of arthritis in your knee joint and to determine how your knee is aligned. The x-rays from the control group will be used to confirm the absence of knee osteoarthritis (as osteoarthritis may be present in people without knee pain) and to obtain the measures of knee alignment for comparison with those with osteoarthritis.
If you have had a recent knee x-ray, with your consent we will view these x-rays to grade the level of knee arthritis and determine your knee alignment.

Final Testing Session: All participants will be asked to return to the Motor Performance Laboratory 8 weeks after the initial testing session. You will complete the questionnaires again. We will also re-test your walking performance, hip muscle strength and sit-to-stand performance.

**Risks**

There are no known risks associated with the procedures used in this study. The radiation dosage for knee x-rays is well within safe limits, provided that participants have not been exposed to large amounts of radiation over the past year. You may experience some mild muscle soreness and fatigue with strength testing of the hip muscles and measurement of your walking performance. If you are participating in the home exercise program you may also experience mild muscle soreness and fatigue with the hip exercises.

**Benefits**

If you are participating in the home exercise program, you may experience a decrease in knee pain and/or improved ability to perform your daily activities as a result of the exercises. The findings from this study may improve our understanding of the role of hip strengthening exercises as part of rehabilitation programs for people with knee osteoarthritis. Thus, the results of this study may benefit those with knee osteoarthritis in the future. There are no expected benefits for those participating as part of the control group.
Exclusions

You will not be considered for this study if you present with any of the following:
corticosteroid injection into either knee within the previous three months; other
significant medical problems (including significant heart disease, stroke, and active
treatment for cancer) that would prevent you from being able to perform a hip exercise
program or to participate in tests of walking performance and hip muscle strength; known
osteoarthritis or previous trauma affecting one or both hips; and previous replacement of
any joint in the lower extremities. You will not be considered if you are receiving
rehabilitation services for knee osteoarthritis or performing a hip strengthening program
at the time of testing.

Confidentiality

All information obtained during this study will be strictly confidential. Your anonymity
will be protected at all times. A file number will be assigned to your data and this
information will be kept in a secure location. Data will be stored in locked files and will
only be available to the Principal Investigator and Faculty Advisor. You will not be
identified in any publications or reports.

Voluntary Nature of Study / Freedom to Withdraw or Participate

Your participation in this study is voluntary. You may withdraw from this study at any
time and your withdrawal will not affect your future medical care with your physician or
at Kingston General Hospital or Hotel Dieu Hospital.

Statement of Subject and Signature
I have read and understand the consent form for this study. I have had the purposes, procedures and technical language of this study explained to me. I have been given sufficient time to consider the above information and to seek advice if I chose to do so. I have had the opportunity to ask questions which have been answered to my satisfaction. I am voluntarily signing this form. I will receive a copy of this consent form for my information.

If at any time I have further questions, problems or adverse events, I can contact:

Elizabeth Sled, Principal Investigator, (613) 533-6000, ext. 75593 OR Dr. Elsie Culham, Acting Director and Faculty Advisor, School of Rehabilitation Therapy, Queen’s University, at (613) 533-6727. If I have questions regarding my rights as a research participant, I can contact: Dr. Albert Clark, Chair, Research Ethics Board, at 533-6081

By signing this consent form, I am indicating that I agree to participate in this study. I am aware that I may refuse to participate or withdraw at any time for any reason without any penalty to the care I will receive.

___________________________ _________________
Signature of Subject                       Date

___________________________ _________________
Signature of Witness                      Date

Statement of Investigator:

I have carefully explained to the participant the nature of the above research study. I certify that, to the best of my knowledge, the participant understands clearly the nature of the study and demands, benefits, and risks involved to participants in this study.

___________________________ _________________
Signature of Investigator  Date