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Characterizing Knee Loading Patterns in People Post-Stroke

Stephanie Marrocco
The University of Western Ontario

Supervisor
Dr. Kara K. Patterson
The University of Western Ontario

Graduate Program in Health and Rehabilitation Sciences
A thesis submitted in partial fulfillment of the requirements for the degree in Master of Science

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(Integrated Article)

by

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The School of Graduate and Postdoctoral Studies
The University of Western Ontario
London, Ontario, Canada

Abstract

Post-stroke temporal gait asymmetry (TGA) could cause excess loading of the non-paretic leg which may cause musculoskeletal (MSK) issues in the knee. This thesis investigated knee loading during gait and its relationship to TGA post-stroke. *Methods*: Gait analysis was conducted in nine people post-stroke. Gait was classified as symmetric (TGS) or asymmetric and external knee adduction (KAM) and flexion moments (KFM) were calculated. Participants were compared to healthy older adults (n=109) on KAM and KFM. The proportion of individuals with increased loading was compared between the TGS and TGA groups. *Results*: Six and four participants exhibited increased loading of the non-paretic and paretic leg respectively. TGA and TGS groups did not differ in the proportion of individuals with increased loading. *Conclusion*: People post-stroke exhibited increased loading of the paretic and/or non-paretic leg but it was unrelated to TGA. Future work should investigate secondary MSK issues post-stroke in a longitudinal study.

Keywords

Temporal gait asymmetry, Stroke, Lower limb loading, Knee adduction moment, Knee flexion moment, Musculoskeletal injury

Co-Authorship Statement

Lucas Crosby – Assisted with data collection

Ian Jones – Assisted with data collection and processing

Dr. Trevor Birmingham – Aided interpreting results & advisory committee member

Dr. Bert Chesworth – Advisory committee member

Dr. Kara Patterson – Supervisor

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Table of Contents

List of Tables

List of Figures

List of Appendices

List of Abbreviations

CMSA	Chedoke-McMaster Stroke Assessment
GRF	Ground Reaction Force
KAM	Knee Adduction Moment
KFM	Knee Flexion Moment
MSK	Musculoskeletal
NIHSS	National Institute of Health Stroke Scale
OA	Osteoarthritis
PKAM	Peak Knee Adduction Moment
pVGRF	Peak Vertical Ground Reaction Force
TGA	Temporal Gait Asymmetry
TGS	Temporal Gait Symmetry
TL	Trunk Lean
TO	Toe-Out

1.0 Introduction

1.1 Disability Post-Stroke

Although cardiovascular disease has been on the decline in the past 50 years, as of 2009 heart disease and stroke still remain two of the three leading causes of death in Canada, respectively, behind cancer. Stroke accounts for 20% of all cardiovascular deaths in Canada, approximately 11,276 deaths in 2007[1]. It is estimated that someone in Canada suffers from a stroke every 10 minutes [2], of whom only 10% recover fully. This has resulted in approximately 315,000 Canadians living with the effects of stroke. This estimate excludes those who have had a stroke and are now residing in an assisted living facility, or other institution[3].

Living with the effects of stroke can vary between individuals. Common effects of stroke include: paralysis or weakness on one side of the body; spasticity; vision problems; trouble speaking or understanding language; inability to recognize or use familiar objects; tiredness; depression; exaggerated or inappropriate emotional responses; difficulty learning and remembering new information; changes in personality; and/or problems with coordination and balance[4–7]. Stroke is also associated with lowered aerobic capacity, increased muscle fatigue, and muscle atrophy, resulting in reduced cardiovascular health[4,8]. These deficits can lead to limited function and independence often manifested in difficulty performing daily activities such as walking, getting up from a chair, bathing and dressing[8–10]. Of all these deficits and impairments, improvement of walking is the number one rehabilitation goal stated by people with stroke[11]. Considering the importance to individuals with stroke and the fact that walking impairment is associated with decreased independence and quality of life[12], investigating walking dysfunction post-stroke is a critical research priority.

1.2 Post-Stroke Gait

Stroke can cause a variety of gait deficits including reduced gait speed[11,13,14], reduced cadence[15], increased time spent in double limb support[16], worsened balance, which can increase the risk of falls, [17,18] and temporal and spatial gait asymmetries[19–21]. Post-stroke gait is also associated with increased fatigue due to increased energy cost of walking[4,20].

As previously mentioned, regaining walking ability is the primary rehabilitation goal, stated by people post-stroke[11]. Rehabilitation can help stroke survivors regain some function by developing strength and confidence to help these individuals live as independently as possible, although the level of function regained varies[3]. Rehabilitation should begin as soon as the patient is stable, to optimize recovery, avoid the reoccurrence of stroke and increase mobility[22]. After rehabilitation, it has been found that 22% of survivors have not regained walking ability, 14% require some kind of assistance while walking and 64% are able to walk independently[23]. However, these numbers give an inaccurate impression about the level of walking disability individuals with stroke exhibit after rehabilitation. Although "independence", defined as "the ability to walk without the assistance of another individual" is achieved, most individuals with stroke are left with significant walking deficits as evidenced by reduced gait velocity[24].

1.2.1 Temporal Gait Asymmetry Post-Stroke

In addition to reduced gait velocity, the majority of individuals with stroke exhibit gait asymmetry after rehabilitation. In a healthy population, gait tends to be symmetrical in both spatial and temporal aspects and this does not change with increasing age alone[25]. For this reason, gait symmetry is a good indicator of impairments resulting from stroke, independent of the person's age[25]. Spatial aspects of gait refer to left and right joint angles, step lengths and

stride lengths. Temporal aspects of gait refer to swing times, stance times, step and stride times. In contrast, post-stroke gait can be characterized as asymmetric in both temporal and spatial aspects. Spatial asymmetry post-stroke is qualitatively characterized as uneven step lengths taken by the paretic and non-paretic lower limbs. Temporal gait asymmetry is qualitatively described as prolonged stance time on the non-paretic side and prolonged swing time on the paretic side. Temporal and spatial gait asymmetry are exhibited by 55.5% and 33.3% of people with chronic stroke, respectively[19].

Gait asymmetry can be calculated using a variety of different equations with different gait parameters from the left and right limbs (e.g. swing time, step length, joint angles, ground reaction forces)[26]. Symmetry of spatiotemporal gait parameters is often used because these parameters are clinically feasible to measure (compared to joint angles and ground reaction forces), easily interpreted and can be used to direct treatment[26,27]. A systematic evaluation of these various equations and parameters resulted in recommendations for a standard measure of asymmetry: the use of the ratio to calculate symmetry with paretic and non-paretic limb values for 1) swing time, 2) stance time and 3) step length[26]. Based on these recommendations this thesis will use the swing time symmetry ratio as a measure of temporal gait asymmetry.

Temporal gait asymmetry has been related to several stroke-related deficits and clinical characteristics. For example, Kim and Eng[21] found that temporal asymmetry is correlated with increased ground reaction force (GRF) through the non-paretic leg[21]. It has been previously found that temporal asymmetry is correlated with gait velocity, although the relationship was non-linear[19]. For this reason, gait symmetry should be a clinical measure, along with gait velocity to measure overall gait performance, and to measure someone's overall walking performance[26]. Motor impairment of the leg and foot (as measured by the Chedoke McMaster

Stroke Assessment [CMSA]) also appear to be correlated with temporal gait asymmetry[19] and explains some, but not all of the variance observed in the stroke population. There have been few studies that investigate the relationship between lesion location post-stroke and gait asymmetries. A study by Alexander[28] found that the posterolateral putamen is more likely to be lesioned in those individuals exhibiting temporal gait asymmetry compared to those individuals post-stroke with symmetric gait[28]. There is also evidence of increased asymmetries in those with neglect[28,29].

Achieving symmetry in gait is a common rehabilitation goal[30]. Therapists may emphasize symmetrical gait in therapy because they are aware (at least intuitively) that gait symmetry is correlated with gait independence, allowing for more effective ambulation in the community[31]. Temporal gait asymmetry is of concern because it has been linked to a number of proposed negative consequences. These proposed consequences (outlined below) have some support from studies directly investigating post-stroke gait and other consequences have indirect support from investigations in related populations that exhibit temporal gait asymmetry (e.g. lower limb amputee population). Temporal gait asymmetry has been linked to increased metabolic and mechanical costs of gait[32], increased pain in the non-paretic leg[33], and decreased muscle activity on the paretic side[34]. Temporal gait asymmetry has also been indirectly linked to musculoskeletal complications, including decreased bone density of the femoral neck on the paretic side, which can increase the risk of fractures[34] and joint pain and degeneration in the non-paretic leg[33,35]. In addition to these potential negative consequences, of concern is the fact that temporal gait asymmetry changes very little over the inpatient rehabilitation period[36]. In a longitudinal study of individuals undergoing inpatient stroke rehabilitation, 59% of individuals exhibited temporal gait asymmetry at admission and 79% of those individuals remained

asymmetric at discharge. This means that these individuals may be at risk to develop the secondary consequences outlined above after they have left rehabilitation care. Adding to this concern is the fact that temporal gait asymmetry may get worse in the long term. Turnbull[37] found that the asymmetric pattern of post-stroke gait is accentuated over a ten year period[37]. This is consistent with findings in a cross-sectional study by Patterson et al[38], which found that swing time, stance time, and step length asymmetries are worse in individuals in the later stages of stroke compared to those in the subacute stage. Given the secondary complications that could arise, temporal gait asymmetry is clearly a significant cause for concern and should be an area of continued focus both clinically and in research. A significant amount of work has characterized the prevalence, nature and underlying factors related to temporal gait asymmetry post-stroke. By comparison, little work has been done to link temporal gait asymmetry directly to the proposed secondary negative consequences. This thesis will be the first step in linking temporal gait asymmetry to musculoskeletal (MSK) issues in the lower limbs post-stroke.

1.2.2 Temporal Gait Asymmetry, Increased Loading of the Lower Limb and Musculoskeletal Issues

The evidence for a link between temporal gait asymmetry and MSK issues in the lower limbs comes from the amputee population. MSK issues are prevalent in the amputee population; seventy-one percent of people with unilateral amputation report pain in their intact limb and/or back[39]. Novell found that men with transtibial and transfemoral amputations were twice as likely to develop pain in their intact limb, when compared to healthy controls[33]. Previous work has shown that this increased prevalence of MSK issues and pain in the legs of individuals with lower limb amputations is related to their gait pattern. Individuals with lower limb amputations walk with an asymmetric gait pattern, spending longer in stance on their intact limb compared to

their amputated limb[40,41]. This pattern is similar to those post-stroke; spending longer time in stance on their non-paretic limb compared to their paretic limb[19]. This temporally asymmetric gait pattern causes loading abnormalities. Nolan[35] found that the temporally asymmetric amputee gait could be responsible for an increase in vertical ground reaction force (VGRF) in the intact limb. Increased forces through the lower limb has been shown in other studies to lead to pain/degeneration at the knee, in the amputee population[33,35]. Increased load on the knee has also been linked to an increased risk of developing knee osteoarthritis (OA) and knee pain/degeneration[42]. Given that individuals with stroke exhibit the same temporally asymmetric gait pattern, and that Kim and Eng[21]demonstrated that this pattern is correlated with increased GRF through the non-paretic limb, it is plausible that individuals with stroke are also more likely to develop MSK issues in the lower limbs. Considering the risk of developing OA and chronic knee pain, lower limb loading patterns in people post-stroke should be further examined.

1.3 Measures of Knee Loading

In order to investigate loading in the lower limbs post-stroke and the risk for the development of secondary MSK issues, valid and reliable measures are required. There are a variety of methods used to measure lower limb loading. This thesis will rely on two established and reliable proxy measures of joint loading that have been linked to the risk of development and progression of joint degeneration and pain; the external knee adduction moment and the external knee flexor moment.

1.3.1 Knee Adduction Moment

The external knee adduction moment (KAM) is a valid and reliable proxy measure for medial knee loading during gait[43–48]. An increased KAM results in the knee going into varus, which causes the lateral joint compartment to open and the loading to be transferred to the medial joint compartment, increasing the load to approximately 60-70% of weight bearing[45,49,50]. Peak KAM is characterized by the highest peak in the KAM curve[12,51] and typically occurs during early stance[52], while impulse KAM is characterized by the space under the curve (Figure 5 – 13, Appendix A), and occurs during the entire stance phase[53,54].

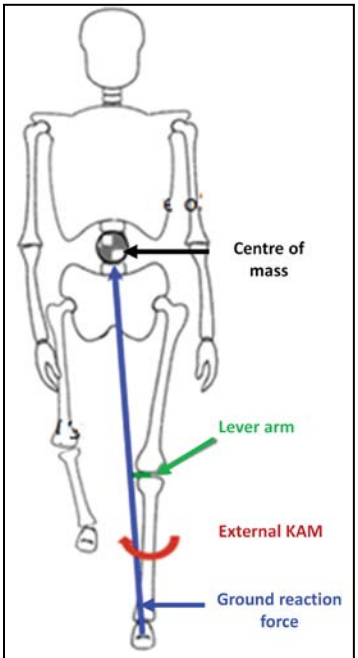

Figure 1: Visual description of KAM. *Adapted from Perry, 1992*[55]

KAM primarily composed of the GRF and the lever arm (Figure 1[55]). The lever arm is the distance between the GRF line and the joint (knee) centre[56]. Inertial factors also influence the magnitude of KAM[46]. Mean (standard deviation) values for KAM in healthy individuals has been reported as 2.24 (0.74) %Bw*Ht[57].

It has been found that individuals exhibiting increased peak KAM were more likely to develop medial knee joint compartment disease[42,58]. Not only does an increase in KAM cause excess loading on the medial compartment, leading to a loss of cartilage[52], it also increases the load on the subchondral bone, which can cause trauma, bone mineral loss and therefore increased pain[58–60]. Increased KAM values have been linked to an increased risk of knee pain severity and disease progression in OA[42,58]. Miyazaki et al[42] found that a 1%BW*Ht increase in KAM increased the risk of OA progression 6 fold and those who exhibited this increased KAM

reported more severe pain in the knee. Elevated KAM values have also been associated with the development of new chronic knee pain in older adults without knee pain at baseline gait testing[58].

1.3.2 Knee Flexion Moment

Knee flexion moment (KFM), can be used along with KAM to provide a more accurate depiction of knee loading[61,62]. KFM occurs along the sagittal plane[61]. KFM is influenced by the muscle contractions of the quadriceps around the knee[62,63]. Shull[63] et al. found that, individuals with increased KFM exhibited no change in loading of the medial compartment but did exhibit increased loading in the lateral compartment. The mean peak KFM for healthy individuals is ~0.97(0.78)%Bw*Ht[57]. Figure 2[64] shows the factors influencing KFM.

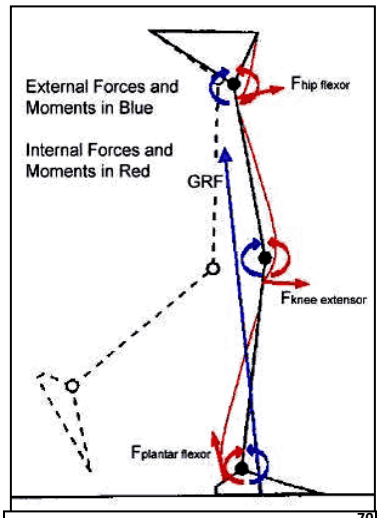

Figure 2 - Visual description of KFM[70]

1.3.4 Compensatory Strategies During Gait

Some compensatory strategies adopted by individuals during gait can reduce the magnitude of KAM, and thus potentially reduce knee loading. Two common compensatory strategies are toe-out (TO) gait and trunk lean (TL)[56,59,65]. Any investigation of KAM during gait should take these compensations into account. TO, caused from a greater foot angle, is characterized by external rotation of the foot[61,63,66]. TL is characterized by a lateral shift of the trunk over the stance limb[67]. Both, TO and TL, reduce KAM by reducing the lever arm (Figure 1), moving the line of action of the ground reaction force closer to the weight bearing knee[66,68]. Although it may reduce KAM, toe-out may increase KFM[61,62], and thus, the overall effect of the gait pattern may not result in decreased loading at the knee. In fact, the increase in KFM could

cause an increased loading of the patellofemoral joint[61]. A suggestion offered by Kemp et al.[47] to reduce knee loading for individuals with OA is to use a cane contralateral to the paretic side. This may not be appropriate for people with stroke, as we predict they have increased loading on their non-paretic side, depending on their functionality, they may not be able to use a cane on their paretic side.

In addition to gait compensations, gait velocity has an influence on the magnitude of KAM[69]. Approximately 9% of KAM variance can be attributed to self-selected walking speed[44], therefore a reduced walking speed may decrease KAM[44,57].

1.4 Summary

To summarize, walking recovery is the number one rehabilitation goal, stated by people post-stroke, although many of these people are left with residual gait deficits at discharge from rehabilitation. A common deficit is temporal gait asymmetry, which can affect the metabolic cost of gait, and may increase the likelihood of developing knee pain and/or degeneration, as gait asymmetry may be related to increased, repetitive loading of the non-paretic lower limb.

Despite evidence of the link between increased loading during gait and the development of MSK complications in the amputee and OA patient populations, little had been done to investigate such a relationship in the post-stroke population. It is important to determine if a relationship exists between temporal gait asymmetry and increased lower limb loading post-stroke. If such a relationship exists, then these individuals, like the amputee and OA population,

are at risk for joint pain and degeneration which should then become a clinical priority for rehabilitation.

1.5 Purpose & Objectives

The primary objectives of this study were to 1) characterize loading in the paretic and non-paretic limb during post-stroke gait using measures widely accepted as proxy measures of joint loading linked to the development and progression of MSK joint disease and 2) investigate the relationship between temporal gait asymmetry and loading in the non-paretic limb. A secondary objective was to characterize compensatory strategies that individuals with stroke may employ to counter these limb loading abnormalities

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2. Is lower limb loading in people post-stroke related to temporal gait asymmetry?

Abstract

Objectives: Gait asymmetry is a common issue post-stroke and could place these individuals at risk for developing joint pain and degeneration due to repetitive, excessive loading of the non-paretic lower limb. Therefore, the primary objective of this study was to characterize loading of the paretic and non-paretic lower limbs during post-stroke gait. A secondary objective was to investigate the relationship between knee loading and temporal gait symmetry. *Methods*: Gait analysis was performed with an 8-camera, 3-dimensional motion-capture system synchronized with a single floor-mounted force plate. Moments were determined throughout the stance phase of gait and expressed as external movements. Variables of interest were peak external knee adduction and flexor moments. Nine individuals with stroke were compared on these variables for both the paretic and non-paretic limbs to those of healthy adults using one-sample t-tests and then classified as having normal or excessive load at the knee. Individuals with stroke were also classified as exhibiting temporally asymmetric or symmetric gait with respect to a published normative cut-point. These groups were compared on proportions of individuals exhibiting excessive loading of the non-paretic limb using Fisher's exact tests. *Results*: Six participants exhibited increased loading of their non-paretic limb and four participants exhibited increased loading of their paretic limb as measured by peak KAM and peak KFM. There was no difference in the proportion of individuals with increased loading between the asymmetric and symmetric groups. *Conclusion*: People post-stroke exhibit increased loading of the lower limbs however this is not restricted to the non-paretic limb and it does not appear related to TGA. Future work should investigate lower limb loading longitudinally in a larger sample size with a wider range of gait function.

3. Introduction

Regaining walking function is the primary rehabilitation goal stated by people post-stroke[1]. One common post-stroke gait deficit is temporal gait asymmetry (TGA). Fifty-nine percent of individuals admitted to inpatient stroke rehabilitation exhibit this gait deficit. Despite gains made with rehabilitation in lower limb motor impairment and other gait domains (e.g. velocity), TGA changes very little over the same period[2,3,4]. Persisting TGA is of particular concern because there are a number of suspected long term consequences including increased metabolic cost, compromised balance and secondary musculoskeletal (MSK) changes including bone loss in the paretic leg and joint pain and injury in the non-paretic leg[2,5,6]. This paper focuses on the potential for MSK injury to the lower limbs post-stroke.

Little work has been done to directly investigate secondary MSK complications of TGA after stroke even though gait deviations are recognized as a potential risk factor for joint pain and degeneration[7,8]. TGA may lead to MSK injury to the non-paretic leg due to increased, repetitive loading sustained during daily activities, and in particular, during walking[7,9]. This loading pattern is generally accepted as a primary mechanism contributing to joint degeneration and pain in osteoarthritis[8]. Support for the proposed link between TGA and secondary MSK issues comes from a related patient population: those with lower limb amputations. Repetitive and excessive loading is also thought to contribute to lower body MSK issues in the amputee population[7]. Individuals with unilateral below or above knee amputation exhibit TGA similar to people with stroke (with the intact limb preferentially loaded during gait) and 71% of this group report pain in their intact limb and/or back[10]. Previous work by Kim and Eng[11] points to the potential for a link between TGA and abnormal lower limb loading after stroke. These authors reported an

association between TGA and asymmetry in ground reaction force (GRF) between the two limbs with greater force through the non-paretic limb in the majority of participants[11]. However, this asymmetrical limb loading was not investigated or discussed in the context of secondary MSK injury.

Two measures that are commonly used to characterize limb loading in the context of MSK joint and health issues are the external knee adduction moment (KAM) and the external knee flexion moment (KFM). The knee adduction moment is the product of the lever arm extending from the center of the knee joint and the line of action of the GRF and the frontal plane component of the GRF and is as an indirect measure of load at the medial compartment of the knee during walking[12]. Increased values of KAM have been linked to an increased risk of knee pain severity and disease progression in knee osteoarthritis (OA)[13,14]. It has also been associated with the development of new chronic knee pain in older adults without knee pain at baseline gait testing[14]. Two common kinematic compensations employed during gait have been shown to reduce the magnitude of KAM. A lateral shift of the trunk over the stance limb can shift the center of mass towards the stance limb and reduce the adduction lever arm magnitude[15]. An increase in the toe-out angle of the foot during gait can reduce KAM by both partially transforming a portion of the adduction moment into a flexion moment (in early stance) and by moving the line of action of the GRF closer to the knee, reducing the adduction lever arm (in late stance)[16]. However, when these compensatory methods are in place to reduce the adduction moment, it does not necessarily reduce knee loading as KFM may increase. KFM runs perpendicular to the adduction moment and is primarily caused by muscle contractions[17,18].

We propose the use of both these indirect measures of dynamic limb loading as a first step to linking post-stroke TGA to MSK joint degeneration and pain in the non-paretic lower

limb. Therefore, the primary objective of this study was to characterize loading of the non-paretic and paretic lower limbs and the compensatory strategies employed during gait in individuals with stroke. If TGA and secondary MSK issues are linked, then we would expect that individuals with post-stroke TGA are more likely to have increased knee adduction moment and/or knee flexor moment in the non-paretic limb. Therefore, a secondary objective of this study was to compare the proportion of individuals exhibiting increased knee adduction and knee flexion moments between groups of individuals post-stroke with and without TGA.

4. Methods

4.1 Participants

Ten participants were recruited from various sources, including an existing database of previous study participants, out-patient clinics at a rehabilitation hospital, a private physiotherapy clinic and in the community. Individuals were included if they could safely walk 5m without physical assistance from another person. Canes but not walkers were permitted during gait testing. Individuals were excluded if they had pre-existing lower limb conditions such as osteoarthritis or other neurological conditions that affect walking such as multiple sclerosis and Parkinson's disease. All participants provided written informed consent and the study was approved by the Western University Research Ethics Board.

4.2 Testing Protocol

All individuals underwent the following assessments in a single session located in a biomechanics laboratory.

a) Clinical Assessment

Two stroke-specific measures were used to characterize participants clinically. The National Institute of Health Stroke Scale (NIHSS) was used as a measure of stroke severity. The reliability and validity of this measure has been well established and greater values indicate greater severity of stroke-related impairments[19]. The Chedoke-McMaster Stroke Assessment (CMSA) was used to measure motor impairment for both the leg and foot. The leg and foot scales for the CMSA are each measured with a 7-point scale[20]. Smaller scores indicate greater motor impairment. The CMSA has good intrarater and interrater reliability and good concurrent validity with the Fugl-Meyer Assessment[21].

b) Gait Analysis

Gait analysis was performed with an 11-camera, 3-dimensional motion capture system (Motion Analysis Corporation, Santa Rosa, CA) synchronized with a single floor-mounted force plate (Advanced Mechanical Technology Inc., Watertown, MA). Twenty-two passive reflective markers configuration (12 mm diameter) were placed on the participants using a modified Helen Hayes marker [22]. Participants were asked to wear their everyday walking shoes, to allow an accurate representation of walking in daily life.

To calculate body mass and knee and ankle joint centers, participants stood on the force plate for an initial static trial. Three-dimensional coordinates for the knee joint centre of rotation (defined as the midpoint between the lateral and medial knee joint line markers) in the laboratory

frame of reference were determined from kinematic data collected during the static standing trial. Joint centers were calculated with the use of additional markers attached to the medial knee and ankle. The hip joint centre was determined by having the participant adduct, abduct, extend and flex at the hip[23] keeping the knee and ankle joints extended. After this was complete, participants walked across the laboratory at a self-selected pace while kinematic (60Hz) and kinetic (1200Hz) data were collected. Participants completed a minimum of 12 walking trials to allow for at least 6 clean force plate strikes for each lower limb.

c) Relationship between peak KAM, Toe out and Trunk Lean

Spearman correlations were used to investigate the association of peak KAM with toe out and trunk lean separately for the paretic and non-paretic sides.

4.3 Data Analysis

Commercial software (Orthotrak 6.0; Motion Analysis Corporation, Santa Rosa, CA) and custom programs were used to calculate moments about the knee during gait from the kinematic and kinetic data using inverse dynamics. Moments were expressed as external movements relative to the tibial anatomical frame of reference. Moments were determined throughout the stance phase of gait, averaged over six trials for each limb and normalized to body weight and height (%BW·Ht). Variables of interest were the external adduction moment and external flexor moment about the knee. The overall peak magnitudes of the knee adduction and flexor moments were identified using an algorithm for a moving window to examine moment values. Local peaks in the waveform were identified by the following criteria; immediately preceded by 5 lesser and

increasing values and immediately followed by 5 lesser and decreasing values. The overall peak was identified as the largest of these local peaks in the waveform.

Toe-out angle and trunk lean angle were also calculated. The toe-out angle was defined as the angle between a line from the centre of the ankle to the head of the second metatarsal and the forward progression of the body. The trunk lean angle was defined as the angle between the vertical and a line from the midpoint of the acromion processes to the midpoint of the anterior superior iliac spines. A trunk lean towards the stance limb was represented by positive angles and a trunk lean towards the swing limb was represented by negative angles. Both toe-out and trunk lean angles were quantified as the magnitude at the point of peak external knee adduction moment.

Events and phases of the gait cycle were identified from the kinematic data using successive foot contacts with the ground. Gait speed was calculated as the average of the left and right mean speed of successive foot contacts. Temporal symmetry ratio was calculated, as per recommendations, using the left and right values of swing time (averaged over 6 walking trials)[24]. The largest value was placed in the numerator so that all values were >1.0. A ratio value of 1.0 denotes perfect symmetry and a ratio >1.06 indicates asymmetric gait[24]. The direction of TGA was defined with respect to the limb with the greater duration of swing phase and noted as paretic or non-paretic.

4.4 Statistical Analysis

Statistical analyses were performed with Statistical Package for the Social Sciences (SPSS) (IBM, Armonk, NY) and SAS 9.3 software (SAS Institute Inc., Cary, NC). Means and standard

deviations (SD) were calculated for all variables for the group of stroke participants. Normative data for each of the variables of interest (knee adduction moment, knee flexor moment, toe-out angle, trunk lean angle) for healthy older adults were also extracted from a pre-existing database. The database included 109 healthy individuals and was produced in the same lab using the same testing procedures and data analysis techniques.

a) Comparison of lower limb loading in individual participants to normative values

One sample t-tests were used to compare each participant to the healthy group on knee adduction moment, knee flexor moment, toe-angle and trunk angle for both the paretic and non-paretic leg. Multiple comparisons performed for each participant were corrected with the Holm method[25]. The initial adjusted level of significance was set to P=0.006.

Participants were then assigned to one of the following two categories for each of these four variables of interest:

i. Abnormal load/angle: participants with a value that was significantly different and greater than the mean value for the healthy group.
ii. Normal load/angle: participants with a value that was not significantly different from the mean value for the health group or was significantly different and lesser than the mean value for the healthy group.

b) Comparison of lower limb loading in individuals with and without temporal gait asymmetry

Participants were classified as asymmetric (swing ratio >1.06) or symmetric (swing ratio ≤1.06). Then, Fisher's exact tests were used to compare the asymmetric and symmetric groups

on the proportion of individuals classified as abnormal vs. normal loads/angles for the knee adduction moment, knee flexor moment, toe-out angle and trunk lean angle.

c) Relationship between peak KAM, Toe-out and Trunk Lean

Spearman correlations were used to investigate the association of peak KAM with toe-out and trunk lean separately for the paretic and non-paretic sides.

5. Results

Participants

Nine individuals successfully completed the clinical assessment and gait analysis. One participant was unable to complete the gait analysis. Despite passing the initial screening for inclusion/exclusion criteria, on the day of testing, this participant decided he was unable to walk safely without his rollator. This participant was not included in the analysis. The mean age (SD) of the stroke group was 57.7 (9.8) years and the mean time since stroke was 37.2 (20.9) months. The healthy group had a mean age of 38.4 (13.5) which was significantly different from the stroke group ($p<0.01$). Clinically, the stroke group presented with a mean NIHSS of 2.4 (1.7), mean CMSA leg and foot scores of 5.7 (0.7) and 5.0 (1.5) respectively. The values for gait velocity, symmetry and daily walking activity for each participant are summarized in Table 1, along with demographic information and CMSA scores for the leg and foot. Five stroke participants exhibited temporal gait asymmetry (swing ratio >1.06).

Table 1 Gait velocity, symmetry and daily walking activity levels for stroke participants. Individuals with temporal gait asymmetry (i.e. swing time symmetry ratio >1.06) are marked with (*). The direction of asymmetry was determined by the limb with greater value for swing time/step length and is indicated by (np) for the non-paretic limb and (p) for the paretic limb

Participant	**Age** (years)	**Gender**	**Paretic side**	**Months post-stroke**	**CMSA leg/foot** (0-7)	**Velocity (m/s)**	**Swing time symmetry** (ratio)
P01	57	M	Right	9	6/7	1.12	1.01
P02	56	M	Right	48	6/5	0.96	1.04
P03	64	M	Left	50	6/5	1.17	1.03
P04	61	M	Left	45	5/5	1.23	1.07*p
P05	34	F	Right	47	7/6	1.63	1.04
P06	58	F	Left	55	5/3	0.75	1.50*p
P07	57	M	Left	11	5/5	0.94	1.24*p
P08	69	F	Left	60	6/7	1.04	1.07*p
P09	63	M	Left	10	5/3	0.69	1.20*p

a) Comparison of lower limb loading in individual participants to normative values

Peak knee adduction and flexor moments and toe-out and trunk lean angles are summarized for the paretic and non-paretic lower limbs for each participant in Tables 2 and 3 respectively. Values that were significantly different from the healthy group (based on one-sample t-test) are denoted by (+) if the value is greater and (-) if the value is less than the corresponding mean value for the group of healthy adults. Based on peak knee adduction moment and/or flexor moment values, 6 of the 9 participants (P02, P05, P06, P07, P08, P09) exhibited increased loading of the non-paretic limb and 4 participants exhibited increased loading of the paretic limb (P03, P04, P06, P07).

Knee adduction and knee flexion moments throughout the gait cycle for each participant are included in Figures 5 - 13 in Appendix A. Also summarized in the Appendix are the results of the one-sample t-tests for each variable of interest for each participant.

Table 2 Measures of non-paretic limb loading and compensatory strategies for stroke participants. Values that were significantly different from the healthy group (based on one-sample t-test) are denoted by (+) if the value is greater and (-) if the value is less than the healthy means.

Participant	**Peak knee adduction moment** (%BW*ht)	**Peak knee flexor moment** (%BW*ht)	**Toe-Out** (degrees)	**Trunk Lean** (degrees)
P01	1.80(-)	0.35(-)	16.61(+)	2.14(+)
P02	2.48	1.98(+)	22.11(+)	2.67(+)
P03	2.44	-0.31(-)	8.72(-)	-0.20(-)
P04	2.51	0.61(-)	11.61	2.30(+)
P05	4.20(+)	3.27(+)	12.15	1.50
P06	1.56(-)	1.42(+)	5.01(-)	0.50(-)
P07	4.14(+)	0.73(-)	11.75	1.97(+)
P08	1.66(-)	2.44(+)	-0.82(-)	1.75(+)
P09	2.93(+)	0.88	18.4(+)	-0.43(-)

Table 3 Measures of paretic limb loading and compensatory strategies for stroke participants. Values that were significantly different (range $p<0.001$) from the healthy group (based on one-sample t-test) are denoted by (+) if the value is greater and (-) if the value is less than the healthy means.

Participant	**Peak knee adduction moment** (%BW*ht)	**Peak knee flexor moment** (%BW*ht)	**Toe-Out** (degrees)	**Trunk Lean** (degrees)
P01	1.72(-)	-0.17(-)	11.99(+)	1.19
P02	2.24(-)	0.71	15.94(-)	0.15(-)
P03	3.35(+)	3.19(+)	19.82(+)	2.43(+)
P04	2.04(-)	0.99(+)	11.94(+)	0.90(-)
P05	2.01(-)	0.50	8.53(-)	2.20(+)
P06	1.85(-)	2.67(+)	5.42(-)	5.10(+)
P07	2.91(+)	1.80(+)	27.54(+)	1.50
P08	1.35(-)	0.14(-)	7.66(-)	0.72(-)
P09	2.59	0.07(-)	7.13(-)	2.01(+)

b) Comparison of lower limb loading in individuals with and without temporal gait asymmetry

There were no significant differences between the symmetric and asymmetric groups in the proportion of individuals exhibiting increased peak knee adduction moment or peak knee flexion moment in the non-paretic limb.

c) Relationship between peak KAM, toe-out and trunk lean

A Spearman correlation coefficient revealed that there was no significant relationship between peak KAM and toe-out for the paretic limb (p=0.15) or for the non-paretic limb (p=0.11). Similarly, there was no significant relationship between peak KAM and trunk lean for the paretic limb (p=0.97) or the non-paretic limb (0.52). These relationships are illustrated by scatterplots in Figures 3 and 4.

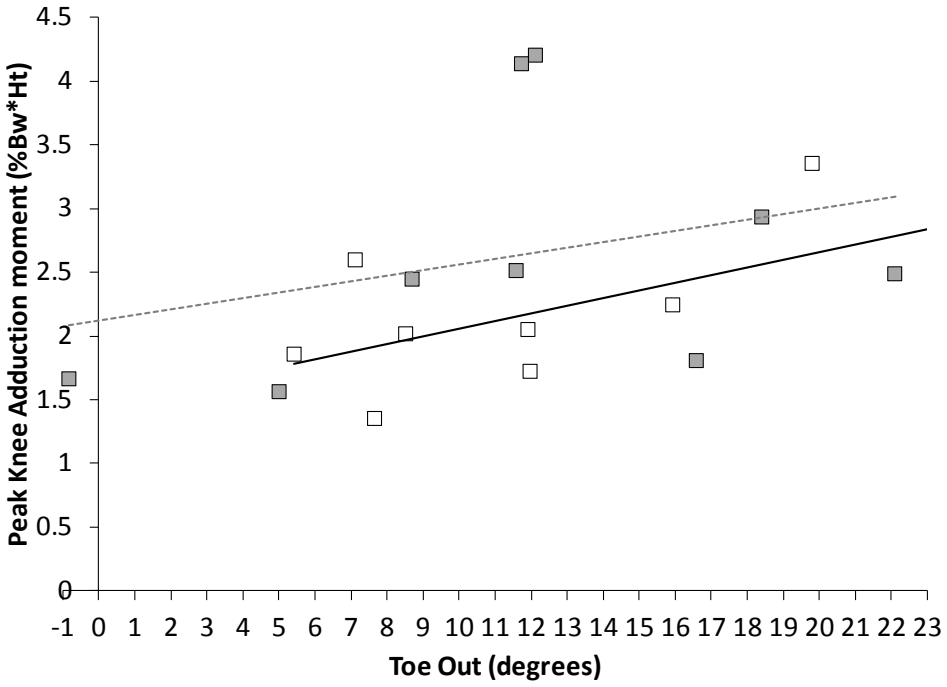

Figure 3 – Relationship of peak KAM and Toe-Out
Scatterplot of peak KAM and toe-out values in the paretic (shaded squares) and non-paretic (open squares) lower limbs for individuals with stroke (n=9). The dotted and solid lines represent the trend lines for the relationships in the paretic and non-paretic limbs respectively.

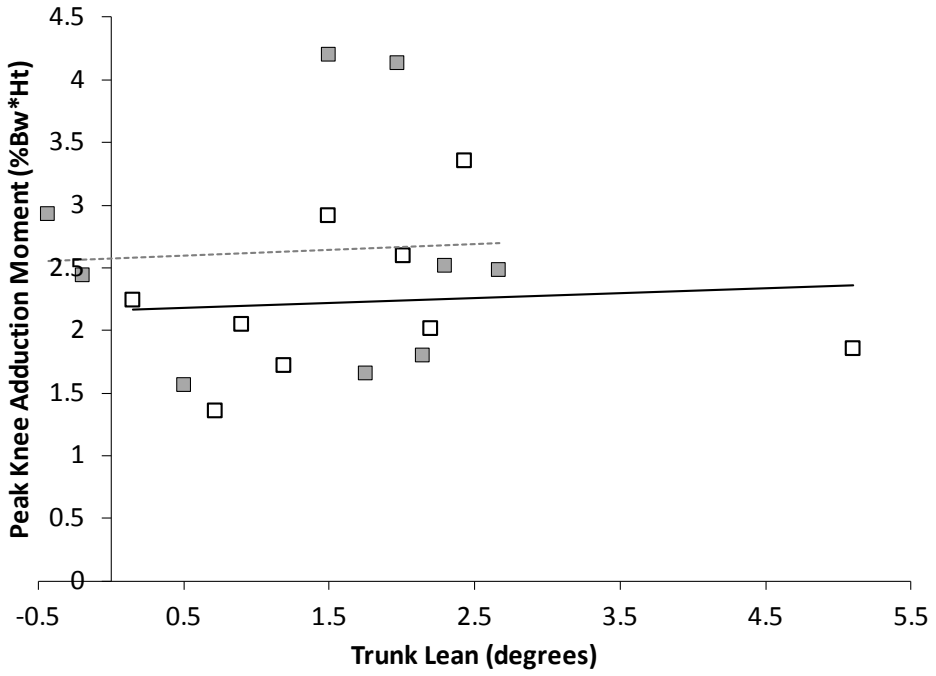

Figure 4 – Relationship of peak KAM and Trunk Lean
Scatterplot of peak KAM and trunk lean values in the paretic (shaded squares) and non-paretic (open squares) lower limbs for individuals with stroke (n=9). The dotted and solid lines represent the trend lines for the relationships in the paretic and non-paretic limbs respectively.

6. Discussion

The main finding of this study is that people post-stroke do exhibit increased loading of their knee joints compared to healthy adults. To the best of our knowledge, this is the first study to examine loading patterns in individuals with stroke in the context of risk for secondary MSK issues. Findings related to our secondary objective are not consistent with our original hypothesis; that people with post-stroke TGA exhibit excess loading on their non-paretic side. Rather, it appears that excess loading can occur in either or both of the paretic and non-paretic lower limbs and this excessive lower limb loading is not associated with TGA. This was not consistent with previous work done on loading patterns in people with lower limb amputations[7,9].

Residual impairments are variable across people post-stroke, causing these individuals to adopt different strategies to achieve steady state walking[26,27]. Similarly, we observed multiple kinematic loading patterns within our group of nine individuals post-stroke. Two participants demonstrated increased loading on only their paretic side (P03; P04), five participants demonstrated increased loading on only their non-paretic side (P02; P05; P06; P08; P09), and one participant exhibited increased loading on both their paretic and non-paretic side (P07). Compared to the healthy group, six participants displayed larger toe-out and trunk lean values on their non-paretic side and seven on their paretic side. But since there was no significant correlation between peak KAM and these values, we cannot say whether they serve to reduce peak KAM in this stroke population.

This variation in excessive loading patterns between the paretic and non-paretic leg in individuals with stroke may be related to their residual impairments in strength. Deactivation or weakness of the hip abductors on the paretic side is common post-stroke[27,28]; this can cause pelvic drop[27,29]. Pelvic drop is characterized by a shift in the centre of mass towards the swing limb during the stance phase of the paretic leg (the leg with the weak hip abductors) which would increase the adduction moment in the frontal plane [27,29]. Pelvic drop, as a result, causes excess loading about the paretic knee[29,30] and could explain why some of our participants exhibited excess loading on their paretic side, and not their non-paretic side as we had predicted.

Another possible reason for increased loading on both the paretic and non-paretic side may be co-contraction of muscles around the knee. Co-contraction at the knee is exhibited by individuals with OA and is associated with further increases in knee loading, which can be exhibited through KAM and KFM[31]. Co-contraction about the paretic and non-paretic knee is

also observed post-stroke[32,33] and may explain why increased values of peak knee flexor moment were observed in both limbs and were unrelated to TGA.

Our preliminary findings have potential clinical implications related to the approach to gait rehabilitation post-stroke and thus warrant further investigation. One suggested strategy for reducing lower limb loading in the OA population is to decrease walking velocity[34]. Since people post-stroke have significantly reduced gait velocity[2], rather than adopting this strategy, it may be beneficial to attempt to restore normal kinematic and kinetic patterns during gait rehabilitation. In the same vein, a common stroke rehabilitation goal is to increase gait velocity and it has been proposed that training at faster gait speeds is better than training at their preferred speeds[35]. However, in light of the fact that increased velocity may increase the magnitude of loading[34], faster gait speeds may not be the best approach for individuals already exhibiting a kinematic/kinetic gait pattern linked with excessive joint loading.

One limitation to this study was that the participants were high functioning; they had mild to moderate motor impairment (measured by CMSA) and relatively fast gait velocities. Therefore, these findings may not be applicable to individuals with greater motor impairment or slower gait speeds.

Now that excessive loading patterns have been identified in the mild to moderately paretic stroke population, future work should look to investigate joint loading in the more severely paretic stroke population. In addition, future work should investigate other factors that could contribute to excessive lower limb loading (e.g. hip abductor weakness, EMG measures of co-contraction) in a larger sample size with a wider range of walking function. A longitudinal

study may be useful to investigate the development of secondary MSK issues and its relationship to gait deviations post-stroke.

7.0 Study Bibliography

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8. Conclusions

The primary objectives of this thesis were to 1) characterize loading in the paretic and non-paretic limb during post-stroke gait and 2) investigate the relationship between temporal gait asymmetry and loading in the non-paretic limb. A secondary objective was to characterize compensatory strategies that individuals with stroke may employ to counter these limb loading abnormalities. To the best of my knowledge, this is the first study to examine loading patterns in individuals with stroke in the context of risk for secondary MSK issues. The main finding of this thesis is, that compared to a group of healthy adults, some people post-stroke do exhibit abnormal excessive loading patterns (as measured by peak KAM and peak KFM) in either or both of the paretic and non-paretic lower limbs. For this reason, they may be at risk for developing joint pain and/or degeneration[1]. However, increased loading in the non-paretic lower limb does not appear to be related to temporal gait asymmetry and common gait compensations such as toe-out and trunk lean and are not associated with peak KAM values in individuals with stroke.

8.1 Variability in the Pattern of Lower Limb Loading Across Individuals Post-stroke

There was considerable variability in the pattern of excessive loading across lower limbs and across the participants in the study. Two participants demonstrated increased loading on only their paretic side (P03; P04). Five participants demonstrated increased loading on only their non-paretic side (P02; P05; P06; P08; P09). Another participant's results indicated increased loading on both their paretic and non-paretic side (P07). While increased loading of the non-paretic limb was expected, increased loading of both limbs or of the paretic limb alone was not. However, variability of observed patterns within a group of individuals with stroke has been

reported by other researchers investigating different aspects of post-stroke gait such as gait asymmetry[2,3], lower limb muscle activity[4] and kinematic and kinetic features[5]. Residual stroke-related impairments (e.g. motor and sensory impairments, spasticity) between people post-stroke are also quite variable, causing these individuals to adopt different strategies to achieve steady state walking[6,7]. Kim & Eng[6] found, some people post-stroke will develop different kinetic strategies to achieve a particular movement; in this case, the movement was walking.

8.2 Increased Loading in the Paretic Limb

An unexpected finding of this study was increased loading in the paretic limb. This occurred in two individuals. One of these individuals (P03) exhibited symmetrical gait. The other individual (P04) exhibited temporal gait asymmetry in the paretic direction which means that they were spending longer in stance phase on the non-paretic side. There are two possible explanations for this increased loading of the paretic limb, despite either gait symmetry or decreased time spent in single limb stance; pelvic drop and co-contraction.

Decreased activation of the paretic hip abductors has been reported in people with stroke[8]. Weakness of the hip abductors has been shown to increase ipsilateral KAM[9] in individuals with OA. During the stance phase of gait, weak hip abductors of the stance limb allow the pelvis to drop excessively in the contralateral swing limb. This causes a shift in the body's center of mass toward the swing limb which in turn increases forces across the medial knee joint compartment in the stance limb[9,10]. Pelvic drop is also exhibited by people post-stroke[5]. Pelvic drop in the stroke population could be caused by hip abductor weakness or deceased activation of the paretic hip[8,9]. This could have contributed to the excessive loading on

the paretic limb observed in the two participants in this study. However, this cannot be confirmed because muscle strength and EMG were not measured in this study.

Co-contraction of muscles about the knee joint may be another explanation for increased loading of the paretic limb. Agonist and antagonist muscles usually work together, where the agonist is responsible for most movement and the antagonist is responsible, mainly for stabilization around the joint[11]. However, in people with OA, it has been found that, at times, there will be co-contraction of the agonist and antagonist, further increasing knee loading[12]. Newham and Hsiao[13] found that in some people post-stroke, there is also co-contraction about the knee, where the hamstrings and the quadriceps will contract simultaneously in the paretic lower limb. This co-contraction may have existed in P03 and P04 and the increased peak KFM (known to be related to muscle activity)[14] seems to support this theory. However, this would need to be confirmed with EMG measurement.

8.3 Potential Consequences of Increased Loading of the Lower Limbs Post-stroke

Regardless of whether the paretic, non-paretic or both limbs exhibit increased loading, the very presence of increased loading in the lower limbs during post-stroke gait is cause for concern because increased load on the knee has also been linked to an increased risk of developing knee osteoarthritis (OA) and knee pain/degeneration[15]. Knee OA is of particular concern for individuals post-stroke because OA limits mobility[16], and their mobility is an existing challenge due to stroke itself[17]. If people exhibiting increased limb loading post-stroke do develop secondary joint degeneration and pain, they could eventually further reduce their ambulatory activity due to the discomfort associated with these secondary MSK consequences. This inactivity increases the chance of stroke reoccurrence, other cardiovascular disease,

metabolic dysfunction, and other co-morbidities[18,19]. For this reason, rehabilitation post-stroke should address the potential risk factors for increased knee loading, and develop strategies to compensate for excess knee loading.

8.4 Strategies to Manage Increased Loading in the Limbs Post-stroke

The results of the current study suggest the need for adjustments to the management of post-stroke gait. In light of the increased lower limb loading, it may prove necessary to decrease the risk of joint pain/degeneration and prevent further mobility issues. This may require long-term monitoring of people post-stroke and of the possible development MSK issues.

Long-term follow up of stroke survivors is a recommendation that has been made previously. It has been recommended that people post-stroke, after discharge receive an initial follow-up after 6 months and annually thereafter[20]. These long term follow-ups may include educating the person about complications that could arise post-stroke, provide support to allow for better community integration, and follow-up on existing conditions[20]. In light of the current findings, these follow-ups should also include an MSK examination, addressing old and new MSK complaints, and gait analysis to look for changes in gait pattern and changes or increases in lower limb loading.

Therapists should be aware of this excess loading of the knee joint, as it could lead to MSK complaints, that may need to be addressed.. However, further investigation is required to determine what the appropriate interventions strategies may be. One proposed strategy to reduce knee loading is to use a cane on the contralateral side[21]. This strategy may not be appropriate for all people post-stroke, depending on their upper limb function. Reducing walking speed is suggested to reduce knee loading in some individuals, although the affect varies depending on

disease severity[22]. This may not be appropriate for people post-stroke, as they already have slow gait speeds. Other strategies to reduce KAM, suggested by other investigators, is to encourage trunk lean and toe-out during gait[23-25], although this does not necessarily reduce knee loading, as with some people KFM is increased[26,27]. Two other lines of inquiry for potential interventions to reduce knee loading post-stroke are related to pelvic drop and co-contraction of muscles around the knee joint during gait. If future work finds these factors to be related to increased loading at the knee, then they may be appropriate intervention targets during stroke rehabilitation.

8.5 Study Limitations

One limitation to this study was the use of proxy measures to measure knee loading. However, direct measures of joint loading are not feasible, because they are too invasive[28]. We used measures along two planes to characterize loading at the knee joint and these measures have been found to be valid and reliable[21,28-32]. A second limitation is the small sample size. The inclusion criteria were restricted to those individuals that could walk without a gait aid, or at most a single point cane, in order for the gait analysis to be conducted. This limited the number of individuals eligible or the study and complicated recruitment. A third limitation is that our participants were highly functioning, which is also related to the inclusion criteria. This limits the generalizability of our findings to individuals more severely affected by stroke. Future studies should examine a larger group of individuals with a wider range of gait and lower limb function.

8.6 Future Directions

Now that it has been established that increased loading of the lower limbs can occur during gait post-stroke, future work should confirm these findings in a larger sample size with a

wider range of walking function. Future work should investigate factors other than temporal gait asymmetry that may be related to excessive loading in the lower limbs such as, hip abductor weakness and EMG patterns of co-contraction. It would also be of benefit to conduct a longitudinal study with the post-stroke population. This would confirm if secondary musculoskeletal complications develop after discharge from rehabilitation and whether they are related to excessive loading of the legs. Finally, future work should also investigate whether gait training and rehabilitation can reduce excessive loading.

8.7 Discussion Bibliography

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Appendices

Appendix 1 - Knee adduction and knee flexion moments throughout the gait cycle

P01

On the paretic side, Participant 1 demonstrated significantly, Toe-Out (TO) ($p<0.001$) and Trunk Lean (TL) ($p<0.001$) during gait, compared to a healthy population. Peak Knee Adduction Moment (PKAM) ($p<0.001$), Knee Flexion Moment (KFM) ($p=0.003$),

On the non-paretic side, participant 1 demonstrated significantly higher and TO ($p=0.001$), and significantly lower PKAM ($p<0.001$), compared to the healthy group.

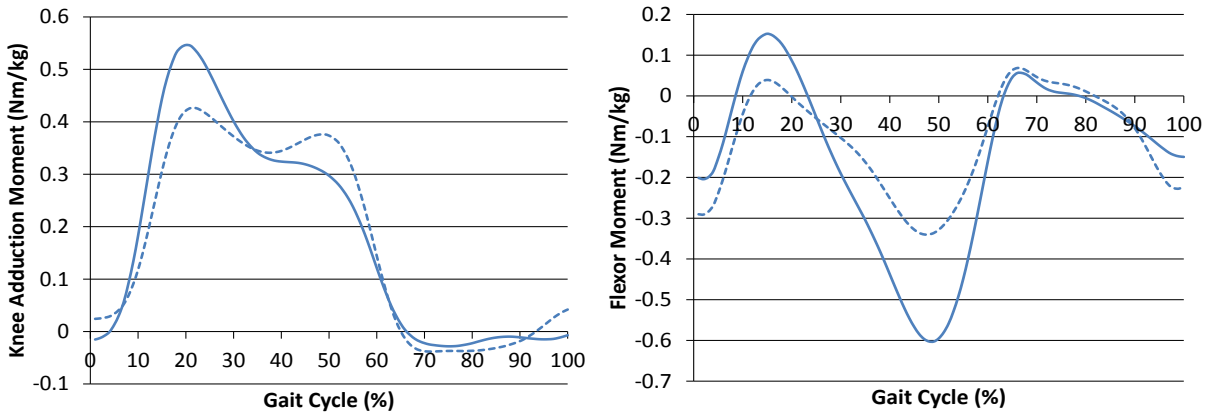

Figure 5A/B: *Participant 1 – KAM (Figure 3A) & KFM (Figure 3B)*
Solid line – Non-paretic leg, Dotted line – Paretic leg

P02

On the paretic side, participant 2 demonstrated significantly higher KFM ($p<0.001$), TO ($p<0.001$), and TL ($p<0.001$).

On the non-paretic side, participant 2 demonstrated significantly higher, and significantly lower TO ($p<0.001$); TL ($p<0.001$);

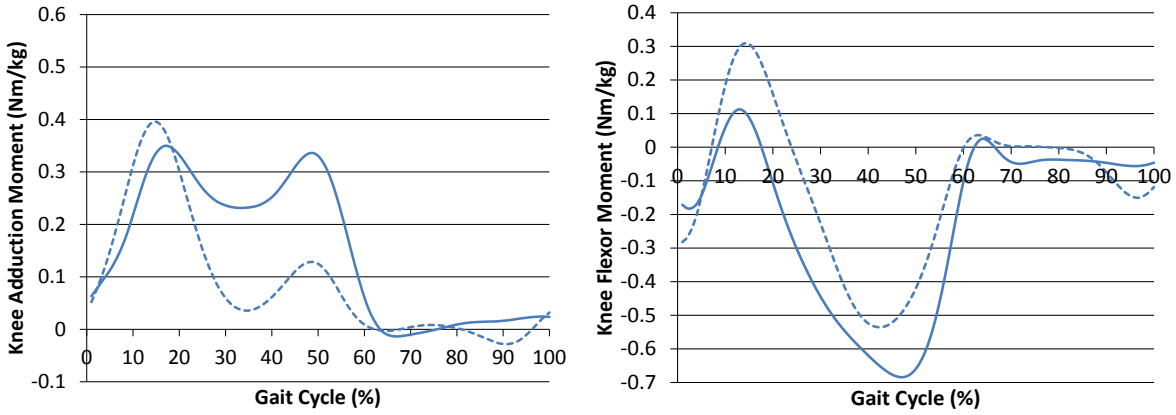

Figure 6A/B: *Participant 2 – KAM (Figure 4A) & KFM (Figure 4B)*
Solid line – Non-paretic leg, Dotted line – Paretic leg

P03

On the paretic side, participant 3 demonstrated significantly lower KFM ($p<0.001$); TO ($p<0.001$); TL ($p<0.001$), compared to a healthy population.

On the non-paretic side, participant 3 demonstrated significantly higher PKAM ($p<0.001$); KFM ($p<0.001$); TO ($p<0.001$); TL ($p<0.001$), compared to a healthy population.

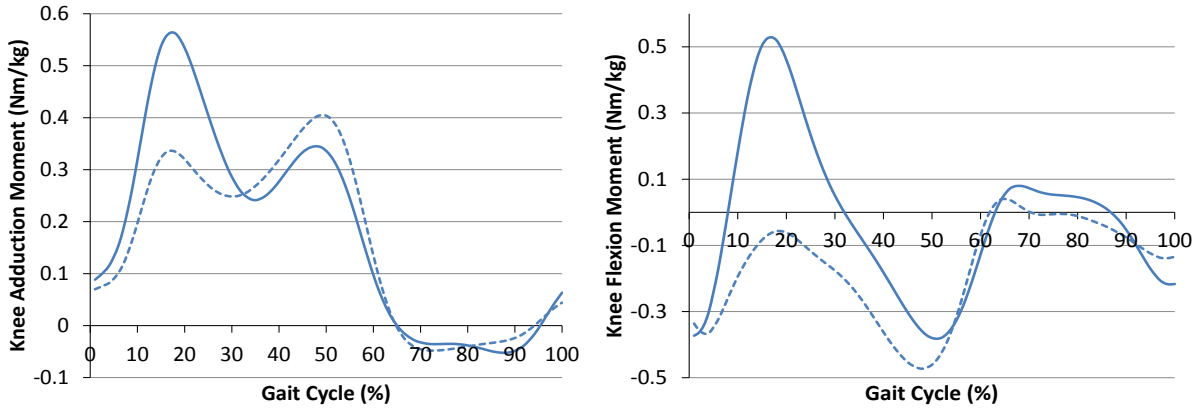

Figure 7A/B: *Participant 3 – KAM (Figure 5A) & KFM (Figure 5B)*
Solid line – Non-paretic leg, Dotted line – Paretic leg

P04

On the paretic side, participant 4 demonstrated significantly higher TL ($p<0.001$), compared to a healthy population. KFM ($p<0.001$); was significantly lower than a healthy population.

On the non-paretic side, participant 4 demonstrated significantly higher KFM ($p=0.003$); and TO ($p=0.001$), and significantly lower PKAM ($p<0.001$); TL ($p=0.003$), compared to a healthy population.

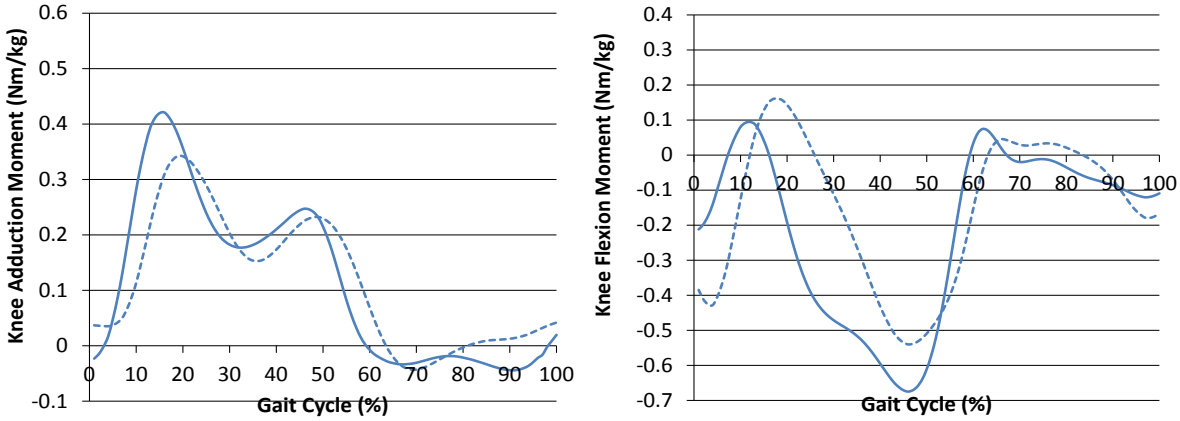

Figure 8A/B: *Participant 4 – KAM (Figure 6A) & KFM (Figure 6B)*
Solid line – Non-paretic leg, Dotted line – Paretic leg

P05

On the paretic side, participant 5 demonstrated significantly higher PKAM ($p<0.001$); KFM ($p<0.001$), compared to a healthy population.

On the non-paretic side, participant 5 demonstrated significantly higher TL ($p<0.001$), and significantly lower PKAM ($p<0.001$); and TO ($p=0.002$), compared to a healthy population.

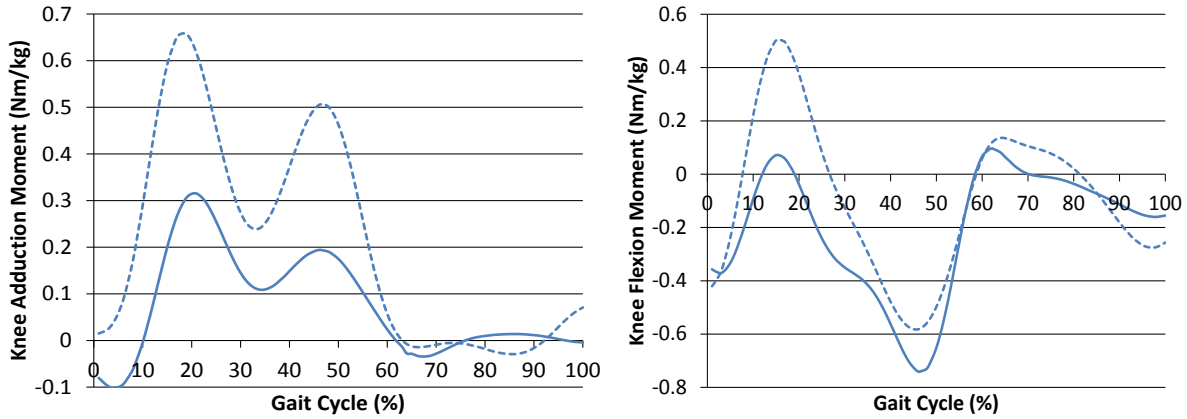

Figure 9A/B: *Participant 5 – KAM (Figure 7A) & KFM (Figure 7B)*
Solid line – Non-paretic leg, Dotted line – Paretic leg

P06

On the paretic side, participant 6 demonstrated significantly higher KFM ($p<0.001$), compared to a healthy population. PKAM ($p<0.001$); TO ($p<0.001$); TL ($p<0.001$), were significantly lower than a healthy population.

On the non-paretic side, participant 6 demonstrated significantly higher; KFM ($p<0.001$); and TL ($p<0.001$), and significantly lower PKAM ($p<0.001$); TO ($p<0.001$), compared to a healthy population.

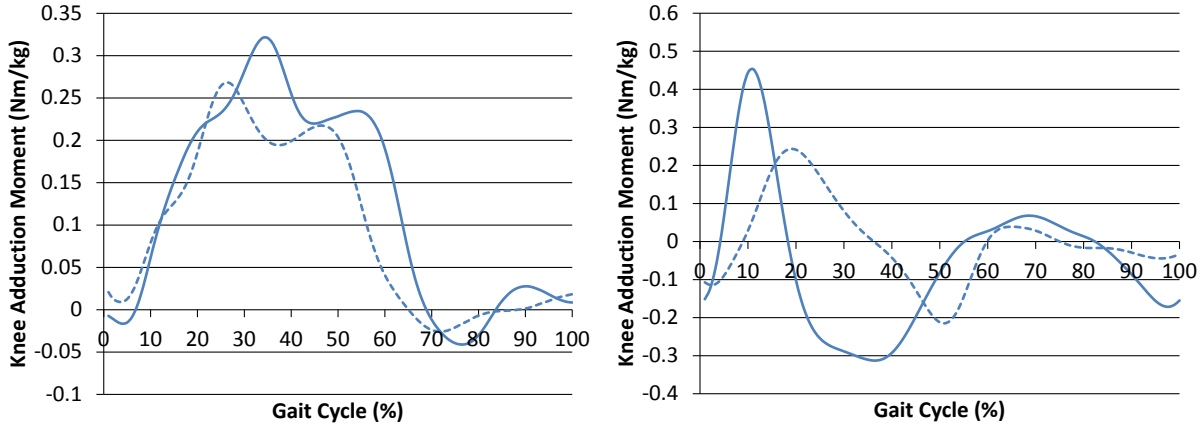

Figure 10A/B: *Participant 6 – KAM (Figure 8A) & KFM (Figure 8B)*
Solid line – Non-paretic leg, Dotted line – Paretic leg

P07

On the paretic side, participant 7 demonstrated significantly higher PKAM ($p<0.001$); and TL ($p<0.001$) compared to a healthy population. KFM ($p=0.004$); was significantly lower than a healthy population.

On the non-paretic side, participant 7 demonstrated significantly higher PKAM ($p<0.001$); KFM ($p<0.001$); and TO ($p<0.001$), compared to a healthy population.

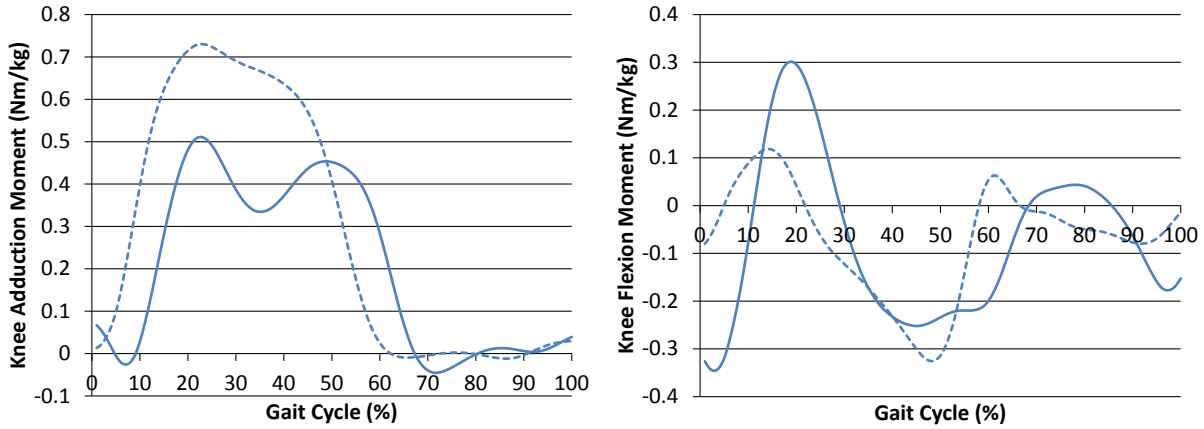

Figure 11A/B: *Participant 7 – KAM (Figure 9A) & KFM (Figure 9B)*
Solid line – Non-paretic leg, Dotted line – Paretic leg

P08

On the paretic side, participant 8 demonstrated significantly higher KFM ($p<0.001$); and TL ($p<0.001$), compared to a healthy population. PKAM ($p<0.001$); and TO ($p<0.001$), were significantly lower than a healthy population.

On the non-paretic side, participant 8 demonstrated significantly lower PKAM ($p<0.001$); KFM ($p<0.001$); TO ($p<0.001$); and TL ($p<0.001$), compared to a healthy population.

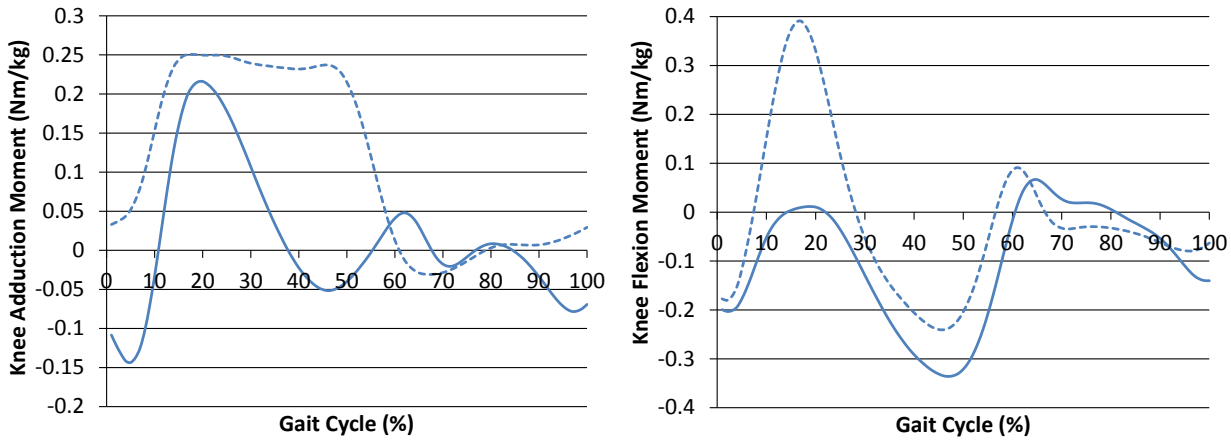

Figure 12A/B: *Participant 8 – KAM (Figure 10A) & KFM (Figure 10B)*
Solid line – Non-paretic leg, Dotted line – Paretic leg

P09

On the paretic side, participant 9 demonstrated significantly higher PKAM ($p<0.001$); and TO ($p<0.001$), compared to a healthy population. TL ($p<0.001$) was significantly lower than a healthy population.

On the non-paretic side, participant 9 demonstrated significantly higher PKAM ($p=0.027$); and TL ($p<0.001$), and significantly lower KFM ($p<0.001$); and TO ($p<0.001$), compared to a healthy population.

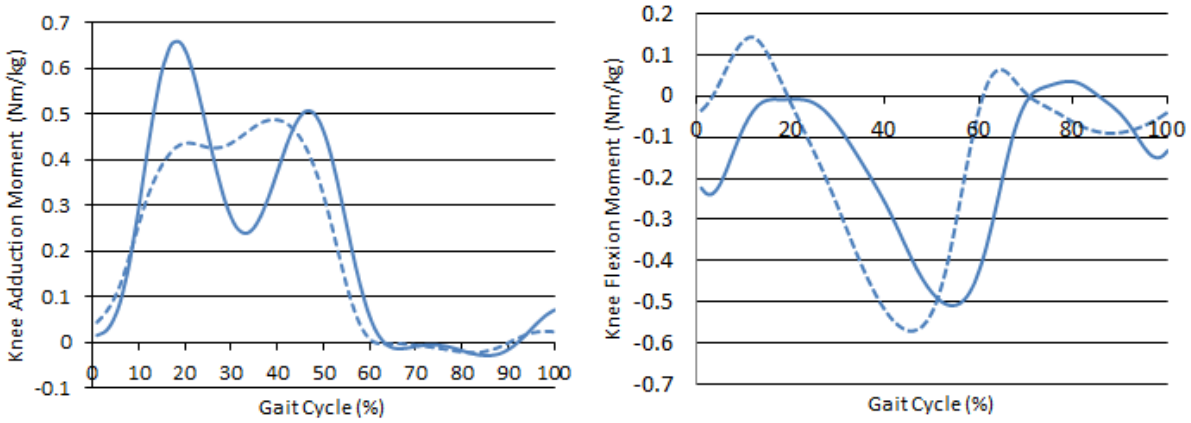

Figure 13A/B: *Participant 9 – KAM (Figure 11A) & KFM (Figure 11B)*
Solid line – Non-paretic leg, Dotted line – Paretic leg

Appendix 2 - Ethics Certificate

Western Research

Research Ethics

Use of Human Participants - Ethics Approval Notice

Principal Investigator: Dr. Kara Patterson
File Number:100878
Review Level: Delegated
Approved Local Adult Participants:20
Approved Local Minor Participants:0
Protocol Title: Establishing a link between gait asymmetry and increased loading of the non-paretic limb in individuals post-stroke - 18231E
Department & Institution: Health Sciences\Physical Therapy,Western University
Sponsor: Physiotherapy Foundation of Canada

Ethics Approval Date: June 28, 2013 **Expiry Date:**June 30, 2014
Documents Reviewed & Approved & Documents Received for Information:

Document Name	Comments	Version Date
Revised Study End Date	The study end date has been extended to June 30, 2014 to allow for project continuation.	

This is to notify you that The University of Western Ontario Research Ethics Board for Health Sciences Research Involving Human Subjects (HSREB) which is organized and operates according to the Tri-Council Policy Statement: Ethical Conduct of Research Involving Humans and the Health Canada/ICH Good Clinical Practice Practices: Consolidated Guidelines; and the applicable laws and regulations of Ontario has reviewed and granted approval to the above referenced revision(s) or amendment(s) on the approval date noted above. The membership of this REB also complies with the membership requirements for REB's as defined in Division 5 of the Food and Drug Regulations.

The ethics approval for this study shall remain valid until the expiry date noted above assuming timely and acceptable responses to the HSREB's periodic requests for surveillance and monitoring information. If you require an updated approval notice prior to that time you must request it using the University of Western Ontario Updated Approval Request Form.

Members of the HSREB who are named as investigators in research studies, or declare a conflict of interest, do not participate in discussion related to, nor vote on, such studies when they are presented to the HSREB.

The Chair of the HSREB is Dr. Joseph Gilbert. The HSREB is registered with the U.S. Department of Health & Human Services under the IRB registration number IRB 00000940.

[redacted]

Signature

Ethics Officer to Contact for Further Information

Erika Basile	Grace Kelly	Vikki Tran	Shantel Walcott

This is an official document. Please retain the original in your files.

Western University, Research,

Western Research

Research Ethics

Western University Health Science Research Ethics Board
HSREB Amendment Approval Notice

Principal Investigator: Dr. Kara Patterson
Department & Institution: Health Sciences\Physical Therapy,Western University

HSREB File Number: 100878
Study Title: Establishing a link between gait asymmetry and increased loading of the non-paretic limb in individuals post-stroke -18231E
Sponsor: Physiotherapy Foundation of Canada

HSREB Amendment Approval Date: September 17, 2014
HSREB Expiry Date: June 30, 2015

Documents Approved and/or Received for Information:

Document Name	Comments	Version Date
Revised Study End Date		

The Western University Health Science Research Ethics Board (HSREB) has reviewed and approved the amendment to the above named study, as of the HSREB Amendment Approval Date noted above.

HSREB approval for this study remains valid until the HSREB Expiry Date noted above, conditional to timely submission and acceptance of HSREB Continuing Ethics Review. If an Updated Approval Notice is required prior to the HSREB Expiry Date, the Principal Investigator is responsible for completing and submitting an HSREB Updated Approval Form in a timely fashion.

The Western University HSREB operates in compliance with the Tri-Council Policy Statement Ethical Conduct for Research Involving Humans (TCPS2), the International Conference on Harmonization of Technical Requirements for Registration of Pharmaceuticals for Human Use Guideline for Good Clinical Practice Practices (ICH E6 R1), the Ontario Personal Health Information Protection Act (PHIPA, 2004), Part 4 of the Natural Health Product Regulations, Health Canada Medical Device Regulations and Part C, Division 5, of the Food and Drug Regulations of Health Canada.

Members of the HSREB who are named as Investigators in research studies do not participate in discussions related to, nor vote on such studies when they are presented to the REB.

The HSREB is registered with the U.S. Department of Health & Human Services under the IRB registration number IRB 00000940.

[redacted]
Ethics Officer, on behalf of Dr. Joseph Gilbert, HSREB Chair

Ethics Officer to Contact for Further Information

Erika Basile	Grace Kelly	Mina Mekhail	Vikki Tran

This is an official document. Please retain the original in your files.

CV

Stephanie Marrocco

EDUCATION

Sept 2012 – March 2015	Masters of Science – Health and Rehabilitation Sciences Awarded Western Graduate Research Scholarship University of Western Ontario London, ON
Sept 2008 – April 2012	Honours Specialization in Kinesiology Minor in French University of Western Ontario London, ON

RESEARCH EXPERIENCE

Sept 2012 – April 2015	Graduate Student Health and Rehabilitation Sciences, University of Western Ontario *Duties:* Data collection and analysis; recruiting participants; providing instruction to participants; assess participant functionality; drafting ethics protocols; development of questionnaires
Sept 2011 – March 2012	Research Assistant Supervisor: Dr. Lynda Fitzgeorge School of Health Studies, University of Western Ontario *Duties:* Providing direction to participants; data entry; data collection

RESEARCH AREAS AND COMPETENCIES

- Rehabilitation
- Functionality Assessments (National Institute of Health Stroke Scale, Chedoke-McMaster Stroke Scale, Berg Balance Scale, Community Balance and Mobility Scale)
- Community Ambulation
- Measurement of Gait Post-Stroke
- Questionnaire Development – Community Ambulation in People Post-Stroke
- Experience conducting large scale systematic reviews
- Experience using Actigraph accelerometers, Gaitrite and Zeno mats
- Experience with PKMAS software, SSPS Statistical Software, Microsoft Excel

RESEARCH IN PROGRESS

- The development and evaluation of a new questionnaire to measure community ambulation post-stroke
- Characterizing Knee Loading Patterns in People Post-Stroke

PRESENTATIONS

October 2014 American Congress of Rehabilitation Medicine Conference, Toronto, ON
Development and Evaluation of Community Ambulation Questionnaire
Marrocco S, Crosby LD, Mansfield A, Chesworth B, Birmingham T, Patterson KK

February 2013 Health and Rehabilitation Sciences – Graduate Research Forum 2013
Effect of Inclines on Gait of Recovering Stroke Patients
S. Marrocco, K. Patterson

PUBLISHED ABSTRACTS

October 2014 American Congress of Rehabilitation Medicine Conference, Toronto, ON
Mirror therapy for the lower-extremities post-stroke: a case series
Crosby L, **Marrocco S**, Brown J, Patterson K

July 2014 ISPGR World Congress, Vancouver, BC
Asymmetric pattern of lower extremity loading during post-stroke gait
Patterson KK, **Marrocco S,** Parsons S, Jones I, Birmingham TB

TEACHING EXPERIENCE

Jan – Dec 2013 Graduate Teaching Assistant
Systematic Approach to Functional Human Anatomy – HS2300
School of Health Studies, University of Western Ontario

ACADEMIC COMMUNITY INVOLVEMENT

May 2013 – 2014 VP Student Development
Health and Rehabilitation Sciences Graduate Student Society
University of Western Ontario

April 2010 – 2011 Charity Executive Committee
Volunteer Coordinator
Western University Student Council
University of Western Ontario

WORK EXPERIENCE

Sept 2013 – Present	Child Minder, All Kids Belong London Children's Connection, London, ON *Duties:* Ensuring safety for children; encouraging learning experiences and problem solving; communicating with parents
March 2011 – Dec 2013	Personal Trainer Body Logic Personal Training, London, ON *Duties:* Designing personalized routines relative to client's needs; goals and abilities; upholding health and safety standards; invoicing; promoting the gym and marketing myself; counselling; interviewing clients; providing nutritional advice; providing feedback and coaching; ensuring safety of clients
May 2011 – May 2013	Server J. Dee's Market Grill, London, ON *Duties*: Customer service; bar tending; balancing/counting float; quality assurance; maintaining cleanliness
May 2006 – August 2010	Supervisor Shoppers Drug Mart, Ottawa ON *Duties:* Providing customer service; supervising other employees; counting the safe; ensuring each till was balanced; balancing and maintaining inventory of lottery tickets; ensuring confidentiality; securing the store

LANGUAGE COMPETENCIES

Fluently bilingual, English and French

CERTIFICATIONS

- Tri-Council Policy Statement: Ethical Conduct for Research Involving Humans Course on Research Ethics (TCPS 2: CORE)
- National Institute of Health (NIH) Stroke Scale, American Heart Association
- Canadian Society of Exercise Physiology – Certified Personal Trainer
- CPR-C, Standard First Aid