QUANTIFICATION OF ASYMMETRICAL STEPPING POST-STROKE AND ITS RELATIONSHIP TO HEMIPARETIC WALKING PERFORMANCE

By

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To Mummy, Appa and Anjan

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TABLE OF CONTENTS

page

ACKNOWLEDGMENTS 4

LIST OF TABLES 10

LIST OF FIGURES 11

ABSTRACT 13

CHAPTER

1 INTRODUCTION 15

2 LITERATURE REVIEW 18

Introduction 18
Part 1: Overview of Walking Function 18
Neuromotor Control of Healthy Gait 18
Quantifying Gait: Phases in the Healthy Gait Cycle 19
Walking Capacity after a Stroke 19
Stroke: incidence and consequences 19
Pathophysiological basis of the locomotor disorder post-stroke 20
Nature and rate of walking recovery 21
Walking disability in the chronic phase 22
Part 2: Asymmetry in Spatiotemporal Step Characteristics and Ground Reaction Forces during Hemiparetic Walking 22
Spatiotemporal Characteristics of Steps 23
Temporal asymmetry 24
Spatial asymmetry 25
Ground Reaction Forces in Hemiparetic Gait 26
Relevance of the Reviewed Literature to Study One 27
Part 3. Intra-Subject Variability during Walking 28
Motor Control and Gait Variability 28
Significance of gait variability: theoretical approach 28
Role of variability during walking 28
Step-by-Step Variability in Spatiotemporal Characteristics 29
Step Variability as Quantifiable Markers of Impaired Walking 30
Relevance of Reviewed Literature to Study Two 32
Part 4. Foot Placement in a Body Reference Frame 32
Defining Foot Placement Relative to Body 33
Control of Foot during Gait 33
Body Center of Mass Position and Velocity: Postulated Link to Foot Placement 33
Foot Placement Relative to Body and its Relation to Walking Sub-tasks 34
Relevance of Reviewed Literature to Study Three 35
Part 5. Pre-Swing and Swing Phase during Hemiparetic Gait 36

Introduction to Pre-Swing and Swing Phase during Gait....36
Kinematic Characteristics during Pre-Swing and Swing Phase....37
Kinetic Characteristics during Pre-Swing and Swing phase....38
Moments, Powers, Angular velocities....38
Interjoint coordination during swing phase....40
Muscle Activity during Pre-Swing and Swing Phase....41
Relevance of Reviewed Literature to Study Four....42

3 RELATIONSHIP BETWEEN STEP LENGTH ASYMMETRY AND WALKING PERFORMANCE IN SUBJECTS WITH CHRONIC HEMIPARESIS....49

Introduction....49
Methods....51
Participants....51
Procedures....52
Data Analyses....53
Statistical Analyses....54
Results....55
Relationship between Step Length Asymmetry (SLR) and GRFs....55
Relationship between Asymmetrical Step Lengths, Hemiparetic Severity and Walking Speed....57
Relationship between Step Length Asymmetry, Time Spent in Pre-Swing and Swing Time....57
Relationship between SLR, Change in Gait Speed and Parameters That Contribute to Change in Speed....57
Discussion....58
Relationship between Step Length Asymmetry and Propulsive Forces during Hemiparetic Walking....58
Relationship between Step Length Asymmetry, Walking Speed and Hemiparetic Severity....61
Relationship between Step Length Asymmetry, Paretic Pre-Swing Time and Vertical GRFs....62
Conclusions....63

4 VARIABILITY IN SPATIOTEMPORAL STEP CHARACTERISTICS AND ITS RELATIONSHIP TO WALKING PERFORMANCE POST-STROKE....70

Introduction....70
Methods....71
Participants....71
Procedures....72
Data Analyses....73
Statistical Analyses....74
Results....75
Differences in Step Variability between Healthy and Hemiparetic Walking....75
Association between Step Variability, Clinical Assessments and Asymmetry Index....76
Discussion....77

Differences in Step Variability between Healthy and Hemiparetic Participants 77
Relationship between Hemiparetic Step Variability and Impaired Performance Post-Stroke 78
Study Limitations 81
Conclusions 82

5 FOOT PLACEMENT IN A BODY REFERENCE FRAME DURING WALKING AND ITS RELATIONSHIP TO HEMIPARETIC WALKING PERFORMANCE 88

Introduction 88
Methods 90
Participants 90
Procedures 91
Data Analyses 92
Statistical Analyses 94
Results 95
Quantifying Foot Placement Relative to the Pelvis 95
Relationship between Anterior-Posterior Foot Placements Relative to Pelvis, Step Length Asymmetry and Paretic Propulsion 96
Relationship between Medial-Lateral Foot Placements Relative to Pelvis, Step Widths, Paretic Weight Support and Dynamic Stability Margin 97
Discussion 98
Anterior-Posterior Foot Placement Relative to Pelvis and its Relationship to Step Length Asymmetry and Forward Progression 98
Medial-Lateral Foot Placement Relative to Pelvis and its Relationship to Weight Supported on Paretic Leg and Dynamic Stability Margin 101
Limitations 103
Conclusions 104

6 EVALUATION OF STEP LENGTH GENERATION DURING POST-STROKE HEMIPARETIC WALKING USING A NOVEL METHODOLOGY OF STEP-BY-STEP VARIABILITY IN GAIT DATA 112

Introduction 112
Methods 115
Participants 115
Procedures 116
Data Analyses 117
Statistical Analyses 118
Results 120
Predictors of Step Length Variability and the Differences in Selected Predictor Variables across the Asymmetrical Sub-Groups 120
Shorter paretic group 120
Symmetric group 121
Longer paretic group 121
Discussion 122

Contralateral Stance Leg Ground Reaction Force (AP Impulse during Ipsilateral Swing) is a Significant Predictor of Step Length Variability 123
Ipsilateral Hip Impulse in Early Swing is a Significant Predictor of Step Length Variability in Persons taking Longer Paretic than Non-Paretic Steps 124
Ankle-Joint Center Velocity at Toe-Off is a Significant Predictor of Non-Paretic Step Length Variability 126
Leg Orientation at Toe-Off and Pelvis Velocity at Toe-Off and their Contribution to Explaining Step Length Variability 126
Within-Subjects Regression Models 127
Limitations 128
Conclusions 129

7 CONCLUSIONS: INTEGRATING THE FINDINGS 140

Step Length Asymmetry during Hemiparetic Walking 140
Step Variability during Hemiparetic Walking 141
Asymmetrical Stepping in a Body Reference Frame Post-Stroke 142
Step Length Generation during Hemiparetic Walking 142
Summary 143

APPENDIX

A LOWER EXREMITY FUGL-MEYER SCALE 144

B DYNAMIC GAIT INDEX SCALE 145

LIST OF REFERENCES 147

BIOGRAPHICAL SKETCH 163

LIST OF TABLES

Table		page
3-1.	Correlation between SLR and walking variables	64
4-1.	Definitions of study variables	82
4-2.	Step variability (expressed as standard deviation) within the hemiparetic population sub-divided based on their performance measures	83
5-1.	Definition of study variables	105
6-1.	Subject characteristics	130
6-2.	Average gait characteristics for individual participants	131
6-3.	Regression models for individual participants to predict paretic step length variability	132
6-4.	Regression models for individual participants predicting non-paretic step length variability	133

LIST OF FIGURES

Figure page

2-1. A simplified model demonstrating neural control of gait 43

2-2. Division of a gait cycle into phases 44

2-3. Methodology for collection of spatiotemporal characteristics during walking using an instrumented mat (GAITRite) 45

2-4. Illustration of ground reaction forces exerted by the limbs and typical force curves 46

2-5. Variability (in stride time) during walking and its relation to risk for falls 47

2-6. Power profiles in swing phase of a healthy gait cycle 48

3-1. Illustration of horizontal GRF impulses 65

3-2. Comparison of GRFs between the paretic and non-paretic legs for subjects walking with differing SLR 66

3-3. Relationship between step length ratio and Propulsion $_{Paretic}$ 67

3-4. Relationship between step length asymmetry, walking speed and hemiparetic severity 68

3-5. Change in speed, cadence and individual step lengths in subjects walking at different SLR [SLR > 1.1 (n = 21), 0.9 < SLR < 1.1 (n = 21), SLR < 0.9 (n = 4)] 69

4-1. Differences in temporal variability between healthy (n = 22) and participants with hemiparesis (n = 94) at Self-selected (SS) walking speeds 84

4-2. Differences in spatial variability between healthy (n = 22) and participants with hemiparesis (n = 94) at Self-selected (SS) walking speeds 85

4-3. Differences in temporal variability in hemiparetic participants based on their performance on clinical assessments 86

4-4. Differences in spatial variability in hemiparetic participants based on their performance on clinical assessments 87

5-1. Illustration of marker positions for kinematic data collection and the SIMM model generated 106

5-2. Calculation of anterior-posterior and medial-lateral foot placements relative to pelvis 107

5-3. Foot placement relative to pelvis during hemiparetic and healthy gait 108

5-4. Relationship between anterior foot placement asymmetry relative to pelvis and step length asymmetry in participants with hemiparesis....109

5-5. Relationship between step length asymmetry and anterior-posterior foot placement relative to pelvis in participants with hemiparesis....110

5-6. Relationship between paretic and non-paretic lateral foot placement asymmetry relative to pelvis and percent weight supported on the paretic leg....111

6-1. An individual participant walking on the split belt treadmill as kinematic, kinetic and EMG data were recorded....134

6-2. Illustration of variables used in the study....135

6-3. Frequency distribution of step-to-step variability in step lengths....136

6-4. Non-paretic AP impulse and Paretic hip impulse during paretic stepping in the asymmetrical sub-groups....137

6-5. Relationship between paretic hip impulse in early swing, non-paretic AP impulse in early and late swing in an individual participant taking longer paretic steps than non-paretic....139

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Asymmetrical stepping is a characteristic feature of hemiparetic walking and a result of sensorimotor deficits post-stroke. Asymmetry measures (that is, relative performance of paretic leg) may characterize hemiparetic gait better than overall gait performance measures (such as gait speed) and can provide insights into underlying paretic leg impairments. Therefore, the major purpose of this dissertation was to quantify the asymmetry in steps post-stroke and understand its relationship to hemiparetic walking performance. Overall, four studies were conducted. Persons with chronic hemiparesis and healthy controls walked overground and over a split-belt instrumented treadmill as spatiotemporal, kinematic and kinetic data were collected. Clinical assessments included lower-extremity Fugl-Meyer grading and Dynamic Gait Index assessments.

In study one, step length asymmetry during walking was quantified. Results showed that step length asymmetry related to propulsive force generation during hemiparetic walking. Further, asymmetrical step lengths may not necessarily limit the self-selected walking speed likely due to other compensatory mechanisms. We suggest that step length asymmetry can be utilized as a clinical measure to evaluate asymmetrical stepping post-stroke.

In study two, step-by-step variability and its relation to asymmetrical stepping were investigated. Results showed that increased spatiotemporal variability, asymmetry in swing and pre-swing time variability and reduced width variability were related to severe hemiparesis, asymmetrical stepping and poor balance. We suggest that step-by-step variability measures are quantifiable markers of impaired walking performance post-stroke.

In study three, asymmetrical stepping was evaluated in a body reference frame. Results showed that anterior-posterior and medial-lateral foot placements relative to body were asymmetrical and this foot placement asymmetry related to step length asymmetry but not step widths. Wider paretic foot placement relative to pelvis than non-paretic also related to reduced paretic leg weight support and lateral instability, suggesting the clinical utility of medial-lateral foot placement relative to pelvis as an outcome to quantify weight support during hemiparetic walking.

In study four, mechanisms underlying step length generation were evaluated. Contralateral anterior-posterior and hip impulses during swing explained the step length variability in the majority of participants. However, relationship of the predictors to step lengths differed in the asymmetrical sub-groups. This implies that mechanisms of step length generation were different across persons showing differing step length asymmetry patterns. Based on these mechanisms, we have proposed specific impairments and therapeutic strategies targeted towards these impairments underlying asymmetrical stepping.

CHAPTER 1
INTRODUCTION

The American Heart Association reports approximately 700,000 cases of stroke or cerebrovascular accident annually [1] . Stroke is the number-one cause of long-term disability around the world [2] and is estimated to result in $30 billion in health care costs and lost productivity each year [3]. As the most disabling chronic disease, the cumulative consequences of stroke are often staggering for individuals, families, and the society [3, 4]. Hemiparesis (unilateral movement dysfunction or weakness of one-half of the body) is commonly seen in three-quarters of persons post-stroke [5]. The residual sensorimotor control deficits of a person with post-stroke hemiparesis involves multiple impairments such as muscle weakness, abnormal synergistic organization of movement, impaired force regulation, abnormal muscle tone, impaired balance and sensory deficits [2, 5, 6]. Additionally, residual cognitive and visual deficits contribute to the reduced functional mobility in this population [7, 8].

Regaining independent walking is the most often-stated goal for rehabilitation in patients post-stroke [9]. Walking deficits in those who have sustained a stroke range from complete immobility to independent mobility, with almost a third of stroke survivors showing severe walking impairments [10]. Although post-stroke hemiparesis appears to reflect a single diagnostic category, there is immense heternogenity in the walking performance post-stroke [11], warranting a systematic characterization of walking performance deficits and underlying impairments. Quantification of impairments that limit functional walking performance and investigation of mechanisms underlying these impairments will directly assist in development of targeted therapies to improve hemiparetic walking function.

Asymmetrical performance between lower extremities is a characteristic hallmark that is unique to hemiparetic gait [12, 13]. While the asymmetrical nature of hemiparetic walking is

well documented in the literature [12-15], there is insufficient understanding of the underlying mechanisms related to gait asymmetry. One of the primary reasons limiting an integrated understanding of gait asymmetry post-stroke is the heterogeneity in asymmetrical patterns among stroke patients primarily due to the diverse motor recovery processes and compensatory mechanisms [16]. Furthermore, the relationship of gait asymmetry to functional performance is unclear, primarily because functional performance has been traditionally evaluated using walking speed [17]. Gait asymmetry is reported to show a weak relation to attained walking speed implying that asymmetry may not limit the functional performance [5, 17-19]. However, faster walking speeds in some persons showing severe asymmetry can be achieved by compensatory strategies from the non-paretic leg, limiting the specific understanding of the contribution of paretic leg performance to functional performance. While it can be argued that compensations post-stroke are central to the walking performance due to altered circuitry and limited ability after a neurological injury [18], it is essential to differentiate impairments from compensations such that therapeutic strategies can be specifically designed to improve the paretic leg performance.

Furthermore, there is no clear consensus in the literature regarding the clinical relevance of evaluating gait asymmetry [5]. As a clinical measure, gait velocity reflects overall gait performance but is limited in its value to evaluate post-stroke gait specific to paretic leg impairments [14]. In turn, asymmetry measures (i.e., relative performance of the paretic leg) can provide insights on the paretic leg performance in relation to the non-paretic leg. Therefore, asymmetry measures could be developed as outcomes that specifically reflect the contribution of the paretic leg towards functional walking. The aim of this dissertation was to systematically quantify asymmetrical performance (specifically in stepping measures) during hemiparetic

walking, to explore underlying mechanisms and to understand how asymmetry might relate to walking performance post-stroke. In particular, this dissertation will focus on stepping (spatial-temporal) asymmetry. A step during walking is the final outcome of all events occurring in the gait cycle [20]. Therefore, spatial-temporal characteristics of steps (such as step length, swing time, etc.) can be used as quantifiable measures of walking performance reflective of underlying gait processes [21]. Furthermore, stepping parameters can be easily recorded enhancing their clinical utility as gait performance measures.

In this dissertation, four studies were conducted to quantify asymmetrical stepping and understand its relationship to hemiparetic walking performance. In the first study, step length asymmetry during walking was quantified and the relationship between spatial asymmetries and hemiparetic walking performance was explained. In the second study, the step-by-step variability in selected spatial and temporal characteristics of steps was evaluated and their relation to stepping asymmetry and hemiparetic performance was explained. The third study of this dissertation evaluated asymmetrical stepping in a body reference frame to gain insights regarding asymmetrical foot placement relative to the body during hemiparetic walking. Finally, in the fourth study of this dissertation, mechanisms related to initial conditions of the leg and swing phase were investigated to explain step length generation during hemiparetic walking. The overall purpose of studies in this dissertation was to provide a foundation for the development of a framework to use asymmetry measures to assess walking impairments post-stroke. Specificity in assessment of walking impairments will, in turn, facilitate the development of rehabilitation strategies that are cost-effective and efficient.

CHAPTER 2
LITERATURE REVIEW

Introduction

The following literature review is composed of six main parts and provides the background underlying specific aims of this dissertation study. Part one, is an overview of walking function and describes walking deficits of persons who have sustained a stroke. In the second part, literature related to motor control mechanisms in hemiparetic walking is presented. This section presents background literature relevant to Study One (Chapter 3). In the third part, intra-subject variability during walking is discussed in relevance to Study Two (Chapter 4) of this dissertation. Part four describes literature related to foot placement during walking and forms the basis for aims of Study Three (Chapter 5). In part five, pre-swing and swing phase control during walking is discussed and this part focuses on the literature relevant to Study Four (Chapter 6) of this dissertation.

Part 1: Overview of Walking Function

Neuromotor Control of Healthy Gait

Walking is essentially an inter-limb coordinated movement in which the limbs move in a symmetrical alternating pattern such that the body can progress forward in a stable and efficient manner [22, 23]. Human walking is remarkable in that the healthy locomotor system integrates input from the motor cortex, cerebellum, and the basal ganglia, as well as synchronizes feedback from visual, vestibular and proprioceptive sensors to produce carefully controlled motor commands that result in coordinated muscle firings and limb movements [24]. Figure 2-1 demonstrates a model of walking from previous works [25, 26]. While the spinal pathways (central pattern generators) can generate the basic locomotor rhythm, sensory inputs from higher

centers (through descending pathways as corticospinal tracts) and feedback from peripheral mechanisms (afferent feedback) enable a rich variation in the basic locomotor rhythm.

Furthermore, the three major requirements for successful locomotion are suggested to be a) progression, defined as the ability to generate a basic locomotor pattern that can move the body in a desired direction; b) stability, defined as the ability to support and control the body against gravity; c) adaptability, defined as the ability to meet the individual's goals and the demands of the environment [27].

Quantifying Gait: Phases in the Healthy Gait Cycle

Walking function is usually quantified as a Gait cycle. A Gait (Stride) cycle is defined as the events occurring from foot strike of one limb to the foot strike of the ipsilateral limb [28, 29]. Each gait cycle is further divided into a stance phase (as when ipsilateral limb is on the ground) and swing phase (as when ipsilateral limb is swinging with no contact on the ground) [29]. Stance and Swing phases of a gait cycle can be further sub-divided into different phases. Figure 2-2 presents these sub-divisions of a Gait cycle phase.

Walking Capacity after a Stroke

Stroke: incidence and consequences

O'Sullivan et al. (2000) has defined a stroke, or cerebrovascular accident (CVA), as "an acute onset of neurological dysfunction due to an abnormality in cerebral circulation with resultant signs and symptoms that correspond to involvement of 19 focal areas of the brain" [30]. The American Heart Association reports stroke as a common neurological event occurring in 700,000 people annually. Over 4 million people currently live with residual deficits [31]. Stroke is the primary cause of long-term disability and is classified as one of the most disabling chronic diseases. It has been estimated that one in five stroke survivors need help walking and seven out of ten cannot return to their previous jobs [32]. Fifty one percent of stroke survivors are unable to

return to any type of work [33]. In particular; ability to return to work is primarily shown to relate to walking function [34, 35].

Pathophysiological basis of the locomotor disorder post-stroke

The pathophysiological basis of walking disability after a stroke is damage to motor neurons and pathways of the central nervous system caused by interruption of arterial blood supply because of a hemorrhage (hemorrhagic stroke) or thrombus (ischemic stroke) usually on one side of the brain [35]. Consequently, paresis (or paralysis) is observed in opposite half of the body (hemiparesis) [36-38]. The types and degrees of disability that follow a stroke primarily depend upon multiple factors such as location and size of brain lesion, severity of the lesion, individual degree of spontaneous recovery, and the duration of stroke onset [39, 40]. Nonetheless, residual deficits are common after a stroke. Typical residual deficits after a stroke include sensorimotor, cognitive and visual deficits, all of which can independently or in combination result in reduced or impaired walking ability.

Motor control impairments of weakness (paresis) [41], loss of volitional movements of the weaker or paretic side (opposite to lesion) or inappropriately graded muscle activations of the weaker side affect locomotor performance immediately after a stroke [36, 39]. Impairments of spasticity and changes in the mechanical properties of muscles further contribute to walking disability, developing a few weeks after the initial insult [42]. Nonetheless, damage to the motor control system and the residual impairments vary with the nature and extent of brain lesion [35]. Therefore, while there are some common motor control impairments that affect walking performance after a stroke, several sub-groups within the population can be identified that present differing motor control impairments.

Nature and rate of walking recovery

Mechanisms of recovery: Recovery following a stroke is a complex process involving both spontaneous recovery and recovery due to the effects of a therapeutic intervention that are usually difficult to separate [5]. In general, while the mechanisms of locomotor recovery after a stroke are largely unknown, it is suggested that cortical and spinal reorganization [43], functional compensation from existing pathways [44], new neuronal sprouting [44] and spinal and afferent reflex modulation [45, 46] are potential mechanisms that contribute to functional locomotor recovery.

Furthermore, motor recovery processes that underlie walking recovery specifically can be explained (at least partially) by the step-wise recovery process earlier proposed by Twitchell and colleagues [47]. Twitchell et al. (1951) argued that motor recovery follows a step-wise and predictable sequence after an initial stage of areflexia and flaccid paralysis. After this initial stage, reflexes return, become hyperactive, muscle tone increases and spasticity develops. In the next stage, voluntary movement appears as part of stereotyped, reflexive flexor and extensor muscle "synergies", after which voluntary movement may be achieved "out of synergy". Finally, normalization of muscle tone and reflexes may occur.

Rate of recovery: Only 23 – 37 % of persons who have sustained a stroke are able to walk independently during the first week [11], but there is a general agreement that 50 – 80 % of survivors can walk at 3 weeks or at discharge [48], and by 6 months as many as 85 % of the population can walk [49]. While it is generally assumed that walking function recovery plateaus after 3 months, there is some evidence that recovery may continue up to 2 years (using gait speed as the outcome measure) [50, 51]. Nonetheless, residual walking impairments last several months to years after the acute onset. Further, these residual impairments limit walking performance [52].

Walking disability in the chronic phase

While, in general, 85% of stroke survivors can walk by 6 months of stroke, the quality of walking remains impaired amongst most patients. Walking after a stroke is characterized by slow walking speed [53], poor endurance [54] and impaired muscle coordination [55, 56]. Specifically, the impaired muscle coordination after post-stroke hemiparesis results in the characteristic asymmetrical nature of walking commonly referred to as 'Hemiparetic Gait'. Furthermore, impaired muscle coordination after a stroke significantly limits the walking ability of persons and restricts their independent mobility about the home and community [57]. Further, difficulty in walking is associated with limited ability to return to work [58] and deterioration in the quality of life.

Risk for falls while walking: Persons who have sustained a stroke are at a high risk for falling [59, 60]. Since gait and balance deficits are primary contributors to falls, risk for falling further increases significantly in those stroke survivors who are ambulatory and have balance deficits [61]. Consequently, a fall after a stroke compounds to the post-stroke disability. For most patients with stroke, functional walking ability is rarely regained even at the end point of their rehabilitation and several therapeutic interventions are aimed at maximizing walking recovery. Nevertheless, there is a lack of targeted interventions to improve walking ability. In part, the problem is because walking impairments are not specifically quantified [62]. Therefore, quantification of walking impairments is necessitated to assist in development of focused therapeutic strategies.

Part 2: Asymmetry in Spatiotemporal Step Characteristics and Ground Reaction Forces during Hemiparetic Walking

Inter- [63] and intra-limb [64] coordination deficits are commonly observed during post-stroke hemiparetic walking. These coordination deficits along with sensorimotor and motor

control deficits result in the asymmetrical performance after stroke. In fact, asymmetry in motor performance between the paretic and non-paretic legs is a characteristic feature of hemiparetic gait [12, 65, 66].

In particular, spatiotemporal characteristics of steps and Kinetic (Ground Reaction Forces) parameters during walking are commonly used to characterize both overall walking performance and specifically asymmetrical performance. Spatiotemporal step characteristics and kinetic parameters are most relevant to studies in this dissertation and therefore are reviewed below.

Spatiotemporal Characteristics of Steps

Spatial (i.e., length and width) and temporal (i.e., timing of events) characteristics of steps are commonly examined since they represent the final outcome of collective motions that contribute to walking [20, 67]. These parameters are also clinically relevant since they are both easily observable and quantifiable. Figure 2-3 shows a common methodology for collecting spatiotemporal (ST) parameters during gait using an instrumented walkway (GAITRite). Given the clinical relevance of the ST parameters of steps, characterization of these parameters and quantification of underlying mechanisms during hemiparetic gait can provide useful insights regarding hemiparetic gait.

The most consistently reported spatiotemporal parameter is slower walking speed. In 17 studies reporting gait speed, the average speeds ranged from 0.23 ± 0.11 m/s [48] to 0.73 ± 0.38 m/s [11]. The attainable maximal speed is also limited by functional limitations imposed due to the pathology of stroke [6]. Consistent with slower walking speeds, persons with hemiparesis take shorter stride lengths and have lower cadence (number of steps taken per minute) compared to age-matched healthy adults [13, 68]. Slower gait speed after a stroke is also associated with a longer gait cycle duration [13, 68]. Further, the proportion of time spent in stance versus swing is

also altered on both paretic and non-paretic sides, when compared to healthy adults walking at normal speeds [5, 13, 16].

While, gait speed or other unilateral measures reflect overall gait performance; asymmetry in ST parameters of steps can reveal deficits in motor coordination between the paretic and non-paretic legs and the performance of the paretic leg relative to the non-paretic. Literature specific to spatiotemporal asymmetry during hemiparetic gait is reviewed in the following paragraphs.

Temporal asymmetry

Asymmetries in temporal parameters of steps in persons who have sustained a stroke are well documented [12, 13] and have been consistently related to disturbances in motor coordination [5]. In particular, it is reported that persons after a stroke spend longer time bearing weight on the stance phase of the non-paretic leg than paretic [16]. Further, stance phase of both paretic and non-paretic sides is longer in duration and occupies a greater portion of the full gait-cycle [5] as compared to both age-matched and speed-matched healthy adults [69].

Similarly, it is reported that persons after a stroke spend longer time swinging their paretic leg, likely because they spend longer time bearing weight on the non-paretic leg (i.e., longer stance time) [15]. This asymmetry in swing time is reported to be a significant predictor of hemiparetic walking performance (since it strongly correlates with stages of motor recovery, walking speed and falls) [13, 15]. Furthermore, a greater proportion of cycle time is spent in double support phase of the gait cycle during hemiparetic gait. Particularly, of the two double support phases, relatively greater time is spent in the second double support of the paretic gait cycle (paretic pre-swing phase) than the non- paretic [5]. Further, Dequervain et al. (1996) reported that the paretic pre-swing phase was markedly prolonged for those persons who had very slow gait velocities [70]. Specifically, it is suggested that this prolonged paretic pre-swing duration indicates a poor progression of hip flexion during swing phase of patients with slow gait

velocity [69, 70]. In summary, several aspects of temporal asymmetry are well characterized in the literature and temporal asymmetry has been consistently related to poor motor performance during hemiparetic walking.

Spatial asymmetry

While temporal asymmetry is well characterized and the direction of asymmetry is consistently reported in the literature, direction of spatial asymmetry varies across studies. It has earlier been reported that, after a stroke, patients may walk with either relatively longer paretic steps or longer non-paretic steps [19, 65, 69]. In a study by Kim et. al. (2003), considerable variability in step length asymmetry was observed in a sample of 28 chronic stroke survivors. While 14 of these 28 participants walked with longer paretic steps than non-paretic, 14 others walked with relatively longer non-paretic steps. Dettman et al. (1987) and Hsu et al. (2003) reported that while, on an average persons with stroke walked with longer paretic than non-paretic steps, step length patterns were inconsistent within sub-groups of the population [65, 71]. Thus, it is unclear whether persons within the hemiparetic population walk with one or the other pattern or both.

Furthermore, reasons for the variability in step length asymmetry patterns and relationship of these variable patterns to walking performance are unexplained. For example, Kim et al. (2003) hypothesized that the variability in patterns of step length asymmetry may be due to compensatory strategies that increase or decrease the step length of either paretic or non-paretic limb [19]. However, they were unable to advance the discussion since they found a non-significant relationship between step length symmetry and symmetry in vertical ground reaction forces. Further, in a recent review article, Lamontagne and colleagues [53] suggest that an inconsistency in the direction of asymmetries between paretic and non-paretic legs is the result of differences in walking ability in the subjects, but they do not specifically report these differences.

Another reason for insufficient understanding of the affect of step length asymmetry on hemiparetic walking performance is that the relationship between step length asymmetry and walking speed is not well documented in the current literature. For instance, shorter stride lengths (bilaterally) have been related to slower walking speed and thereby poor walking performance [70], and yet a non-significant relationship has been suggested between step length asymmetry and walking speed [19]. Since kinetic characteristics represent the underlying causes for the kinematic and spatiotemporal patterns [72], it might be useful to examine the kinetic characteristics to quantify the step length asymmetry.

Ground Reaction Forces in Hemiparetic Gait

Ground reaction forces (GRFs), as measured by force platforms, reflect the net vertical and shear forces acting on the surface of the platform [73]. Forces are exerted by the limbs (due to muscle activity) on the ground while a person walks and is recorded as the equal and opposite reaction force exerted by the force platforms in response (Figure 2-4). Mathematically, GRFs are the algebraic summation of the mass-acceleration products of all body segments while the foot is in contact with the platform [73]. The net GRF has three components: Vertical, Horizontal and Mediolateral. The vertical force has a characteristic double-hump (first related to weight acceptance and the second related to push-off), (Figure 2-4). The horizontal force has a negative phase in the first half of stance (indicating a net deceleration or braking of the body) and a positive phase in the second half (indicating a net acceleration or propulsion of the body forward), (Figure 2-4).

The vertical force curve is shown to be variable across subjects and most commonly has an initial low peak. Carlsoo et al. (1974) reported three different patterns of Vertical GRFs: 1) first peak during heel contact and second during push-off, 2) pattern showing continuous plateau, 3) pattern showing single peak in midstance [74]. Kim et al (2003) further showed that the

symmetry in vertical GRFs is accompanied by symmetry in temporal parameters but not in the symmetry of distance variable [19]. This is expected since the vertical GRFs primarily act on the vertical acceleration of the center of mass and symmetry in distance variables are likely related to a horizontal component of the GRF (i.e., Anterior-Posterior GRF). Conversely, Mediolateral GRFs during hemiparetic walking have not been systematically reported. Rogers and associates study on voluntary leg flexion movements in the hemiparetic persons provides some insight into the relevance of the M-L forces to stepping [75]. The results of their study revealed asynchrony and reversals in usual directions of lateral forces, suggesting the inter-limb coordination deficits in this population.

In a recent study, A-P GRFs during hemiparetic walking was quantified for the first time [76]. Bowden et al. (2006) showed that anterior (propulsion) forces by the paretic leg are reduced compared to the propulsive forces by the non-paretic leg [76]. In this study, a measure from the A-P forces was developed that quantified the coordinated output of the paretic limb to the task of body propulsion during walking. This measure was referred to as paretic propulsion (Pp), which represented the percentage of total propulsion generated by the paretic leg during walking. Pp was also found to correlate with both walking speed and hemiparetic severity.

Relevance of the Reviewed Literature to Study One

Overall, current evidence suggests that persons after a stroke walk with different patterns of step length asymmetry that may be unrelated to the attained walking speed. However, these spatial asymmetries are not quantified and it is unclear how the different asymmetrical patterns relate to post-stroke hemiparetic walking performance and why asymmetry in step lengths may not necessarily limit the attained walking speed. Therefore, study one of this dissertation aimed at quantifying the step length asymmetry and explaining its relation to hemiparetic walking performance.

Part 3. Intra-Subject Variability during Walking

While variability during walking exists both between individuals (inter-subject) and within individuals (intra-subject), intra-subject variability in the performance of tasks has received less attention [77]. Intra-subject variability during walking is the variation observed in an individual's walking performance (i.e., variability in steps for individual participants). No two steps during walking are exactly similar and there is some natural variability from step to step [78]. Walking variability can be quantified using spatiotemporal, kinematic, kinetic and EMG characteristics.

Motor Control and Gait Variability

Significance of gait variability: theoretical approach

Walking is a rhythmical inter-limb coordinated task and it is suggested that pattern generators located in the spinal cord generate the basic motor rhythm during walking [79]. These pattern generators are considered to be closely coupled for walking movements, suggesting little variability (stability) in the pattern of walking. Nevertheless, walking movements are not strictly rhythmical and emerge as a consequence of the interaction of neural and mechanical dynamic systems, pattern generators, modulation from supraspinal neural system and afferent modulation [80, 81]. These multiple modulation in the neuromuscular system may induce variability in walking movements. Walking, specifically, is an example of flexible coordination where stability co-exists with the abundant variability in movements [82-84]. Further, the degree of the variability has also been linked to the health of a biological system [78, 85], suggesting that impairments in gait might alter the variability. In summary, coordination patterns like walking are highly flexible, being simultaneously stable and variable.

Role of variability during walking

The traditional approach in motor control is to consider intra-subject variability as an index of noise in the sensorimotor system [86]. More recently, positive aspects of movement variability

have been proposed [87-89]. Such perspectives suggest that variability in movement constitutes a pattern of stimulation. This pattern of stimulation provides task-relevant information about the dynamical interaction between a person and the environment [88]. There is also some indication that intra-subject motor variability can well predict performance of motor tasks and that variability is essential for many aspects of motor performance [86]. Furthermore, stride-to-stride variability in gait parameters might reflect the inherent flexibility in the locomotor system [90] and therefore might be a requisite for adaptability.

Step-by-Step Variability in Spatiotemporal Characteristics

Gait variability is most commonly quantified as variability in spatiotemporal (ST) characteristics of steps. The concept of variability within the ST parameters, its quantification and relation to falls risk is presented in Figure 2-5. Stride-to-stride variability in stepping patterns during walking is consistently reported to be low in healthy persons [91, 92] during free unperturbed walking at natural walking speeds. Kinetic and EMG variability is also reported to be low in healthy gait [93, 94], although higher than variability in ST parameters of steps.

The magnitude or degree of variability is commonly reported, using parameters like standard deviation (SD) [95, 96] and coefficient of variation (CV) [97, 98]. However, recent studies suggest the use of measures that can quantify not only the magnitude of variability, but also the structure of variability (e.g. fractal dynamics of gait rhythm) [99, 100]. In Study two of this dissertation, only the magnitude of variability will be evaluated.

SD reflects the absolute variation of a parameter while CV, is the variability computed relative to the mean of the distribution [CV= standard deviation / mean]. SD and CV are expected to correlate because they are mathematically derived similarly (i.e., CV is defined from SD). SD and CV are also reported to be correlated in patient populations [101]. There is no clear

consensus in the literature regarding the measure suited to quantify variability and there are few reports on reliability and validity of each of these measures.

Advantages and disadvantages of gait variability measures: SD is reported to be unrelated to the mean of the parameter distribution [102] and therefore, might better quantify the absolute variation across parameters or when comparisons are made within variables across persons. In cases where comparisons of variability are made across parameters, CV might be more advantageous since it normalizes the variation in reference to the individual mean of each parameter distribution [86]. However, extremely low parameter means can drive the CVs to infinity and thereby, suggest spuriously large variations.

Step Variability as Quantifiable Markers of Impaired Walking

Increased or decreased variability is commonly reported in populations with gait abnormalities like elderly fallers [103, 104], older frail adults [105] and persons with neuro-degenerative diseases (e.g, Parkinson's disease) [106, 107], suggesting that gait variability strongly associates with gait impairments. It is also suggested that alteration in gait variability is specific to pathology and that healthy aging might not alter gait variability [108].

Step variability and its relation to gait impairments: Altered gait variability has been strongly related to walking impairments and is suggested to be a quantifiable biomechanical marker to evaluate impaired performance [77]. Increased gait variability has been related to slower gait speed and poor cognitive status in adult fallers [77, 96, 109]. Similarly, central nervous system impairments (like cognitive functioning and motor performance) have been related to increased stance time variability [110], while decreased step width variability has been related to sensory impairments and balance deficits during walking [104, 110, 111]. Gait variability is also suggested to predict mobility disability [110]. Increased gait variability has also been related to risk for falls, implying that excess variability in steps might relate to balance

impairments [101]. Further, there is a strong suggestion in the literature that altered (increased or decreased) variability in steps is directly related to dynamic balance impairments since altered variability has consistently shown to predict the risk for both past and future falls and those falls specific to walking [77, 96, 101, 103].

Direction of alteration in gait variability: Motor control theories (like older hierarchical models and more recent dynamical systems theories) support both views of increased and decreased step variability as being beneficial to walking. While older motor control theories suggest that increased motor variability is reflective of decreased motor skill, more recently positive aspects of movement variability have been proposed [86]. It is suggested that stride-to-stride variability in gait parameters might reflect the inherent abundance of the locomotor system and therefore might be a requisite for adaptability [90].

Variability in step characteristics like step length, swing time, stride time and stance time is consistently reported to increase during impaired gait [96, 98, 103, 106, 112]. However, there is no clear consensus on the direction of alteration in step width variability and studies report both increase [91, 112] and decrease in step width variability [96, 98, 113] in populations with altered gait patterns. For example, while results of the study in healthy elderly population reported that step width variability increased [95], Brach et al. (2001) and Maki et al. (1997) reported that older adults with a history of falls show decreased step width variability [96, 98]. Brach et al. (2005) also showed that there is an optimal variability in step width that might be required and that either too little or too much variability might be related to falls risk [104]. One of the reasons for the inconsistency in the literature regarding step width variability is due to the differences in the way step width is defined across studies, population groups tested and the testing environment [114] [96].

Relevance of Reviewed Literature to Study Two

While alterations in step characteristics during walking are frequently reported (as shorter step lengths, spatiotemporal asymmetries), characterization of gait variability may provide quantifiable measures to evaluate additional aspects of impaired performance (like dynamic balance and risk for falls) post-stroke. Further, in the stroke population, it is unclear whether there would be an asymmetry in step variability and how this might relate to walking performance. With the current suggestion in literature regarding the association between step variability and walking impairments, it seems that characterization of variability in step characteristics will provide insights into motor and balance control mechanisms in a stroke population. Further, investigation of the relationship between stepping asymmetry and step variability would help determine those persons with asymmetry showing specific performance deficits (as evaluated by their step variability). Therefore, study two of this dissertation characterized the step-by-step variability in ST characteristics and explained its relation to hemiparetic walking performance.

Part 4. Foot Placement in a Body Reference Frame

One of the essential tasks during gait is appropriate positioning of the foot relative to the body [115]. Especially, placement of the foot at the end of swing phase serves to establish a stable base of support such that the body can progress forward efficiently during walking. Therefore, foot placement is closely related to trunk/upper body movements and vice versa. Biomechanical models of trunk movements and foot positions/placements during walking have been earlier presented [114, 116-119]. However, assumptions of these models are validated mainly in healthy young subjects and not in neurologically impaired populations. Investigation of foot placement in a body reference frame (i.e., relative to the body) in a hemiparetic population can directly assist in examination of parameters underlying generation of a step.

Defining Foot Placement Relative to Body

Note that spatiotemporal characteristics of steps that were discussed earlier in this literature review specifically refer to kinematics of one foot as defined relative to the other foot (e.g., the spatiotemporal parameter of step length is defined as anterior distance from the leading foot to the trailing foot). Specifically, 'Foot placement' refers to the position of the foot in a step relative to the body. For example, anterior foot position at heel strike relative to the body indicates the instance at which foot was placed anterior to the body in a step.

Control of Foot during Gait

While basic limb movements are primarily determined by central pattern generators [79], animal studies suggest that movements of the foot during gait are further fine-tuned and regulated by cortical control [120, 121]. The cortical influences on foot during walking contribute to the adaptability of the gait pattern [122, 123]. It is widely accepted that the coordination of multiple degrees of freedom involved in locomotion is constrained by the central nervous system through a small number of behavioral units [79, 124-126]. In human walking, studies also indicate that while control of foot is implemented by ankle, knee and hip rotations, the dynamics of the foot are centrally coded to generate the coordinated movements of stance and swing phase [127, 128]. David Winter proposed that foot kinematics is a precisely controlled sensorimotor task and is under the multisegmental motor control of both stance and swing phase [127, 128]. Control of the foot during gait can also be understood within the premise of the motor equivalence theory, where a given invariant task goal (as foot clearance or foot trajectory) can be achieved through variable means [129, 130].

Body Center of Mass Position and Velocity: Postulated Link to Foot Placement

Approximately two-thirds of our body mass (head, arm, trunk), which dominates the calculation of the center of mass position, is precariously balanced over the two legs. Such a

postural state imposes critical demands on the balance control system [131]. Unlike upright weight-bearing postures where balance is maintained when the vertical projection of body center of mass falls (COM) within the base of support (BOS), stability during locomotion is challenged because both BOS and COM are in motion [132, 133]. COM is within the BOS only during the two double support phases, which constitutes only 20% of a stride [132, 133]. During walking, COM is controlled by support forces generated from the legs [132, 133]. Direction and point of application of support forces provided from the ground acts at the centers of pressure on the foot during the stride cycle. Foot placement at the end of each swing phase provides the primary method of moving the COP in both sagittal and frontal plane [132, 133]. Thus, one of the essential functions of foot position relative to body is to maintain the position of COM with respect to the BOS such that dynamic balance during walking is maintained [134].

COM velocity during walking: Townsend proposed, through simulation analyses, that stable gaits can be defined by foot placements which are a linear function of the position and velocity of body center of mass at the time of foot placement [117]. Especially during walking, the COM velocity, in addition to COM displacement, needs to be considered. Pai and group demonstrated that balance may be impossible if COM velocity is directed outward, even if the COM is above the BOS [135, 136]. As a refinement of this rule, Hof et al. (2007) recently showed that the COM position plus its velocity (extrapolated COM: xCOM) should be within the base of support [137]. They also suggested a measure of stability the 'margin of stability', which is the minimum distance from the xCOM to BOS [137].

Foot Placement Relative to Body and its Relation to Walking Sub-tasks

Appropriate foot kinematics relative to body is essential to maintain functional sub-tasks during gait. In the stance phase, body center of mass is propelled within limits of the foot placed (to avoid falling). In the swing phase, trajectory of the foot needs to be appropriately controlled

such that foot positioning at the end of the swing phase is appropriately timed and placed. Foot placement relative to body is also related to minimization of energy, conservation of forward momentum or compensation for some musculoskeletal deficit [118]. Therefore, given the importance of appropriate kinematics of the foot to maintain smooth and efficient locomotion, it seems that investigation of foot kinematics during hemiparetic walking will serve as a tool to investigate the unique impairments in this population.

Balance during gait, involves controlling movement of the whole body COM relative to the BOS (often defined as the area enclosed between the foot placements) [132]. Thus, as explained earlier, one of the essential roles of foot placement during gait is believed to be in determining a new base of support at each step and thereby, maintaining the dynamic balance during walking. Foot placements relative to the body and its relationship to the maintenance of walking balance have earlier been explained in healthy gait [116-119].

Redfern and Schuman in 1994 postulated that foot placement requires symmetry of the limbs with respect to the pelvis at heel contact such that the body center of mass is placed equidistant from both feet during double support, creating a stable support base during the transition to the next step [118]. Mackinnon et al. (1993) and Townsend MA (1981) showed that the most important factor affecting frontal whole body balance is the mediolateral foot placement relative to the center of mass established at initial contact [116, 134]. Therefore, these studies suggest that foot placement relative to body is closely related to dynamic balance during gait.

Relevance of Reviewed Literature to Study Three

Post-stroke, quantifying where the foot is placed relative to body could provide a deeper understanding of the mechanisms of hemiparetic walking than is possible when foot kinematics alone are known (as when it is defined relative to other foot). For example, in persons who take asymmetrical step lengths (relatively longer or shorter paretic step lengths), it is unclear whether

their foot placements relative to pelvis (or trunk) would also be asymmetrical. Further, since the above literature review suggested that foot placement relative to body is also related to motor and dynamic balance control mechanisms during walking, we expected that investigation of foot placement relative to body would provide insights into motor control impairments during hemiparetic walking. Therefore, study three of this dissertation quantified asymmetrical stepping post-stroke in a body reference frame and explained its relationship to hemiparetic walking performance measures.

Part 5. Pre-Swing and Swing Phase during Hemiparetic Gait

During walking, pre-swing of gait precedes the generation of a step and swing phase occurs as the leg is stepping. Therefore, it is likely that several parameters (muscle activity, kinetics and kinematics) that determine the pre-swing and swing phase of walking directly affect where the foot is placed in the step. Consequently, investigation of mechanisms underlying the generation of the pre-swing and swing phase during hemiparetic walking are likely to enable the evaluation of the underlying causes of generation of stepping.

Introduction to Pre-Swing and Swing Phase during Gait

In healthy gait, pre-swing phase occupies 10% of the gait cycle and is more commonly referred to as the second double support [67]. Swing phase occupies 40% of the gait cycle and begins as the foot takes-off from the ground and ends when the foot strikes the ground again [20, 67]. Essential function of the pre-swing phase is to propel the trunk forward in preparation of leg swing initiation [138, 139]. Essential functions of the swing phase include limb clearance from the floor, advancement and forward progression of the leg and positioning the foot at the end of swing phase in preparation for stance phase weight-bearing [20, 67].

Swinging motion of the leg is often likened to the unforced swinging of a compound pendulum, suggesting that swing phase is a rather passive phenomenon [140, 141]. Furthermore,

activity in the leg muscles during swing is low [142, 143] and joint torques in the hip, knee and ankle are also small in the swing phase relative to the stance of walking [144]. Nevertheless, while joint torques are small in the swing phase they cannot be disregarded [145-147]. Furthermore, muscle activity is consistently reported during the swing phase of walking suggesting the active constraints during swing [139, 148, 149]. Most importantly, precise trajectory of the swinging limb and adequate clearance of the limb need to be planned and optimized for efficient locomotion [128, 150]. It is also suggested that the swing phase of walking is under fine regulation by the higher cortical centers in the central nervous system (that are mediated by spinal and interneuronal networks) [151].

Kinematic Characteristics during Pre-Swing and Swing Phase

Pelvis excursions: Increased pelvic hiking during swing [152] to clear the paretic foot and large lateral pelvis displacements [153] related to impaired side-to-side balance [154] are most commonly reported in hemiparetic gait. Dequervain et al. (1996) also reported that the pelvis was retracted at terminal swing in eleven of the 12 participants with slow gait speed and posteriorly tilted throughout swing [70].

Hip excursions: Decreased pre-swing hip extension (bilaterally), decreased paretic hip flexion (attributed primarily to slower speed) in mid-swing and at terminal swing have been consistently observed in hemiparetic gait [16, 70]. Dequervain et al. (1996) also reported that there was a delay in initiation of paretic hip flexion at toe-off and progression of paretic hip flexion during swing, especially in those persons walking at extremely slow walking speeds [70]. Paretic hip circumduction to clear the foot is also commonly reported in persons with stroke [18]. Paretic hip is also shown to be excessively abducted and externally rotated during the swing phase [152].

Knee excursions: Knee flexion at pre-swing and during swing phase is crucial for toe clearance, which is the primary function of swing phase during gait [155]. In hemiparetic gait, reduced paretic knee flexion during swing [16, 156, 157] is most commonly observed. Reduced paretic knee flexion at pre-swing [69] and slow progression of paretic knee flexion during the swing phase [70] have also been observed.

Ankle excursions: In hemiparetic gait, paretic ankle plantarflexion during pre-swing and ankle dorsiflexion during swing [16, 70, 152, 156] are substantially reduced. Further, Kim et al. (2004) reported that the paretic ankle remains in a relatively plantarflexed position even during terminal swing [18]. Paretic ankle plantarflexion at toe-off is reported to be reduced, especially, in those persons with stroke walking at slower walking speeds [70].

Kinetic Characteristics during Pre-Swing and Swing phase

Moments, Powers, Angular velocities

Even in healthy gait, moments, joint torques and powers are substantially reduced in the swing phase compared to the stance. Therefore, most studies on healthy and hemiparetic gait report either peaks or averages in these kinetic characteristics. This limits the understanding of kinetic characteristics specifically in the swing phase of hemiparetic gait. There is general consensus that most moments and power bursts are reduced in amplitude throughout the gait cycle in persons with stroke compared to both age-matched [157] and speed-matched healthy adults [158, 159]. Further, the reductions are reported to be greater on the paretic side than non-paretic [158, 159].

There is much variation in the kinetic (see appendix for definition of kinetics) profiles of persons with stroke [18]. In general, different kinetic strategies can be used to achieve similar kinematic outcomes, increasing the challenge in the quantification of sub-groups that might have specific strategies within the stroke population [160].

Hip, knee, ankle power bursts during swing: In healthy gait, power bursts in the swing phase involve the A2 ankle burst (by concentric plantarflexor activity) during pre-swing and early swing, K3 knee burst (by concentric extensor activity) during pre-swing and early swing, K4 knee burst (by eccentric flexor activity) and H3 hip burst (by concentric flexor activity) occurring in pre-swing and early swing [94, 131, 161]. See Figure 2-6 for explanation of these power profiles. In hemiparetic gait, it is observed that A2 burst is substantially reduced on the paretic side in slow walkers [5]. Whereas, for those who walk fast, K3 burst is reported to be even greater than normal on the non-paretic side [5]. Similar to the A2 burst, H3 burst is reported to be considerably reduced on the paretic side [5]. Therefore, the amplitude of these kinetic variables seems to be positively scaled to the gait speed or functional capacity of persons with stroke [158, 159, 162, 163].

Inter-compensations between hip and ankle powers: Slow walkers especially present with a marked reduction in pre-swing ankle push-off (A2) and early swing hip pull-off (H3) [159] on both paretic and non-paretic sides. Contrarily, persons walking at faster walking speeds or higher functional capacities show less reduction in these power bursts and even present with larger positive work by bilateral hip extensors in early stance (H1) and by early swing paretic hip flexors (H3) [158, 159]. The greater magnitude of hip bursts (H1 and H3) can assist in propulsion, compensating for the reduction in ankle push-off (A2) in persons with stroke who walk fast [158, 159]. Similarly, larger K3 burst is reported in the fast walkers [158, 159]. Thus, several kinetic asymmetries are accentuated between limbs in the fast walkers, suggesting that locomotor capacity is not necessarily recovered through ‘normalization’ of kinetic profiles but may be directly related to compensatory mechanisms [53]. In the literature, this result is usually interpreted to mean that symmetry should not be a primary focus for rehabilitation [5, 18, 53].

However, investigation and differentiation of impairment from compensation is essential to retrain function maximally. Identification of impairment, on the other hand, will assist in normalization of some of the underlying kinetics (e.g., improving the paretic ankle plantarflexor force generation to improve paretic ankle power burst).

Angular velocity of knee in pre-swing determines knee flexion during swing: With regards to angular velocity, dynamic simulations of swing phase in healthy gait performed in the absence of muscle joint torques approximated normal knee kinematics by selecting the initial angular velocities and positions alone [140, 164]. This suggests that the initial angular velocity is an important determinant of normal knee kinematics during swing. Further, Piazza and Delp (1996) found that the amount of knee flexion achieved during swing is decreased by either increasing hip flexion velocity or decreasing knee flexion velocity at pre-swing [145]. In hemiparetic gait, Chen et al. (2005) showed that persons with stroke showed impaired paretic swing initiation that they characterized as inadequate paretic limb propulsion in pre-swing, reduced paretic knee flexion at toe-off and mid-swing [69].

Interjoint coordination during swing phase

Shemmell and associates (2007) recently showed that dynamic torques generated across hip, knee and ankle are tightly coupled during swing phase of normal gait and that a single kinetic time series can describe the pattern of torque production at each joint during this phase [165]. They proposed that such a flexible inter-joint coupling might serve to simplify the control of the swing phase by the CNS. Inter-joint coordination during swing is also revealed indirectly through studies, which report that an increase in hip flexor moment during swing also increases knee flexion [166]. Some inter-joint coordination is also expected given that bi-articular muscles that span two joints are physiologically capable of affecting segments further distally. Further,

the complex inter-segmental dynamics of the body can also explain the inter-joint coordination of kinetics in the swing phase [167, 168].

Muscle Activity during Pre-Swing and Swing Phase

Pre-swing and swing phase muscle activity in healthy gait: Although low, muscle activity is consistently reported in swing phase of healthy gait. In general, hip flexors have shown to dominate swing phase [169-171]. Specifically, Nene et al. (1999) and Neptune et al. (2001) show that rectus femoris (RF) is responsible for swing initiation [139, 148]. Furthermore, medial gastrocnemius (MG) is also suggested to aid in swing initiation [148]. On the other hand, Gotschall and Kram (2005) based on their novel methodology of application of external forces during treadmill walking, showed that while both iliopsoas (IP) and RF initiate and propagate swing, ankle extensors do not directly contribute to swing initiation [172]. Further, indirect evidence of specific contributions of muscular activity in swing phase is revealed in the study by Goldberg et al. (2003) where they evaluate muscles that influence knee flexion velocity in the pre-swing phase (knee flexion velocity directly influences knee kinematics during swing) [173]. Goldberg et al. (2003) in their study conclude that while MG and IP directly contribute to swing by increasing the knee flexion velocity in pre-swing, vastii, RF and soleus decrease knee flexion velocity thereby reducing knee flexion in swing [173]. Further, Winter DA (1992) suggested that Tibialis anterior (ankle dorsiflexor) were specifically required during mid-swing for limb clearance, whereas biarticular hamstrings (long head of biceps femoris) decelerates the limb in terminal swing to prepare it for weight acceptance [128].

Pre-swing and swing phase muscle activity in hemiparetic gait: There are much fewer reports on muscle activity in pre-swing and swing phase of hemiparetic gait. In general, large interindividual variability is observed in the EMG patterns that characterize muscle activity in individual patients. Den Otter et al. (2007) reports that paretic TA activity significantly increases

in the swing phase of persons with stroke compared to healthy persons [174]. Further, since EMG activity can be observed as premature activation, abnormal coactivation or as compensatory or adaptive coactivation, it is difficult to suggest whether observed muscle activity is a sign of impaired motor control or adaptive behavior to promote functional walking. Nevertheless, association of EMG activity to walking performance might provide insights into the role of observed activity.

Relevance of Reviewed Literature to Study Four

Overall, the above literature review suggests that several studies have reported kinematic, kinetic characteristics and muscle activity during pre-swing and swing of hemiparetic gait. Nevertheless, a holistic picture of control strategies in hemiparetic pre-swing and swing phase are lacking, primarily because pre-swing and swing phase dynamics have not been correlated with walking performance after hemiparesis. It is likely that events during the pre-swing and swing phase determine where the foot is placed in the step. Thus, patterns observed in these phases during hemiparetic gait might explain the underlying reasons for the variability in stepping asymmetry. Therefore, in study four of this dissertation specific pre-swing and swing phase variables were used to explain step length variability. Specifically, selected kinematic and kinetic variables corresponding to initial conditions of the leg, swing phase and contralateral stance phase (occurring at the same instance as swing phase) explained step length generation during hemiparetic gait.

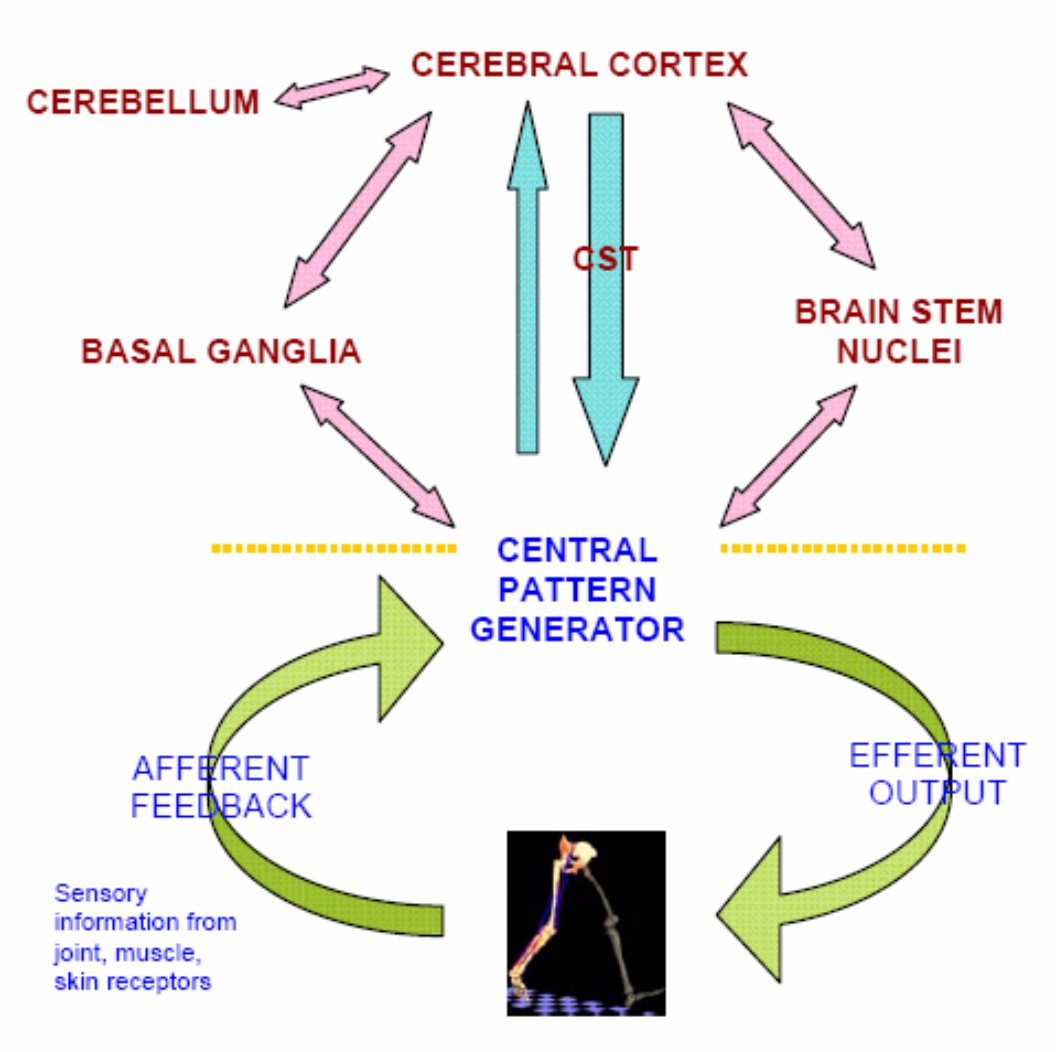


Figure 2-1. A simplified model demonstrating neural control of gait

The central pattern generators in the spinal cord generates the locomotor rhythm that is modified by afferent information from the higher centers and the periphery. Abbreviations: CST – Corticospinal Tract.

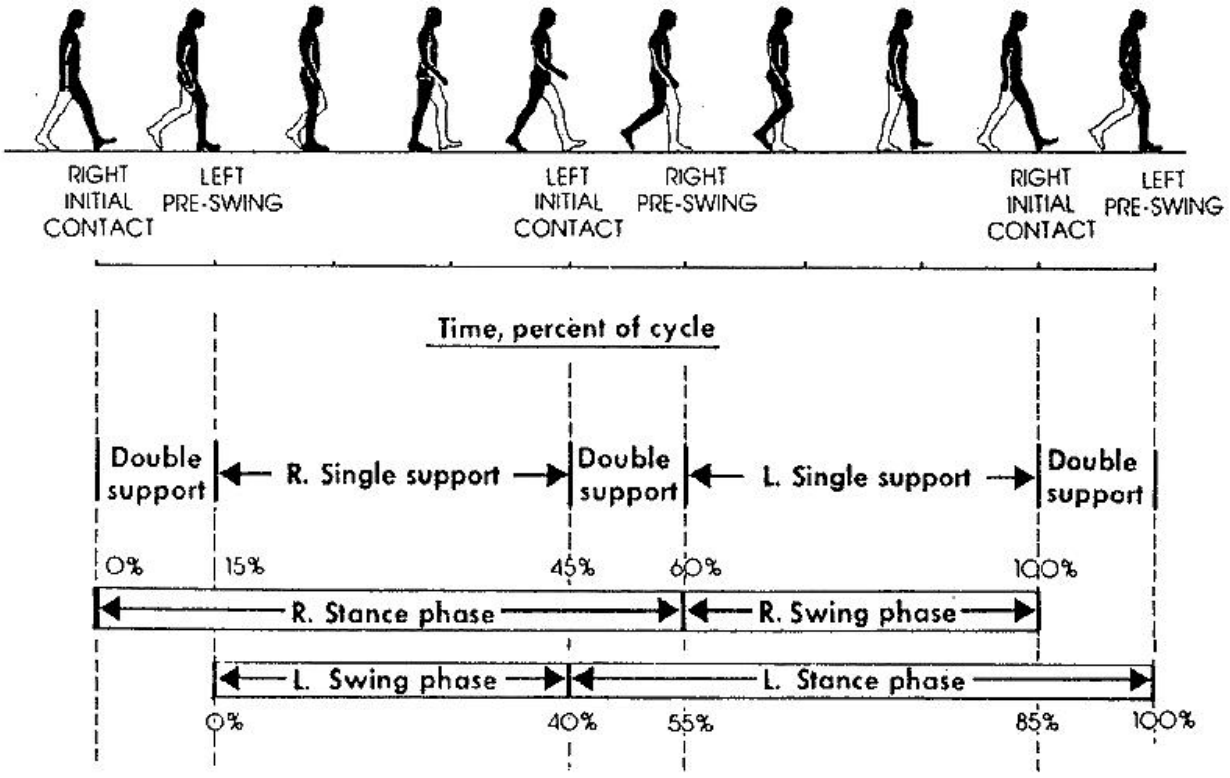


Figure 2-2.Division of a gait cycle into phases

The gait cycle is divided into stance (0 – 60%) and swing (60-100%) phase. The stance phase can be further sub-divided into two double support phases (10% each) and a single support phase (40%). Source: http://www.vard.org/mono/gait/gaitcov.htm

Figure 2-3.Methodology for collection of spatiotemporal characteristics during walking using an instrumented mat (GAITRite)

GAITRite is an electronic walkway with sensors embedded within it that record the spatial and temporal characteristics of steps as a person walks over it. A sample walk that was recorded by GAITRite is presented in this Figure. Note the parameters generated by the software.

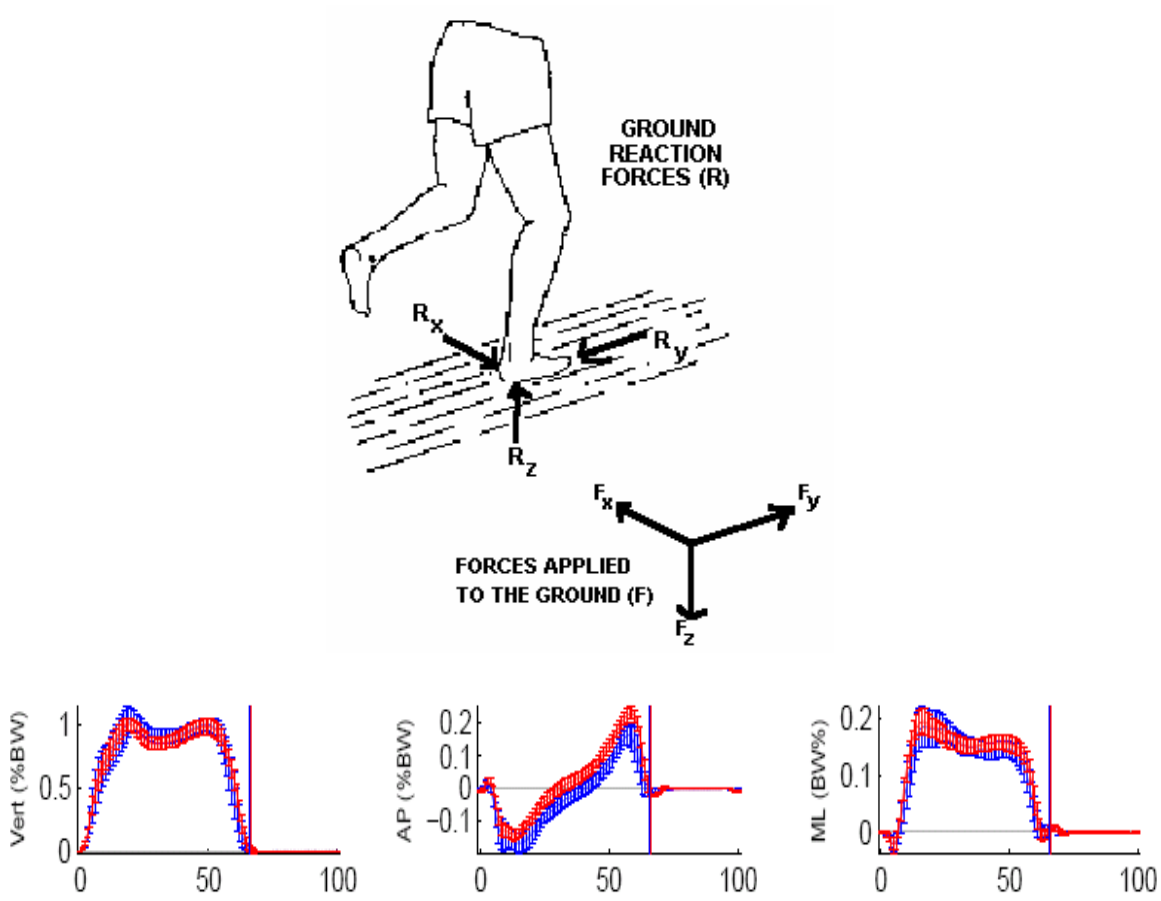


Figure 2-4. Illustration of ground reaction forces exerted by the limbs and typical force curves

This Figure illustrates the 3-D forces applied to the ground from the legs (Fx, Fy, Fz) and the measured Ground reaction forces (Rx, Ry, Rz). The plots on the bottom demonstrate these GRF curves from the right (red) and left (blue) legs normalized to the body weight of the person for the three force components [Vertical GRF (Rz), AP - Anterior-posterior GRF (Ry), ML- Medial-Lateral GRF (Rx)].

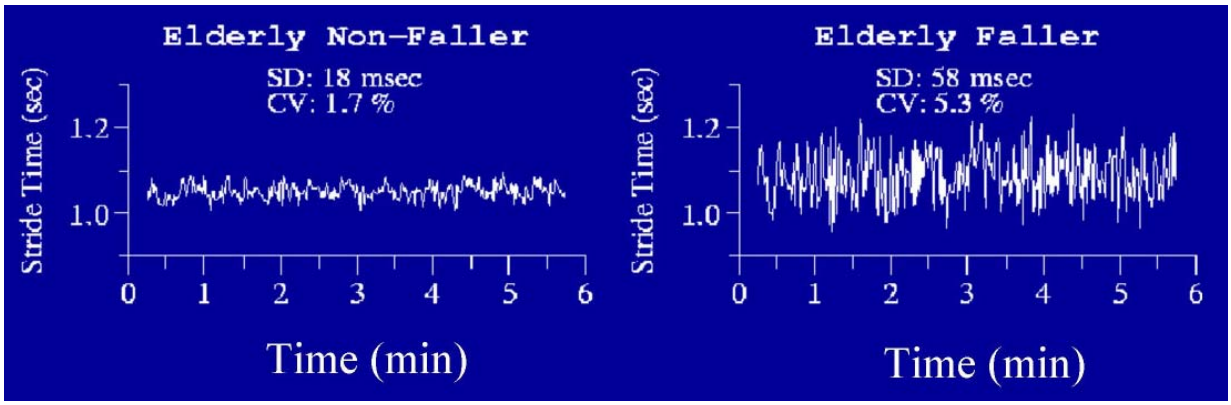


Figure 2-5. Variability (in stride time) during walking and its relation to risk for falls

Source: Hausdorff, J. M. (2005), Gait variability: methods, modeling and meaning. *J Neuroengineering Rehabil, 2*, 19

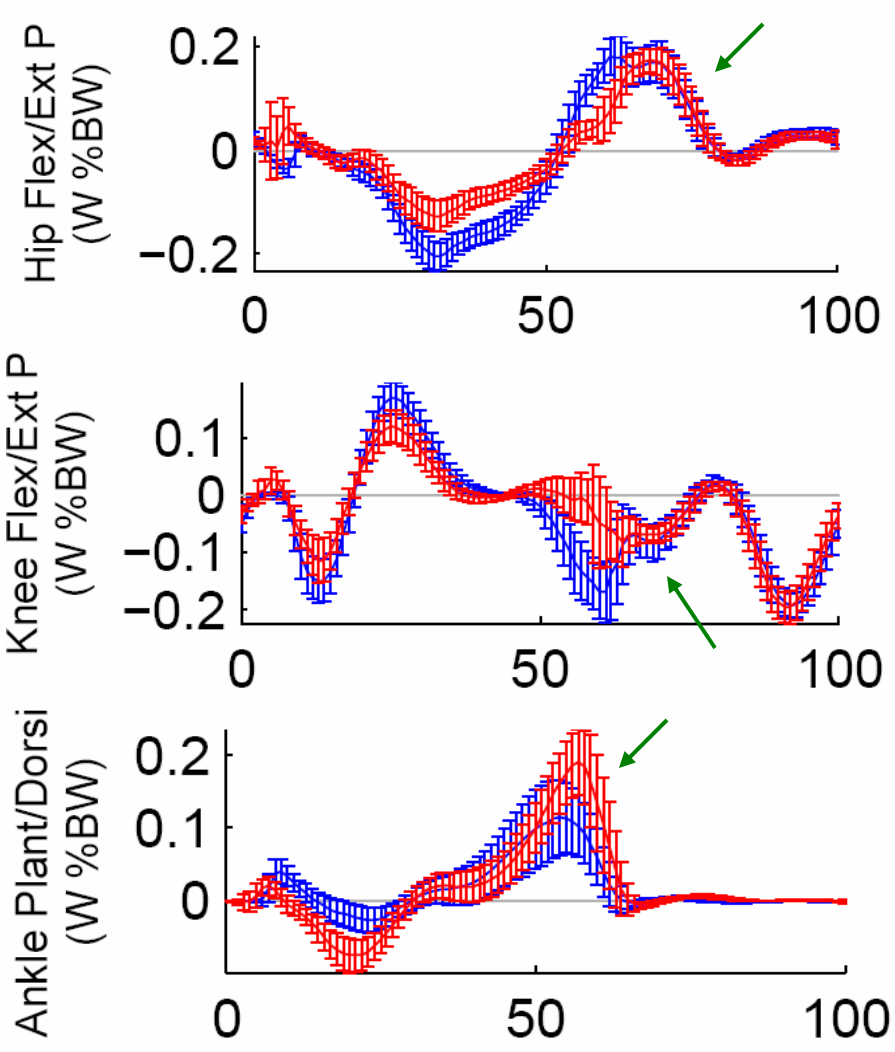


Figure 2-6. Power profiles in swing phase of a healthy gait cycle

The swing phase begins at approximately 60% of the gait cycle. The arrows show the power bursts in hip (H3), knee (K3) and ankle (A2) during swing phase of healthy gait. Red is right leg and blue is left leg.

CHAPTER 3
RELATIONSHIP BETWEEN STEP LENGTH ASYMMETRY AND WALKING PERFORMANCE IN SUBJECTS WITH CHRONIC HEMIPARESIS

Introduction

The asymmetrical nature of hemiparetic walking is well documented in persons who have sustained a stroke, [12, 13, 175] with the asymmetries in spatiotemporal, kinematic and kinetic parameters of walking related to disturbances in motor coordination [5]. Specifically, asymmetry in spatiotemporal parameters have been commonly used in the clinic to examine the walking patterns in patients who have experienced a stroke [19]. Previous studies [13, 176] have reported that temporal (swing time) asymmetry is a significant predictor of hemiparetic walking performance since it strongly correlates with stages of motor recovery, walking speed and falls. However, the relationship between spatial (step length) asymmetry and hemiparetic walking performance is unclear.

It has earlier been reported that, after a stroke, patients may walk with either relatively longer paretic steps or longer non-paretic steps [19, 65, 71]. Therefore, consistent patterns of step length asymmetry have not been observed. Moreover, the reasons for the variability in step length asymmetry patterns and the relation of the variable patterns with walking performance have not yet been explained. For instance, in the study by Kim et al. (2003), considerable variability in step length asymmetry was observed in a sample of 28 chronic stroke survivors [19]. While 14 of these subjects walked with longer paretic steps than non-paretic, 14 others walked with relatively longer non-paretic steps. Consequently, they hypothesized that the variability in the patterns of step length asymmetry may be due to compensatory strategies that increase or decrease the step length of either the paretic or non-paretic leg. However, they were unable to advance the discussion since they found a non-significant relationship between step length symmetry and symmetry in vertical ground reaction forces. Further, other studies indicate

that; on an average, individuals who have sustained a stroke walk with relatively longer paretic steps [65, 71]. Therefore, neither consistent patterns of step length asymmetry have been observed nor have the asymmetrical patterns been characterized. Further, the relationship between step length asymmetry and walking speed is not well documented in the current literature. This may be another reason for insufficient understanding of the affect of step length asymmetry on hemiparetic walking performance. For instance, shorter stride lengths (bilaterally) have been related to slower walking speed and thereby poor walking performance [70], and yet a non-significant relationship has been suggested between step length asymmetry and walking speed [19]. Overall, current evidence suggests that persons after a stroke may walk with different patterns of step length asymmetry that may be unrelated to the attained walking speed. However, it is not clear how the different asymmetrical patterns relate to post-stroke hemiparetic walking performance and why asymmetry in step lengths may not necessarily limit the attained walking speed.

We propose that correlating the asymmetry observed in step lengths with propulsive ground reaction forces (GRFs) during walking can provide a basis to evaluate different asymmetrical patterns and their effect on hemiparetic walking performance. Propulsive GRFs represent the net forces generated by the legs to accelerate (propel) the body's center-of-mass forward. The propulsive GRFs quantify forward propulsion during walking, which is an essential requirement of locomotion along with body support [27]. In our recent work, we have shown that the paretic leg propulsive GRFs can provide a quantitative measure of the coordinated output of the paretic leg during hemiparetic walking [76]. Specifically, we found that the percentage of propulsive forces generated by the paretic leg ($Propulsion_{Paretic}$) can quantify the contribution of the paretic leg to the coordinated task of forward propulsion during walking. In our present

study, we hypothesized that the Propulsion $_{Paretic}$ would correlate with asymmetry observed in step lengths since generation of a step requires forces exerted by the legs to propel the body's center-of-mass. Further, we suggest that correlating the asymmetry observed in step lengths with propulsive GRFs during walking can help understand the relationship between spatial asymmetry and walking performance after a stroke.

Therefore, the primary purpose of our study was to explore the correlation between step length asymmetry and propulsive forces in an attempt to quantify the affect of spatial asymmetry on post-stroke hemiparetic walking performance. Further, we investigated how asymmetrical step lengths may be related to hemiparetic severity (as rated by Brunnstrom staging), walking speed, vertical GRFs and other spatiotemporal walking parameters (walking speed, swing time, pre-swing time and ability to change speed) to gain a holistic understanding of the relationship between asymmetrical step lengths and hemiparetic walking performance.

Methods

Participants

We recruited 49 subjects with chronic hemiparesis [42 men, 7 women; ages = 62.7 ± 10.2 (SD) years; time since stroke = 4.25 ± 3.67 (SD) years; affected side – left = 25, right = 24] were recruited at the Palo Alto Department of Veterans Affairs Medical Center. The data presented in this study were collected as part of a larger study that investigated the links between gait characteristics and bone density in chronic stroke survivors [177]. Inclusion criteria were: at least 6-12 months post stroke, unilateral weakness, and the ability to walk 10 meters in 50 seconds or less without manual assistance. Subjects were allowed to use their assistive devices (AFO and/or Cane) during the testing. Subjects were excluded from the study if they had any orthopedic or neurological conditions in addition to the stroke, had more than one CVA incident

and were unable to provide informed consent. Brunnstrom motor recovery stages were used to determine the severity of hemiparesis for the subjects [178]. Subjects varied in their ability to perform voluntary movements within and outside of flexor and extensor synergy patterns (as assessed clinically by Brunnstrom staging). Based on their Brunnstrom stage, three groups of subjects with differing hemiparetic severity were identified. Subjects in the 'Severe hemiparesis' group (Brunnstrom stage 3, n=19) were limited to movements within the synergy patterns (e.g., only basic limb flexion or extension synergies can be performed voluntarily). Subjects in the 'Moderate hemiparesis' group (Brunnstrom stages 4-5, n=20) were able to produce some movement combinations outside of the synergy patterns. Subjects in the 'Mild hemiparesis' group (Brunnstrom stage 6, n=10) were able to produce both isolated joint movements and movements in synergy patterns. All participants in the study signed a written informed consent and the Stanford Administrative Panel on Human Subjects in Medical Research approved the protocol.

Procedures

The subjects walked separately over the GAITRite and force platforms at their self-selected and fastest safe speeds to collect the spatiotemporal parameters and GRFs, respectively. GAITRite (CIR Systems, Inc) is a portable instrumented electronic walkway 4.3 meter long and is a valid and reliable system for measuring spatiotemporal parameters [179]. Force platforms (Advanced Medical Technology, Inc) embedded across a 10 m walkway were used to collect three-dimensional GRFs during the gait cycle. Before testing, clear explanations were provided to the subjects regarding the importance of walking in their natural manner during the testing and avoidance of targeting the force plates. During GAITRite data collection, the GAITRite was placed over the force plates. However, the force plate data was not collected while subjects were walking on the GAITRite. Subjects started walking 5-6 steps before the GAITRite and stopped

5-6 steps after passing the GAITRite to get constant speed data over the mat and avoid the effects of acceleration and deceleration. Subjects walked a total distance of 10 meters over the GAITRite. Three good walking trials, each at self-selected and fastest safe speeds, were collected over the GAITRite. Some subjects were asked to walk 1 or 2 more trials due to problems like tripping during walking. The number of trials collected for the force plate data were variable and depended on whether both or at least one leg were determined by visual inspection to have had adequate contact on the force platforms. GRF data were acquired at 200 Hz and the horizontal and vertical forces (normalized to the individual's body weight) were used for analyses. A therapist provided close supervision during the walking trials. Subjects were allowed to take rests between trials if they needed to. Walking speed was calculated by the GAITRite and no estimates of speed were used from the force plate trials.

Data Analyses

GAITRite data: We analyzed all the collected trials (3 for each speed). The data from individual trials were averaged together to determine the spatiotemporal variables for each participant. Spatiotemporal variables included in this study were self-selected and fastest walking speed, step lengths, swing times and pre-swing times (time spent in double support phase of gait cycle). Paretic and Non-paretic step length, swing time and pre-swing time data were averaged only from the trials of self-selected walking speed. Subjects' self-selected walking speed were categorized as: speed < 0.4 m/s (household ambulatory); 0.4 – 0.8 m/s (limited community); and speed > 0.8 m/s (community ambulatory) [180]. Step length asymmetry was quantified using a step length ratio (SLR), which was defined as the Paretic step length [181] divided by the Non-paretic step length [181]. The fastest safe walking speed data were utilized to calculate the percentage change (from self-selected to fastest safe speeds) in walking speed, cadence, paretic step lengths, and non-paretic step lengths.

Force plate data: We collected a minimum of 4 trials and a maximum of 15 trials to assure adequate contact on the force platforms to determine the ground reaction force patterns for each participant. The GRF values that were analyzed for individual participants were variable and depended on the number of trials with good foot contacts on the force platforms. Good foot contacts were determined if both legs made contact with the force platforms in entirety. When possible, multiple good foot contacts were averaged to generate ground reaction force values, but in one participant, only one trial could be analyzed.

Raw anterior - posterior and vertical GRF data were normalized to body weight and processed using a custom Matlab program. Note that anteriorly directed forces are propulsive (positive) and posteriorly directed forces are braking (negative) (Figure 3-1). The impulse for each leg was calculated as the time integral of the GRF (the area under the GRF curve) :

$$Ixl = \int GRFxdt \quad \text{(Equation 1)}$$

where I = Impulse, force component x= v (vertical), p(propulsive) or b (braking) and leg l = p (paretic) or n (non-paretic). Paretic propulsion (Pp) was then calculated from the propulsive impulse:

$$PP\ (\%) = Ipp / (Ipp + Ipn) * 100$$

Statistical Analyses

A paired sample t-test was used to test whether the differences between the paretic and non-paretic step length were statistically significant. Relationships between the step length asymmetry (SLR), GRFs and walking speed were characterized using Pearson's correlation coefficient (r) and that between SLR and hemiparetic severity using the non-parametric Spearman's correlation coefficient (ρ). For other walking variables included in the study, descriptive analyses were conducted to understand their relationship with the asymmetrical patterns. All statistical analyses were performed with SPSS 12.0 software.

Results

The walking variables were collected at the self-selected walking speeds for all subjects and at the fastest safe speeds for 46 subjects. Data were not collected for 3 subjects at their fastest speeds due to safety concerns. Nineteen of the 49 subjects used a mobility aid [i.e., a cane or an ankle foot orthosis or both] to ambulate. Paired sample t-tests revealed that the paretic step lengths were significantly different from the non-paretic step lengths ($p = .0001$). Therefore, step length asymmetry was characterized and three patterns of step length asymmetry were identified. The symmetrical group were defined as subjects with SLR between 0.9 to 1.1 [$SLR = 1 \pm 0.1$ (10%) SD]. Asymmetrical groups were those with $SLR > 1.1$ (longer paretic steps than non-paretic) and $SLR < 0.9$ (longer non-paretic steps than paretic).

Relationship between Step Length Asymmetry (SLR) and GRFs

Correlation analysis revealed a strong negative correlation between SLR and Pp with Pp explaining 62 % ($r = 0.785$, $p < 0.001$) of variance in step length asymmetry (Table 3-1). Subjects that demonstrated impaired paretic leg propulsion (Pp) and increased non-paretic leg propulsion walked asymmetrically with longer paretic steps than non-paretic [Figure 3-2, top tracing]. In contrast, subjects generating symmetrical GRFs with the two legs walked nearly symmetrically [Figure 3-2, middle tracing]. Subjects generating relatively proportionate or greater Pp walked asymmetrically with longer non-paretic steps than paretic. However, the greater Pp generated by these subjects walking with longer non-paretic steps was lesser in magnitude compared to those walking symmetrically. Refer to Figure 3-2 and compare the Pp between GRF curves of the bottom and middle tracing.

Asymmetrical group with longer paretic step lengths than non-paretic (n = 16): Subjects that generated the least paretic propulsion ($Pp < 20\%$) walked with relatively longer

paretic step lengths. Five of these 16 subjects generated 5% or less Pp and walked with the longest paretic steps relative to the non-paretic leg (SLR > 1.5) (Figure 3-3).

Asymmetrical group with longer non-paretic step lengths than paretic (n = 4): There were only 4 subjects who were classified as asymmetric and walked with relatively longer non-paretic step lengths. Three of these four subjects generated substantial propulsive forces with the paretic leg (Pp > 55%; however one subject generated only 25% Pp).

Symmetrical group (n = 23): Twenty-three subjects walked symmetrically. Seventeen of these 23 subjects generated almost symmetrical propulsive forces (Pp = 35 – 57 %) and the rest generated lesser paretic leg propulsion (Pp = 15 – 30%).

Only 10 of 49 subjects in the study population generated a net braking impulse in the pre-swing (normally propulsive) phase of the gait cycle and these 10 subjects walked at SLR > 1.1, with 5/10 subjects walking at SLR > 1.5. Nine of these 10 subjects used a mobility aid for ambulation and only one subject walked independently. We separately analyzed the data for subjects who used a mobility aid from those who did not to investigate whether the relationship between step length asymmetry and paretic leg propulsion changed by using a mobility aid. Subjects walking with a mobility aid generated less Pp in comparison to those who walked without one. However, the relationship between SLR and Pp was not different for those who did or did not use a mobility aid.

With respect to the vertical GRF, all subjects (irrespective of their step length asymmetry pattern) supported a greater percentage of body weight on the non-paretic leg than the paretic leg during the two double support phases in the gait cycle. However, moderate correlations ($r = -0.447$, $p < .001$) were found only between SLR and percentage of body weight supported on the paretic leg during the pre-swing phase of the paretic leg (Table 3-1).

Relationship between Asymmetrical Step Lengths, Hemiparetic Severity and Walking Speed

While SLR correlated weakly with walking speed ($r = -0.351$, $p < .05$), a stronger correlation existed between SLR and hemiparetic severity ($\rho = -.526$, $p < .001$) (Table 3-1). Eleven out of the 19 subjects with severe hemiparesis walked asymmetrically with relatively longer paretic steps than non-paretic and yet walked at differing walking speeds (Figure 3-4). Note that 4 subjects with severe hemiparesis that walked asymmetrically with longer paretic steps generated much less paretic leg propulsion ($Pp < 25\%$) and yet walked at speeds greater than 0.8 m/s (community ambulatory) [180], (Figure 3-4). In contrast, the 3 subjects (Figure 3-4) with mild hemiparesis (Stage 6) who walked symmetrically or asymmetrically (with longer non-paretic steps) generated much greater paretic leg propulsion ($Pp \geq 45\%$) and yet walked at slower speeds between 0.4 – 0.8 m/s (limited community ambulatory) [180].

Relationship between Step Length Asymmetry, Time Spent in Pre-Swing and Swing Time

Only 8 of 49 subjects spent greater than 20% of cycle time during the paretic pre-swing phase (compared to 10-20% cycle time in their non-paretic pre-swing). These 8 subjects walked asymmetrically with longer paretic steps than non-paretic. With respect to swing time, only 2 of 49 subjects spent greater time swinging their paretic than the non-paretic leg. Subjects walking at SLR > 1.1, on an average, spent the greatest time swinging their paretic leg.

Relationship between SLR, Change in Gait Speed and Parameters That Contribute to Change in Speed

Absolute mean differences were evaluated for these analyses. Subjects walking symmetrically increased paretic and non-paretic step lengths and cadence at faster walking speeds. In contrast, those walking asymmetrically at SLR > 1.1 had little increase in their paretic step length at the faster walking speeds even though they increased their cadence as much as the symmetrical group (Figure 3-5).

Discussion

Relationship between Step Length Asymmetry and Propulsive Forces during Hemiparetic Walking

Step length asymmetry during walking is related to propulsive forces generated by persons with hemiparesis. Our data revealed that subjects generating relatively lesser propulsive forces with the paretic leg walked asymmetrically with longer paretic steps than non-paretic (SLR > 1.1) On the other hand, subjects walking symmetrically (0.9 < SLR < 1.1) generated near symmetrical propulsive forces with the two legs. In Figure 3-2 comparison of P_P between subjects with SLR > 1.1, 0.9 < SLR < 1.1 and SLR < 0.9 indicates that there may be distinct differences in paretic leg propulsion between subjects with different patterns of step length asymmetry.

To our knowledge, there is little direct evidence for the mechanisms that control step length in normal and hemiparetic walking. However, indirect evidence for the control of step length during walking is provided by a few studies. Varraine et al. (2000) suggests two potential controlling mechanisms for intentionally lengthening a stride in healthy individuals: control of trunk progression and control of leg trajectory [182]. In their study, subjects were able to lengthen their stride by generating greater propulsive forces. The increased propulsive forces enabled the trunk to progress further forward and thereby, generate a longer step. Additionally, subjects took a longer step by holding the leg longer in the swing phase. Further, recent studies have highlighted the causal relationships between muscle activity in pre-swing and the resulting swing leg trajectory [148, 183, 184]. These studies indicate that kinematics of the leg in its pre-swing phase affect trajectory of the leg in its swing phase and thereby, magnitude of the step length. This implies that the leg producing greater forward propulsion in pre-swing might take the longer step length. However, the results from our study suggest the opposite. We showed that

persons generating impaired paretic leg propulsion walked with a relatively longer paretic step. These persons generating impaired paretic leg propulsion also generated relatively greater non-paretic propulsion, likely to compensate for the lesser paretic leg propulsion. For example, in Figure 3-2, compare the relative contributions of non-paretic propulsion in the top and middle GRF tracings. These results suggest that a high SLR (i.e., paretic step length > non-paretic step length) is in large part the result of the relatively greater non-paretic leg propulsion. For instance, greater forward propulsion by the non-paretic leg in its stance phase will cause the trunk, including the pelvis to move forward. This forward motion of the pelvis will increase with increased propulsion and can cause the swinging paretic leg to move forward relative to the ground even if it moves little relative to the pelvis. Therefore, greater non-paretic leg propulsion is one mechanism for the longer paretic steps (high SLR). Further, persons with high SLR also spent a longer time swinging their paretic leg than others who walked with symmetrical steps. Therefore, one might hypothesize either/both greater non-paretic leg propulsion or a longer paretic swing phase as potential candidates to explain the mechanisms underlying a relatively longer paretic step length.

The strong relationship between the patterns of step length asymmetry and propulsive force asymmetry might suggest a mechanical relation between step length and propulsive force. For example, if the leg were to be an inverted pendulum, the generation of horizontal forces would be directly related to the position of the foot relative to the body's center-of-mass. Specifically, foot placement anterior to the center-of-mass (as during heel strike) would induce a braking force and foot placement posterior to the center-of-mass (as in pre-swing phase) would induce a propulsive force. In this sense, an asymmetry in the placement of the feet may induce asymmetrical propulsive forces when the foot is placed more anterior to the center-of-mass than

posterior. However, the inability to place the foot further behind the center-of-mass than forwards (as with longer paretic steps than non-paretic) suggests specific impairments underlying the asymmetrical step lengths. For example, the inability to achieve adequate hip extension may limit the propulsive forces exerted during the terminal stance. Furthermore, during the terminal stance of the paretic stride the paretic foot is likely posterior to the body's center-of-mass and yet there is no propulsive force generated (see Figure 3-2, top tracing). Therefore, it is more likely that there may be an active reduction in propulsive force generation. The active reduction in propulsive force generated in the pre-swing may, in turn, suggest impaired uniarticular ankle plantarflexor activity [148]. On the contrary, in the participants walking with relatively shorter paretic steps (SLR < 0.9), the greater paretic propulsive force generation suggests that plantar flexors may be providing reasonable propulsion. Stepping, however, depends not only on the ability of the plantarflexors to propel the body forward but also on the ability of the hip flexors to generate power to the swinging leg [184]. Therefore, having shorter paretic steps relative to non-paretic steps may indicate an inability to advance the paretic leg due to impaired swing initiation by the hip flexors. In addition to direct mechanical effects of foot placement on propulsive force generation, it is likely that the mechanisms underlying asymmetrical steps reflect distinct muscular impairments that determine the observed patterns of step length asymmetry during hemiparetic walking. Future studies targeted at investigating the underlying muscular impairments shall further determine the underlying causes for high SLR. Yet, the strong relationship between SLR and propulsion can be utilized in the clinic as a tool to distinguish persons in their ability to generate propulsive forces. Beyond simply promoting symmetry, SLR can be utilized to develop individual goals that train propulsive force production,

equalize bilateral biomechanical involvement by improving hip extension, or promote paretic step initiation.

Relationship between Step Length Asymmetry, Walking Speed and Hemiparetic Severity

We were also able to investigate the relationship between walking speed, hemiparetic severity and asymmetry in step lengths. A weak relationship between step length asymmetry and walking speed was observed, indicating that asymmetrical patterns need not necessarily limit the attained walking speed. However, hemiparetic severity (as rated by Brunnstrom staging) seemed to predict step length asymmetry since the majority of subjects with severe hemiparesis walked asymmetrically at SLR > 1.1. Furthermore, differences in Pp between the persons with mild and severe hemiparesis were unrelated to the attained walking speed. For example, in Figure 3-4, four subjects with severe hemiparesis had impaired Pp and yet they were walking at speeds > 0.8 m/s. In contrast, three subjects with mild hemiparesis generated greater Pp and yet walked at slower speeds between 0.4-0.8 m/s. The weak relation between Pp and walking speed indicates that other compensatory mechanisms could help some persons to attain a relatively functional walking speed. Quantification of these compensatory mechanisms may be difficult when speed alone is the outcome measure since walking speed is the net outcome of the two legs. However, asymmetrical step lengths might indicate compensation in those persons walking with a high SLR and faster walking speeds compared to those walking with a high SLR and slower walking speeds. This is because subjects with relatively longer paretic steps would have lesser paretic leg propulsion, and if they continue to walk at faster speeds they might accomplish these speeds via compensatory strategies. For example, one of the ways to attain a faster speed (i.e., acceleration of the body's center-of-mass) would be to generate greater propulsive forces with the non-paretic leg that serves to accelerate the center-of-mass forward. Other compensations can also arise from

the paretic leg itself or from the trunk (e.g., forward lean) to attain relatively functional walking speeds despite decreased propulsive force with the paretic leg.

Relationship between Step Length Asymmetry, Paretic Pre-Swing Time and Vertical GRFs

Furthermore, we were able to determine the relationship between step length asymmetry, other spatiotemporal parameters and vertical GRFs. The evidence that impaired paretic leg function prolongs the paretic pre-swing phase supports our finding that persons walking at SLR > 1.1 supported less weight on the paretic leg during the pre-swing phase. This finding thereby increases the need to develop compensatory strategies to overcome these deficits [70]. In particular, note that in our study we allowed participants to walk naturally as they would in the community and 19 persons used some mobility aid for ambulation even during the testing. Although persons walking with a mobility aid generated less Pp in comparison to those who walked without one, the relationship between SLR and PP did not change while analyzing only the data for those subjects who used an aid or those who did not. This corroborated our hypothesis that a high SLR was related to specific problems in propelling the body forward with the paretic leg. However, the high SLR may neither necessarily limit the attained speed nor may substantially limit persons from changing their walking speeds. As revealed in Figure 3-5, compared to the symmetrical group persons with high SLR seemed to primarily increase their speed by increasing cadence with much less increase in the paretic step lengths. This indicates that persons walking with asymmetrical step lengths may utilize different strategies to increase their walking speeds. Further, even though the subjects with SLR > 1.1 walked with relatively longer paretic steps than non-paretic at their self-selected walking speeds, they were unable to change the paretic step length as much as the non-paretic when changing their speed.

Conclusions

We were able to provide some insights into the relationship between asymmetrical step lengths and hemiparetic walking performance. Asymmetry in step lengths strongly relates to the propulsive forces generated by the paretic leg. Greater non-paretic leg propulsion to offset the impaired paretic propulsion is likely one of the mechanisms for the high SLR (paretic step length > non-paretic step length). Despite mechanical relations between foot placement and force generation that are expected, we believe that there are additional muscular impairments underlying the asymmetrical patterns. However, further research is warranted to confirm this. Yet, the strong relationship between SLR and propulsion can be utilized in the clinic as a measure to evaluate the propulsive forces generated by the paretic leg. We were also able to provide a basis to evaluate the different asymmetrical patterns by correlating the asymmetry observed in step lengths with propulsive GRFs during hemiparetic walking. Moreover, the relationship between SLR, speed and hemiparetic severity indicates that SLR, when used along with speed as an outcome measure, can help understand compensatory strategies that some persons (who are asymmetrical and yet walk at faster speeds) use to offset the lesser propulsive force ability of the paretic leg. The relationship between SLR and other spatiotemporal walking parameters further reveals how asymmetrical step lengths may affect hemiparetic walking. In summary, we propose that SLR is a promising tool that rehabilitation therapists might use to further the understanding of hemiparetic walking performance. Clinically, this would enable the identification of walking impairments in hemiparetic individuals and tailor locomotor retraining specifically to address the root causes of impaired ambulation for each individual.

Table 3-1. Correlation between SLR and walking variables

Walking variables	Step length asymmetry (SLR)
P_P (paretic leg propulsion)	-0.785†
Self-selected walking speed	-0.351*
Hemiparetic severity (Brunnstrom stage)	-0.526†
Percentage weight supported during pre-swing phase	
Paretic pre-swing phase	
Weight on paretic leg	-0.447†
Weight on non-paretic leg	-0.291*
Non-paretic pre-swing phase	
Weight on paretic leg	0.072
Weight on non-paretic leg	0.212

* significant at $P < .05$, † significant at $P < .001$

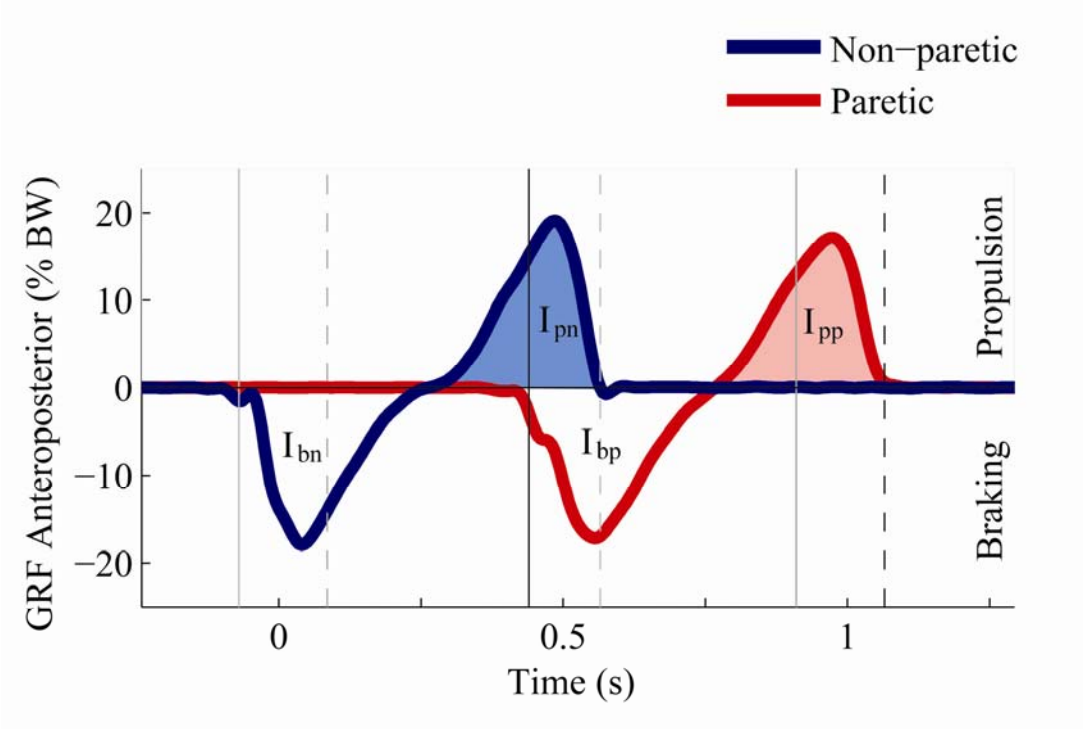
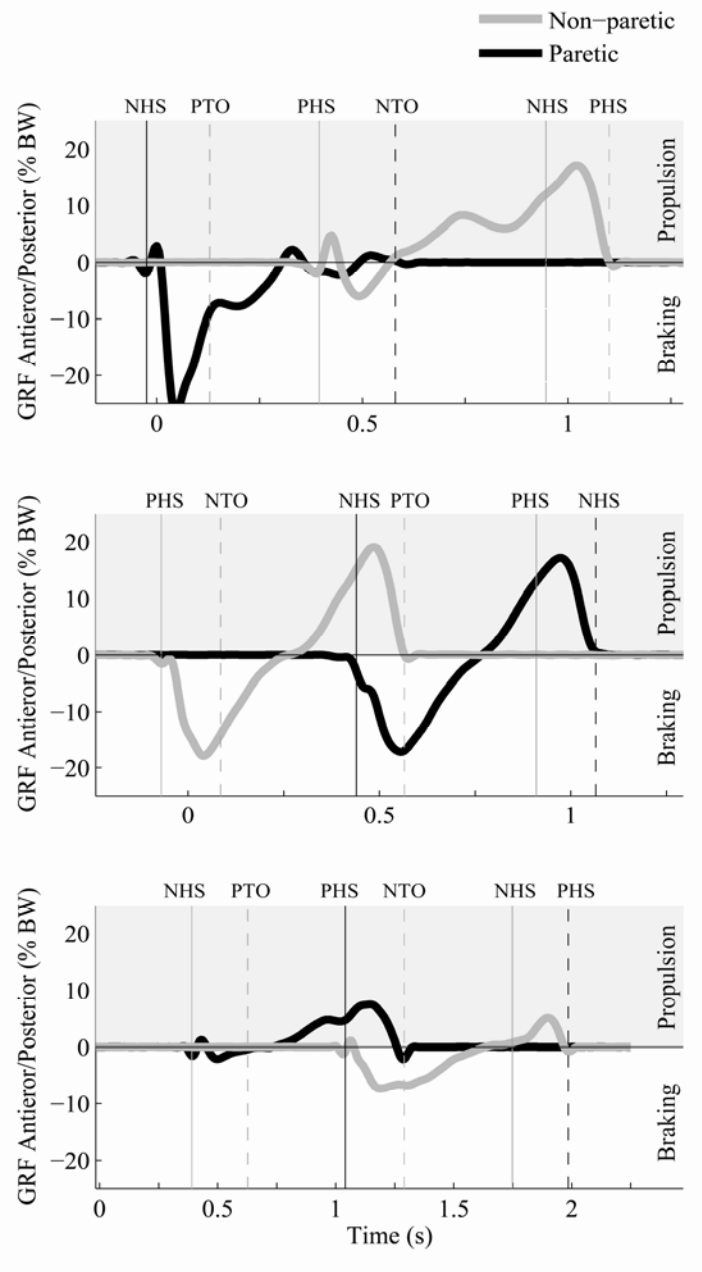


Figure 3-1. Illustration of horizontal GRF impulses

Positive values (shaded area) represent propulsion. Negative values (unshaded area) represent braking. Paretic leg is the bold curve and non-paretic leg is the light curve. Ipp – Propulsive impulse by the paretic leg is the shaded area under the bold curve; Ipn -Propulsive impulse by the non-paretic leg is the shaded area under the light curve; Ibp - Braking impulse by the paretic leg is the unshaded area under the bold curve; Ibn - Braking impulse by the non-paretic leg is the unshaded area under the light curve.



SLR = 1.47

Gait Speed = 0.71 m/s

Severe hemiparesis

SLR = 0.98

Gait Speed = 0.75 m/s

Mild hemiparesis

SLR = 0.68

Gait Speed = 0.52 m/s

Mild hemiparesis

Figure 3-2. Comparison of GRFs between the paretic and non-paretic legs for subjects walking with differing SLR

Abbreviations: PHS = paretic heel strike, NTO = non-paretic toe off, NHS = non-paretic heel strike, and PTO = paretic toe off. Positive values (shaded area) represent propulsion, and the positive area under the curve is the propulsive impulse. Subject walking with SLR = 1.47 (i.e, SLR > 1.1) generates decreased paretic leg propulsion (P_P). In contrast, the subject walking at SLR = 0.98 (i.e., 0.9 < SLR < 1.1) generates symmetrical propulsive impulse and the subject walking with SLR = 0.68 (i.e., SLR < 0.9) generates relatively greater paretic leg propulsion, P_P (although low in magnitude).

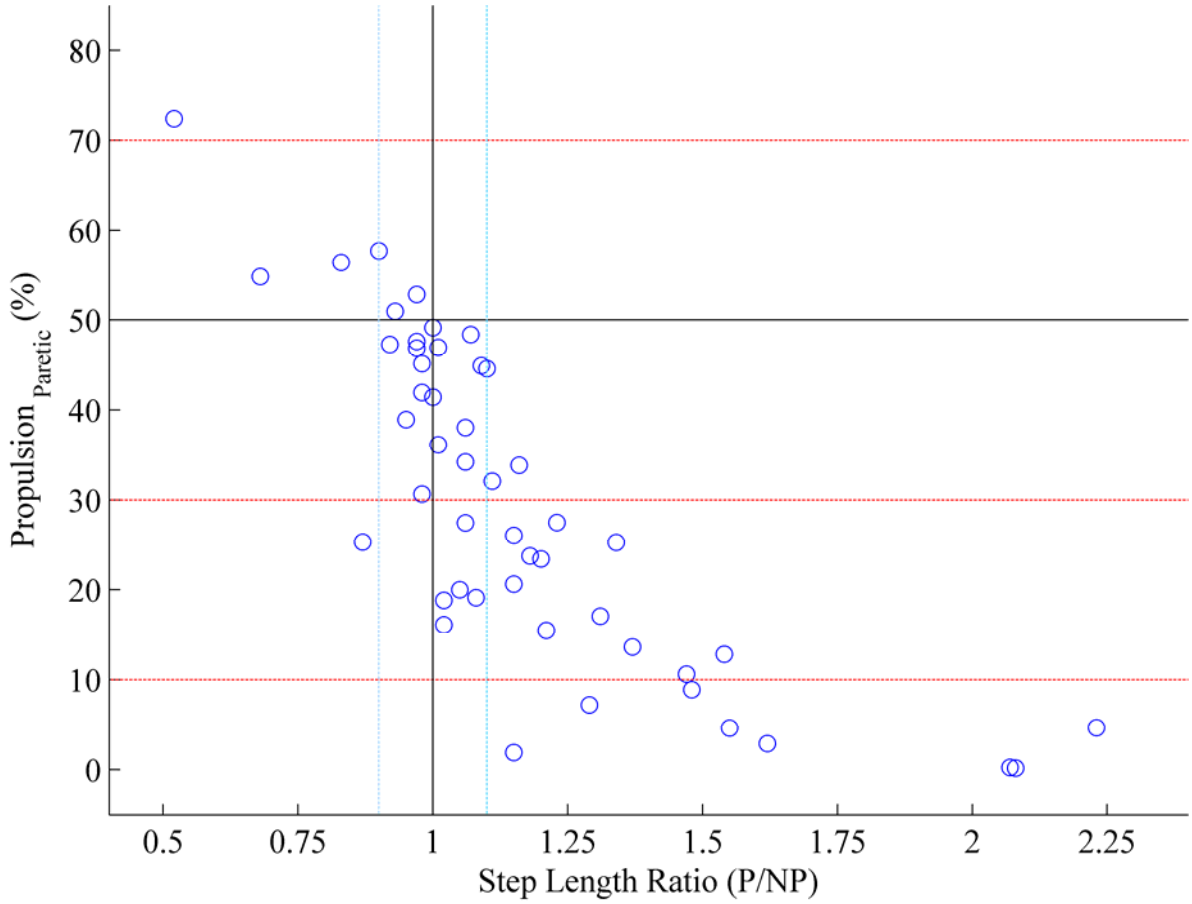


Figure 3-3. Relationship between step length ratio and Propulsion $_{Paretic}$

Abbreviations: Propulsion $_{Paretic}$ = paretic leg propulsion (in %), P = Paretic leg, NP = Non-paretic leg. Solid vertical line indicates symmetric steps (SLR = 1), vertical dashed lines indicate the SLR subdivisions at SLR = 0.9 and SLR = 1.1. Solid horizontal line indicates symmetric propulsive force generation by the paretic leg (P_P = 50%), horizontal dashed lines indicate differing levels of paretic leg propulsion (10%, 30% and 70 % P_P).

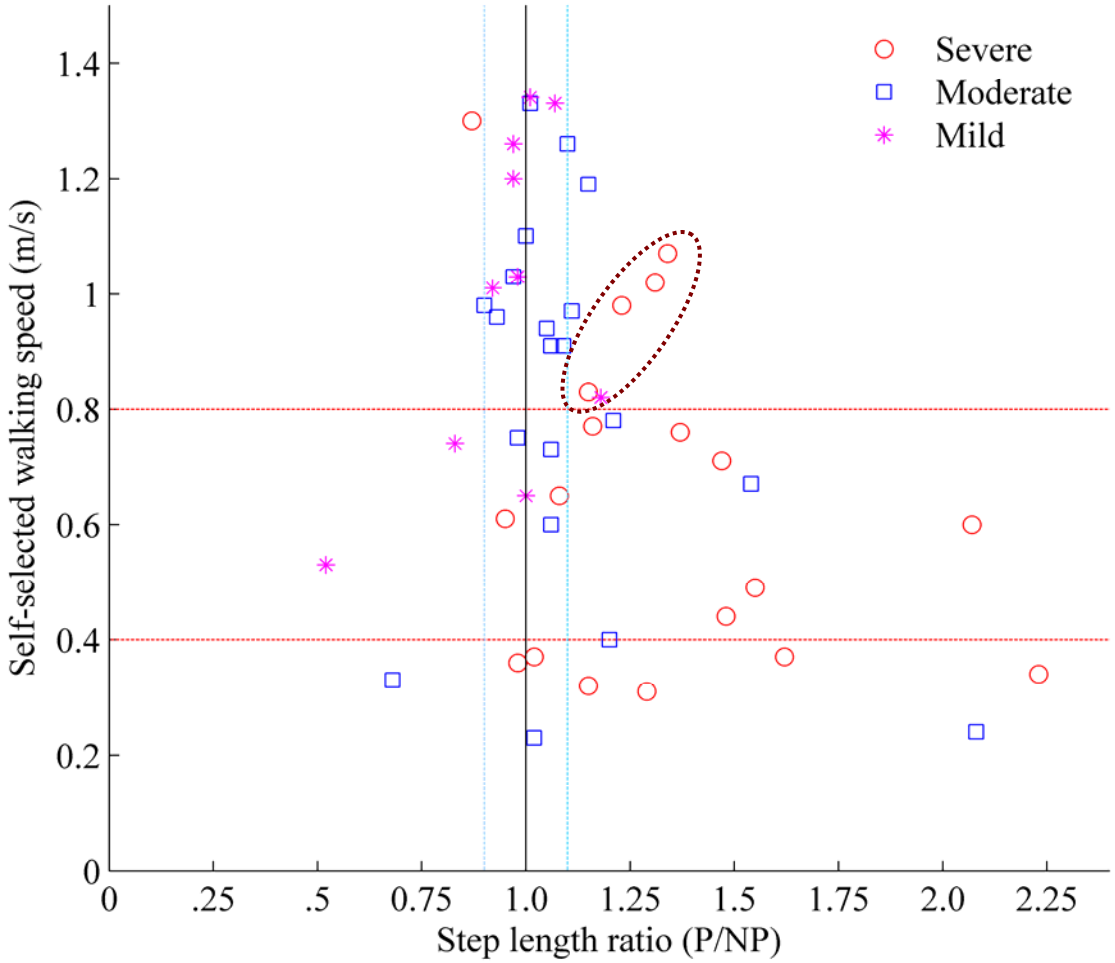


Figure 3-4. Relationship between step length asymmetry, walking speed and hemiparetic severity

Abbreviations: P = Paretic leg, NP = Non-paretic leg. Solid vertical line indicates symmetric steps (SLR = 1), vertical dashed lines indicate the SLR subdivisions at SLR = 0.9 and SLR = 1.1. Horizontal dashed lines indicate sub-divisions of walking speeds (< 0.4 m/s – household walkers, 0.4 - 0.8 m/s – limited community walkers, > 0.8 m/s – community walkers). Note that subjects with different SLR walk at all levels of walking speeds, yet majority of those with severe hemiparesis walk asymmetrically at SLR > 1.1.

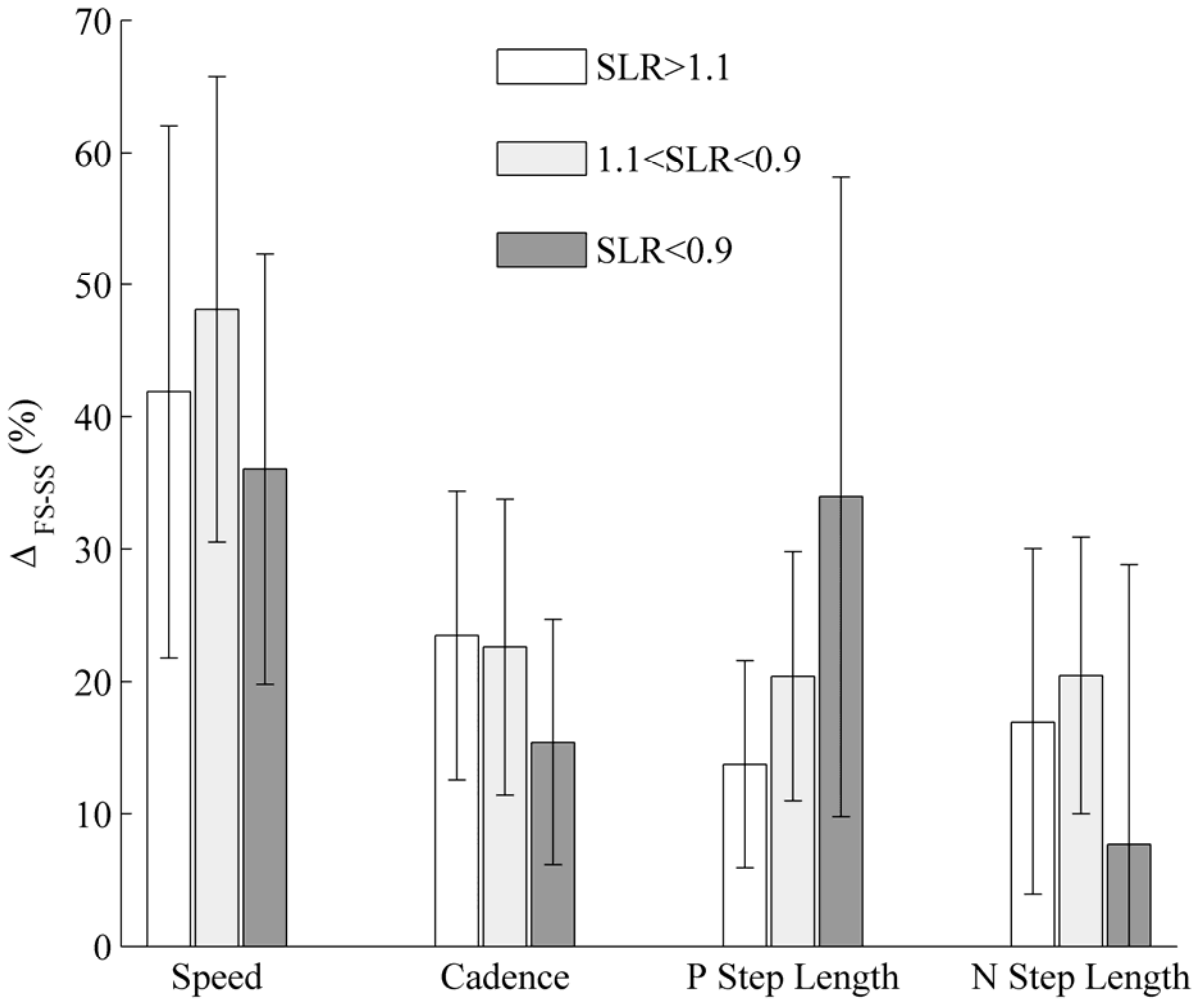


Figure 3-5. Change in speed, cadence and individual step lengths in subjects walking at different SLR [SLR > 1.1 (n = 21), 0.9 < SLR < 1.1 (n = 21), SLR < 0.9 (n = 4)]

Abbreviations: P = Paretic, N = Non-Paretic. Subjects walking with SLR > 1.1 increase their speed by primarily increasing their cadence, with little increase in the paretic step length.

CHAPTER 4
VARIABILITY IN SPATIOTEMPORAL STEP CHARACTERISTICS AND ITS RELATIONSHIP TO WALKING PERFORMANCE POST-STROKE

Introduction

Gait variability, defined as fluctuations in gait characteristics from one step to the next, is reportedly low during walking [108]. However, increased or decreased variability is commonly reported in populations with gait abnormalities like elderly fallers [103, 104], older frail adults [105] and persons with neuro-degenerative diseases (e.g, Parkinson's disease) [106, 107], suggesting that gait variability strongly associates with gait impairments. Increased gait variability has been related to risk for falls, implying that excess variability in steps might relate to balance impairments [101]. Similarly, central nervous system impairments (like cognitive functioning and motor performance) have been related to increased stance time variability [110], while decreased step width variability has been related to sensory impairments and balance deficits during walking [104, 110, 111]. Gait variability is also suggested to predict mobility disability [110]. Therefore, current evidence suggests that gait variability is related to walking impairments and can be used as quantifiable biomechanical markers to evaluate impaired performance.

In persons with post-stroke hemiparesis, neuromuscular and sensorimotor impairments can influence the generation of a smooth coordinated walking pattern resulting in gait and balance deficits that impair walking performance. Walking performance post-stroke is commonly characterized using average gait characteristics [53]. However, measures of gait variability may provide a sensitive assessment of the motor control system performance reflective of additional aspects of impaired performance. Nonetheless, gait variability has not been evaluated as measures of walking performance in the post-stroke hemiparetic population. Specifically, it is unknown whether variability in paretic steps differs from non-paretic steps, whether the

variability in steps might relate to severity of hemiparesis, and how gait variability might be associated with measures of impaired hemiparetic performance suggesting that a systematic characterization of gait variability is warranted in this population.

Therefore, the purpose of this study was 1) to evaluate whether gait variability differs in persons with post-stroke hemiparesis by comparing their variability patterns with a similarly-aged healthy population and 2) to determine if measures of gait variability may be indicative of impaired walking performance by investigating the association between gait variability and clinical assessments that evaluate impaired hemiparetic performance.

Methods

Participants

Ninety-four participants (Age = 61.4 ± 11.4 years, 69 men, 51 left-side hemiparesis, Walking speed = 0.63 ± 0.32 m/s) with chronic hemiparesis and twenty-two similarly aged healthy subjects (Age = 66.2 ± 10.0 years, 6 men, Walking speed = 1.29 ± 0.21 m/s) participated in this study (refer to Table 1 for participants' demographics). Seventy participants with hemiparesis and the healthy control subjects were part of an ongoing study at the VA-UF Human Motor Performance Laboratory, Gainesville, FL. Twenty-four participants with hemiparesis had participated in a larger gait study at the Palo Alto Medical Center and unreported data from these participants were retrospectively used for analyses. All participants signed a written informed consent. University of Florida Institutional Review Board and Stanford Administrative Panel on Human Subjects in Medical Research approved the protocols.

Participants were at least 6 months post-stroke, had unilateral weakness, could walk 10 m in 50 seconds or less without assistance from another person and had no severe perceptual, cognitive or cardiovascular impairments contraindicative to walking. Subjects were excluded if they had other neurological conditions in addition to stroke, had more than one cerebrovascular

accident, or were unable to provide informed consent. Healthy participants (serving as similarly-aged controls) were excluded if they had orthopedic or neurological disorders that influenced their walking pattern.

Procedures

All participants walked at their self-selected speeds over an instrumented walkway (GAITRite) to record spatiotemporal step characteristics. GAITRite is valid and reliable for measuring spatiotemporal characteristics [179]. All aspects of data collection at the two facilities were similar. Participants began walking 2m in front of the GAITRite, continued walking 2m after the mat (overall distance ~ 10m) to get constant speed data, and used their assistive devices (if any) during walking. All participants completed at least two walking trials (average = 3.4, range = 2 – 5 trials). Number of trials varied across some participants because a) many participants with hemiparesis were unable to walk more than 2 trials due to fatigue and low functional performance, and b) some participants walking fast completed more number of trials (4 – 5 trials) so that the effective number of steps were increased for comparative analyses.

Hemiparetic performance was evaluated using step length asymmetry index and clinical assessments. The asymmetry in step lengths during hemiparetic walking was evaluated using an asymmetry index called the Paretic step ratio (PSR), which is calculated as Paretic step length/ (Paretic + Non-paretic step length) and expressed as a percentage. Asymmetry in the hemiparetic participants was characterized based on symmetry ranges calculated from the similarly-aged healthy participants in this study and the asymmetric groups were defined as follows: “Longer” paretic steps than non-paretic ($PSR > 0.525$), “Shorter” paretic steps than non-paretic ($PSR < 0.475$) and “Symmetric” step lengths ($0.475 \leq PSR \leq 0.525$).

Clinical assessments in sub-sets of the population were available for analyses. Eighty-one study participants underwent Lower-extremity Fugl-Meyer (LE-FM) evaluations, which is a valid [71] and reliable [185] scale to evaluate hemiparetic severity. Only synergy items (22-point) of LE-FM were utilized to grade hemiparetic severity similar to earlier studies [186]. Severity of hemiparesis was graded as: severe (0 – 14: perform only within-synergy movements), moderate (15 – 18: perform movements combining synergy) and mild (19 – 22: perform movements out of synergy). Dynamic gait index (DGI) evaluated dynamic balance in thirty-two study participants. DGI rates performance of 8 walking-related tasks on an ordinal scale (0-3) (e.g., the ability to change speed and direction while walking, obstacle negotiation during walking, etc.). DGI is valid and reliable to evaluate dynamic balance in ambulatory persons with chronic stroke [187]. Performance on DGI was graded as: DGI score ≤ 19 (poor balance performance), DGI score > 19 (good balance performance) [188]. Only subsets were available for LE-FM and DGI assessments since a) only participants in the Gainesville facility underwent DGI assessments and b) since participants in the Gainesville study were part of a larger gait study, some participants were unable to complete these clinical assessments due to insufficient time.

Data Analyses

All collected footfalls from all trials were analyzed. This methodology to evaluate gait variability in clinically impaired populations is similar to earlier works [104, 110, 189-191]. Average number of footfalls collected and analyzed per subject was 25 steps (range = 12 – 42 steps) for the hemiparetic population and 13 steps (range = 9 - 20 steps) for the control population collected from all trials. Note that, for the same average number of trials, three hemiparetic participants took many more steps than other study participants (48, 50 and 65

steps). The results did not differ when these participants were removed from the analyses compared to when they were included in the analyses. Therefore, these participants' data were included in the study. The number of steps collected and analyzed in this study is similar to that reported in earlier works [91, 92, 98, 104, 110, 111, 192] that have assessed step variability and the relationship of the spatiotemporal variability to falls risk, CNS impairments, gait speed and mobility disability [98, 104, 110, 193].

Swing time, pre-swing (double-support) time, stride time, step length and stride width were selected for analyses based on a) literature review that suggests their importance in evaluating walking impairments post-stroke [70, 194], and b) evidence that the chosen variables have earlier revealed meaningful conclusions on walking impairments in gait variability studies of clinically relevant populations [101, 189, 195]. Stance and step time were not calculated since the aim was also to select mutually independent variables for analyses. Refer to Table 4-1 for definitions of calculated variables. Both stance and step times include variables that were independently calculated in the study [i.e., stance time is the two-double support phases plus single stance time (proportional to the contralateral swing time) and step time is the swing time plus pre-swing time]. Variability was quantified using the standard deviation in spatiotemporal characteristics across steps.

Statistical Analyses

Kolomogorov-Smirnov tests revealed departures from normality for spatiotemporal characteristics in participants with hemiparesis. To allow for the use of parametric statistical processing, the data were $\log_{10}$ transformed to achieve normality. Dependent t-tests revealed that there was no difference in variability between right and left legs for healthy participants in step length ($p = .32$), swing time ($p = .51$) and pre-swing time ($p = .22$) variability. Therefore, a one-way ANOVA tested differences in step length, swing and pre-swing time variability between

paretic, non-paretic and healthy (left) leg. Results did not differ when the right leg of the healthy participants were used in the analyses. For stride time and stride width, independent t-tests were conducted to test differences between population groups (hemiparetic and healthy). Note that since stride time and stride width were calculated using both right and left steps, there was only one factor (population) and only paretic strides were analyzed to avoid redundant steps in the analyses.

To test differences in variability across severity and PSR groups a 3 (group) x 2 (leg) Mixed ANOVA (repeated on leg factor) was conducted for step length, swing time, pre-swing time variability and a one-way ANOVA for stride time and width variability. To test differences across DGI groups a 2 (group) x 2 (leg), Mixed ANOVA (repeated on the leg factor) was performed for step length, swing time, pre-swing time variability and an independent t-test for stride time and stride width variability. When significant effects were detected, post-hoc pairwise comparisons were performed using Bonferroni-adjusted t-tests. All statistical analyses were conducted in SPSS (version 13.0).

Results

Differences in Step Variability between Healthy and Hemiparetic Walking

Variability in step length, swing, pre-swing and stride time was increased in participants with hemiparesis compared to healthy control subjects, while stride width variability ($p = .153$) was not changed in hemiparetic walking (Figures 4-1 and 4-2). However, if only the slower walkers (speed < 0.4m/s) were compared to controls, then width variability was significantly reduced ($p = .038$).

For between-leg comparisons, swing time variability was greater in paretic steps than non-paretic and there was a trend for paretic pre-swing (PPS) time to show greater variability

compared to non-paretic pre-swing (NPS) time ($p = .065$, Figure 4-1). There was no difference in the variability between paretic and non-paretic step lengths (Figure 4-2).

Association between Step Variability, Clinical Assessments and Asymmetry Index

Differences in variability across the three groups (determined by the assessments) for each spatiotemporal characteristic are presented below. Additionally refer to Figure 4-3 and Figure 4-4 for presentation of differing patterns of spatiotemporal variability within the severity, asymmetrical and DGI groups and Table 4-2 for the mean variability across-groups.

Swing time variability, Main effects (ME) of Group and Leg were significant across severity ($p<.01$), asymmetrical ($p<.01$) and DGI groups ($p\leq.02$). Paretic steps showed greater variability than the non-paretic steps across all groups, with greatest between-leg differences in persons with severe and moderate hemiparesis, those taking Longer paretic steps and showing poor balance performance (DG1 $\leq$ 19).

Pre-swing time variability, ME of both Group and Leg were significant across severity ($p<.01$) groups, ME of leg ($p<.001$) was significant across asymmetrical groups and ME of group was significant across DGI. PPS showed greater variability than NPS in the severe and moderate groups and across asymmetrical groups. Both PPS and NPS showed greater variability in persons showing poor balance performance compared to those showing good balance performance.

Stride time variability differed across severity groups ($p < .0001$). Severe and moderate groups showed greater stride time variability than the mild group ($p < .003$), but did not differ from each other ($p > .05$, Table 4-2). Stride time variability differed across asymmetrical groups ($p = .023$) but post-hoc tests showed that asymmetrical groups showed only a trend (of greater variability) to differ from the symmetrical group ($p \leq .109$). Stride time variability ($p = .007$) was greater in persons showing poor balance performance (DGI $\leq$ 19), (Table 4-2, Figure 4-3).

Step length variability showed a trend to differ across severity groups ($p = .069$) and asymmetrical groups ($p = .094$). However, ME of group and leg were significant for step length variability (group: $p = .026$, leg: $p = .03$) across DGI groups ($p \leq .03$), with the non-paretic leg showing greater variability than the paretic leg.

Stride width variability showed a trend to differ across severity groups ($p = .045$) but did not differ across the asymmetrical or DGI groups ($p > .95$).

Discussion

Differences in Step Variability between Healthy and Hemiparetic Participants

We found that, similar to other populations with gait deficits [111, 190, 196], variability in all spatiotemporal characteristics (except stride width) increased in persons with post-stroke hemiparesis compared to healthy controls (Figure 4-1, 4-2). While increased variability has been related to gait deficits in impaired populations [106, 111, 190, 196], our study is the first to report between-leg differences in step variability. We found between-leg differences in swing and pre-swing time variability suggesting a direct association between underlying paretic leg impairments and step variability. Paretic swing time variability was greater than the non-paretic leg and this difference in variability between-legs was greatest for persons showing most impaired performance (severe and moderate hemiparesis, asymmetrical steps and those at risk for falling as predicted by lower DGI scores) (Figure 4-3, 4-4). Increased step variability specifically in the paretic leg might relate to the neuromuscular impairments after a hemiparetic stroke, such as altered neural inputs to the paretic spinal half-centers, altered effects of afferent feedback to the paretic leg and impaired inter-limb coordination during walking. Furthermore, PPS variability showed a trend to be greater than NPS. Prolonged time in PPS has been related to impaired progression during hemiparetic walking [70]. The increased variability in PPS relative to NPS suggests that the neuromotor deficits in this phase may limit hemiparetic walking

performance. Step length variability did not differ between-legs but was greater during hemiparetic walking compared to healthy. It is likely that spatial variables such as step length (that determine the base of support during gait) are inherently more tightly coordinated between-legs than temporal variables such that step-to-step variation in one leg is counter-balanced by variation in the other leg to maintain steady-state walking. Furthermore, stride time variability was also increased during hemiparetic walking compared to healthy. Increased stride time variability is reported to be strongly related to falls risk [101, 103], suggesting that increased variability post-stroke might relate to poor dynamic balance.

Relationship between Hemiparetic Step Variability and Impaired Performance Post-Stroke

To test the use of gait variability measures as markers of impaired performance, we wanted to investigate the relationship between step variability and hemiparetic performance. This relationship also provided insights on potential mechanisms underlying the variability patterns. For example, the ability to produce independent voluntary movements of the paretic leg is related to motor recovery of the paretic leg and is graded using the LE – FM (higher score greater recovery). The inverse relation between hemiparetic severity and step variability suggests that variability might decrease as motor recovery progresses. Similarly, greater step length asymmetry (both Longer paretic and Shorter paretic groups) has been related to motor control impairments [194]. In support of this hypothesis, our results revealed that both asymmetrical groups presented with greater swing time variability compared to the symmetrical group but did not differ from each other. Moreover, the association between step variability and hemiparetic performance is revealed in the relation between DGI scores and variability (Figure 4-3, 4-4). Since DGI has been used to evaluate dynamic balance [187] (lower score lower balance), the inverse relation between DGI scores and step variability suggests that persons showing greater step variability might have poor dynamic balance. Specifically, DGI scores less than equal to 19

are reported to identify persons at risk for falling [188] and participants with hemiparesis in our study with scores less than equal to 19 exhibited greater step variability.

While the overall increased step variability was related to impaired performance (lower LE – FM score, greater asymmetry and lower DGI score), the pattern across the sub-groups was not consistent for all spatiotemporal parameters (e.g. between-leg differences in pre-swing time variability was observed across severity groups but not across DGI groups). Since the stroke population is immensely heterogeneous, we expected the spatiotemporal step variability patterns would differ across sub-groups of participants suggesting that specific spatiotemporal measures are more strongly associated with particular aspects of hemiparetic performance. For instance, between-leg differences in swing time variability were significant across all sub-groups of participants (severity, asymmetry and DGI groups) suggesting that between-leg differences in swing time variability were most strongly related to impaired hemiparetic performance (as measured by severity, asymmetry and DGI). Stride time variability differed across all sub-groups. In comparison, for step length variability, there were no differences between-legs when considering the hemiparetic population as a group. However, in persons at risk for falling, paretic variability was increased relative to non-paretic; suggesting that step length differences between-legs might be unmasked in the most impaired persons. Nonetheless, the relatively small sample size of the DGI subgroups could have potentially influenced these results.

While step variability in different spatiotemporal parameters varied across sub-groups of participants in combination, variability in the spatiotemporal parameters seemed to robustly predict impaired hemiparetic performance. For instance, a participant having markedly increased PPS variability (black-bold arrow in Figure 4-3), increased paretic step length and reduced width variability (black-bold arrow in Figure 4-4) shows impaired hemiparetic walking performance.

The inferences from other measures, however, were inconsistent (poor balance performance (DGI = 8), moderate hemiparetic severity, symmetric steps). Similarly, another participant had good balance performance (DGI = 22) but walked asymmetrically with much longer paretic steps, again indicating the contrasting inferences on performance as predicted by these measures. This person had markedly increased stride time variability and reduced width variability (red arrow in Figure 4-4). Overall, these examples exhibit a marked increase of variability in one or other spatiotemporal characteristic and reduced variability in stride widths, suggesting that although poor hemiparetic performance was not consistently evident across all clinical assessments, gait variability robustly identified impaired walking performance.

Unlike other spatiotemporal characteristics, stride width variability was reduced in the slower walkers when compared to controls. Stride width is calculated in the frontal plane unlike other spatiotemporal characteristics that are sagittal plane measures, suggesting an inherent difference in control of this parameter. Furthermore, in population groups susceptible to falls (such as elderly fallers, persons with Parkinson's disease and community dwelling elderly), width variability is reported to be reduced and this reduction in variability is shown to predict falls [104, 107, 189]. In our study, we also observed that participants showing markedly reduced width variability walked with wide strides. A wider step provides a wider base of support for side-to-side motion of the center of mass and might be accompanied by a reduction in variability of medio-lateral foot placement to ensure that steps are consistently wide. Therefore, it is likely that observed reduction in width variability is compensatory to maintain stability. Decreased width variability in comparison to increased variability in other spatiotemporal characteristics implies that altered variability might be specific to step characteristics and should not be generalized.

Study Limitations

There were some limitations in our study. Data was collected from a limited number of steps that could have influenced the accuracy of our results [95]. However, a major strength of our methodology was the ability to measure spatial variables as well. Methodologies that capture hundreds of steps are based on recording temporal characteristics only [104]. Furthermore, despite the fewer number of steps collected in our study, observed patterns of gait variability were consistent with those observed in other impaired populations [101, 106, 111]. Further, use of the GAITRite based protocol is clinically relevant and therefore, our study results have the potential to rapidly translate to the clinical settings. We also evaluated gait variability using coefficient-of-variation (CV = standard deviation/mean) in step characteristics. Using CV to evaluate variability may result in questionable conclusions of increased variability, specifically when the mean value is of low magnitude. It is also likely that the differences in stride variability could be due to differences in strategies (i.e., length-frequency combinations) employed to achieve a certain speed [197]. Nonetheless, several studies have shown that stride variability, could be independent of stride length and frequency [91, 189].

Furthermore, it is likely that the trial-to-trial variability in speed resulted in observed changes in the variability. However, the speed variability did not differ ($p = 0.268$) between hemiparetic and healthy participants, suggesting that the trial-to-trial speed variation did not contribute to the observed patterns of gait variability. Slower walking speeds post-stroke are likely to be associated with greater alterations in step variability. Yet, observed patterns of gait variability cannot be solely attributed to effects of speed because if healthy persons walk at slower speeds similar to persons with gait deficits, gait variability is likely to remain relatively low during healthy gait even at the slower speeds [111]. In addition, while persons walking with assistive devices showed overall greater step variability than those not using assistive devices,

the inverse relationship between variability and walking performance was apparent when we evaluated the data for those using assistive devices separately from others who did not.

Conclusions

In conclusion, results of this study suggest that step variability is altered post-stroke compared to healthy controls and relates to hemiparetic walking performance. Specifically, between-leg differences in swing time and pre-swing variability, increased step length and stride time variability and reduced width variability can be indicative of underlying sensorimotor impairments post-stroke; suggesting these as quantifiable measures of impaired hemiparetic performance. Future studies should investigate the underlying causes of altered variability and the effect of therapeutic interventions on gait variability to further validate its use to assess hemiparetic performance.

Table 4-1. Definitions of study variables

STEP LENGTH (cm): measured along the line of progression, from the heel center of the current footprint to the heel center of the previous footprint on the opposite foot.
PRE-SWING TIME (sec): Pre-swing (or double support) time occurs from opposite footfall first contact to support footfall last contact.
SWING TIME (sec): time elapsed between the Last Contact of the current footfall to the First Contact of the next footfall of the same foot.
STRIDE TIME (sec): time elapsed between the first contacts of two consecutive footfalls of the same foot.
STRIDE WIDTH (cm): vertical distance from midline midpoint of one footprint to the line formed by midline midpoints of two footprints of the opposite foot.

Table 4-2. Step variability (expressed as standard deviation) within the hemiparetic population sub-divided based on their performance measures

	Severity groups			PSR groups			DGI groups	
	Severe	Moderate	Mild	Longer	Shorter	Symmetric	≤ 19	>19
Avg. walking speed	(n=24) (0.5m/s)	(n=23) (0.5m/s)	(n=34) (0.8m/s)	(n=35) (0.7m/s)	(n=11) (0.5m/s)	(n=48) (0.6m/s)	(n=30) (0.5m/s)	(n=9) (0.7m/s)
Step length variability (cm)								
Paretic	3.37	4.01	2.89	3.13	3.76	3.14	3.78	2.51
Non-paretic	3.44	3.72	2.96	3.47	3.69	3.02	4.11	3.18
Swing time variability (s)								
Paretic	0.07	0.07	0.03	0.07	0.06	0.04	0.07	0.03
Non-paretic	0.04	0.04	0.02	0.03	0.05	0.03	0.05	0.03
Pre-swing variability (s)								
Paretic	0.08	0.13	0.02	0.06	0.16	0.07	0.13	0.03
Non-paretic	0.07	0.08	0.02	0.04	0.08	0.05	0.09	0.03
Stride time variability (s)	0.13	0.16	0.05	0.12	0.19	0.08	0.16	0.06
Stride width variability (cm)	1.83	1.55	1.90	1.78	1.77	1.77	1.87	1.68

Standard deviation in the spatiotemporal characteristics is rounded to the second decimal place. Abbreviations: PSR – Paretic step ratio, DGI – Dynamic gait index.

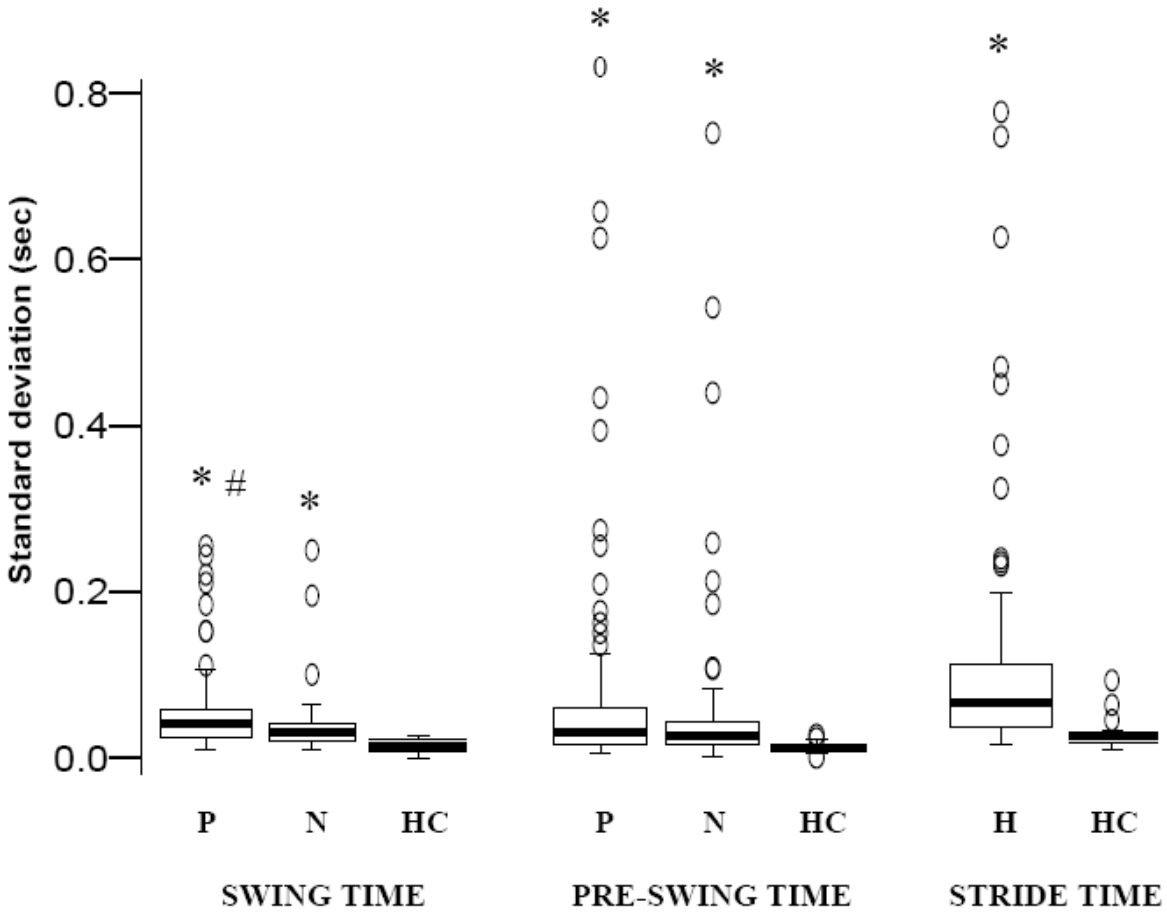


Figure 4-1. Differences in temporal variability between healthy (n = 22) and participants with hemiparesis (n = 94) at Self-selected (SS) walking speeds

The box plots indicate the range in the data. The central horizontal line is the median of the sample. The length of the box indicates the inter-quartile range with the upper and lower boundaries of the box indicating the upper and lower quartile, respectively. Circles represent sample values that statistically indicate outlier or extreme values (by SPSS software). In impaired populations, these outlier values are true indicators of behavior and represent those persons showing excessive variability. * indicates statistically significant differences from healthy leg at $p < .0001$, # indicates statistically significant difference from non-paretic leg at $p < .0001$. Note that the statistical significance is based on the mean of the log-transformed values of the respective temporal characteristics. Variability in all temporal characteristics was increased during hemiparetic walking. Abbreviations: P – Paretic leg, N – Non-paretic leg, HC – Healthy control leg, H – Hemiparetic walking (value includes steps from both legs).

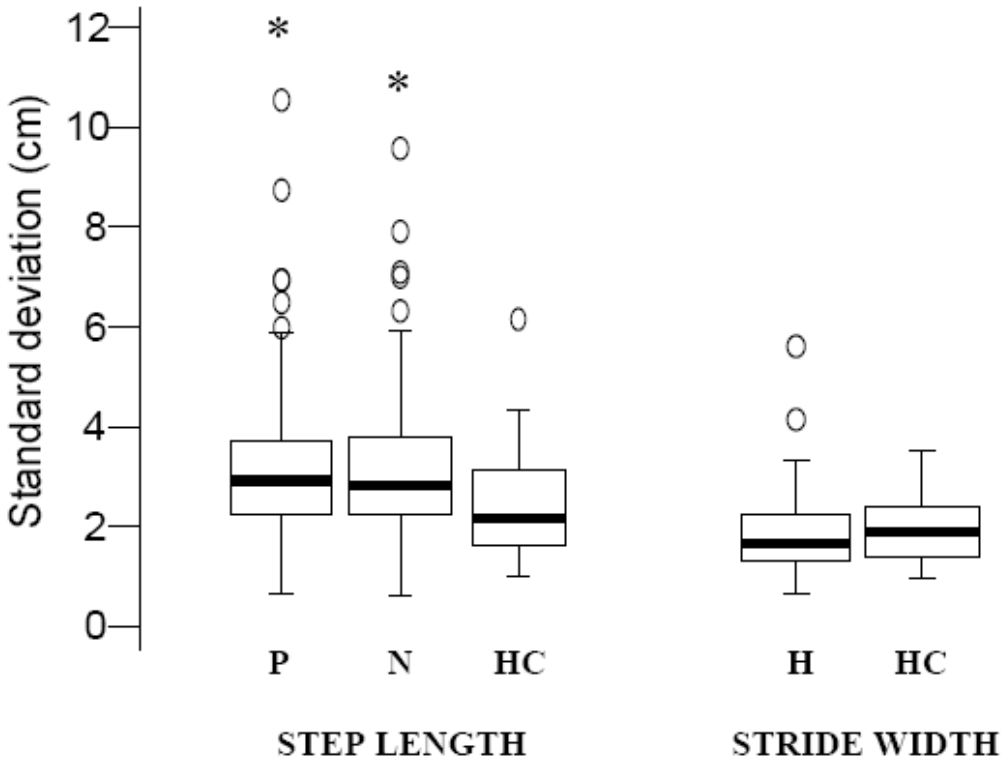


Figure 4-2. Differences in spatial variability between healthy (n = 22) and participants with hemiparesis (n = 94) at Self-selected (SS) walking speeds

The box plots indicate the range in the data similar to Figure1. * indicates statistically significant differences from healthy leg at $p < .0001$ and the statistical significance is based on the mean of the log transformed values of the respective temporal characteristics. Variability in step length characteristics was increased during hemiparetic walking and that in stride width showed a trend to decrease during hemiparetic walking. Abbreviations: P – Paretic leg, N – Non-paretic leg, HC – Healthy control leg, H – Hemiparetic walking (value includes steps from both legs).

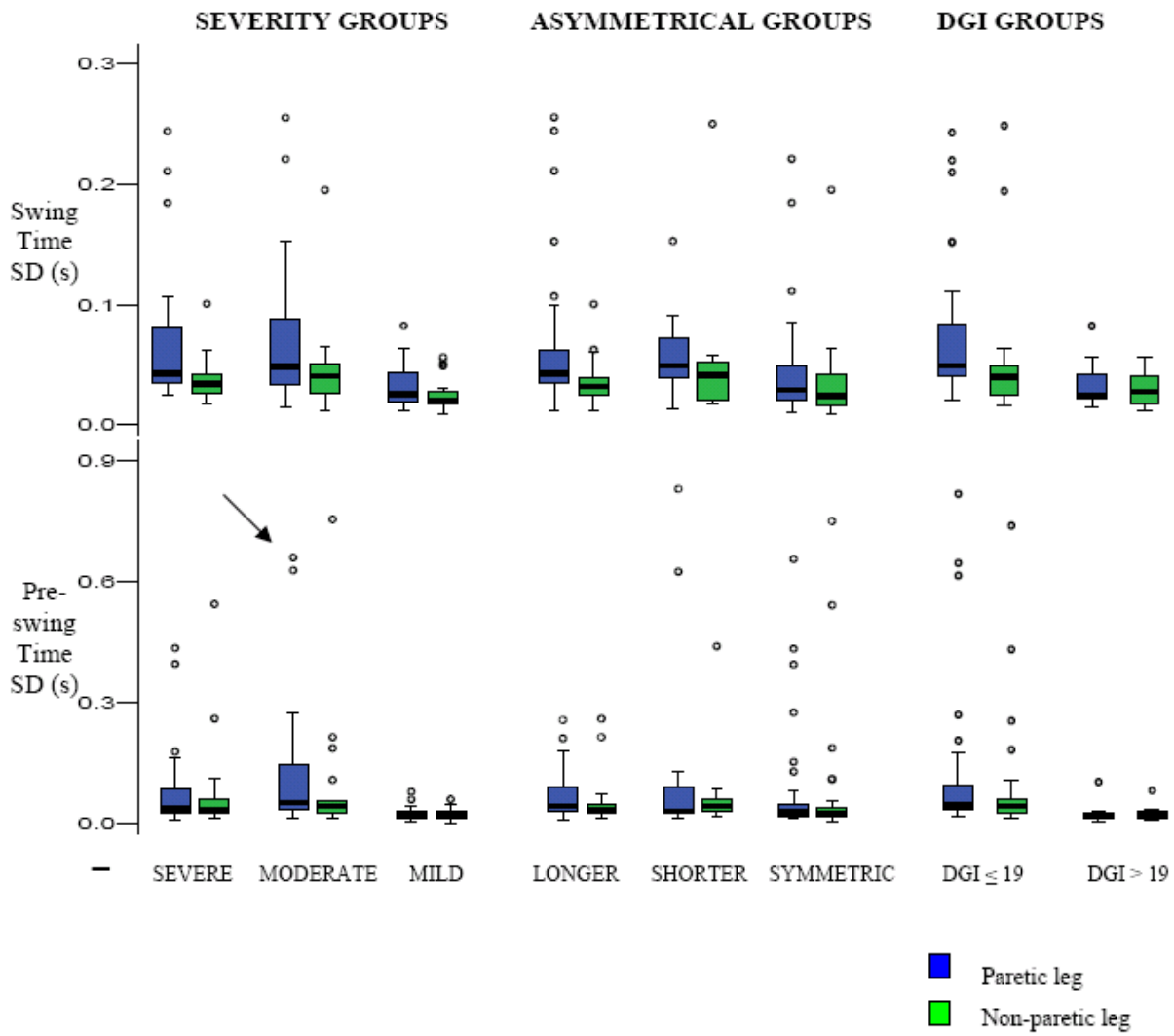


Figure 4-3. Differences in temporal variability in hemiparetic participants based on their performance on clinical assessments

The box plots indicate the range in the data similar to Figures 1. Blue represents paretic leg and Green represents non-paretic leg for swing time and pre-swing time variability. In general, poor performance is indicated by more severe hemiparesis (lower LE-FM scores), asymmetrical gait (longer or shorter paretic steps) and poorer balance performance (lower DGI scores)]. Note the between-leg differences in swing and pre-swing time in persons with moderate and severe hemiparesis, those walking asymmetrically and in persons showing poor balance performance. Note that while stride time variability differences were observed in sub-groups of the hemiparetic population, these have not been represented in the Figure.

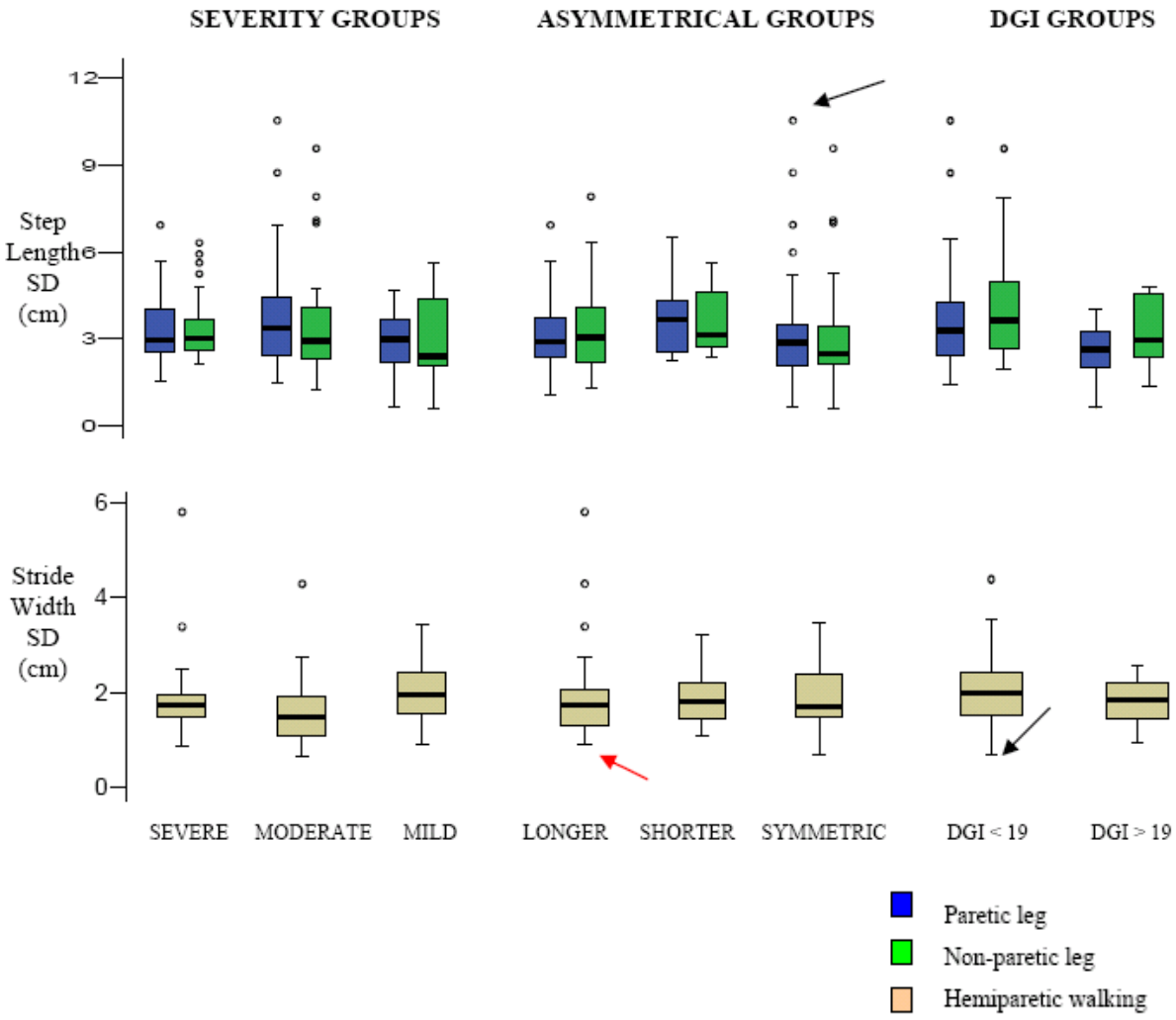


Figure 4-4. Differences in spatial variability in hemiparetic participants based on their performance on clinical assessments

The box plots indicate the range in the data similar to Figures 1. Blue represents paretic leg and Green represents non-paretic leg for step length variability. Note that stride width variability does not show a consistent trend to differ across the different sub-groups of the hemiparetic population.

CHAPTER 5
FOOT PLACEMENT IN A BODY REFERENCE FRAME DURING WALKING AND ITS RELATIONSHIP TO HEMIPARETIC WALKING PERFORMANCE

Introduction

Foot placement during walking is commonly quantified using parameters that are determined relative to the other foot during a step (e.g., step length and step width). Nonetheless, foot placement closely relates to body movements [117, 118]. For instance, forward body progression during walking requires controlling the movement of the body's center-of-mass relative to where the foot is placed. Similarly, dynamic stability during walking is established by foot placement in a body reference frame (in relation to body movement). Specifically, by foot placement in a body reference frame, we imply calculation of where the foot is placed relative to the body during walking (and not relative to the other foot). For instance, step length in a body reference frame can be calculated as the anterior distance between the leading foot center-of-mass and the center-of-mass of the pelvis at heel strike. Patterns of foot placement relative to the body are rarely investigated during gait. In some earlier studies, foot placement relative to body was investigated during healthy gait and related to motor control during gait [116-119, 134]. However, foot placement relative to body has not been investigated in neurologically impaired populations and specifically in an asymmetrical population such as stroke. The observations of asymmetric foot and trunk kinematics in isolation [198, 199] suggests that foot placement relative to body likely are asymmetric post-stroke but it is unclear whether persons with specific motor control deficits would have particular patterns of where they place their foot relative to body.

Post-stroke, quantifying where the foot is placed relative to body could provide a deeper understanding of the mechanisms of hemiparetic walking than is possible when foot kinematics alone are known (as when it is defined relative to other foot). For example, in persons who take

asymmetrical step lengths (relatively longer or shorter paretic step lengths), it is unclear whether their foot placements relative to pelvis (or trunk) would also be asymmetrical. It is possible that, in those persons who take longer paretic steps than non-paretic, while the paretic step is longer relative to the other foot; relative to pelvis both feet are placed symmetrically. Furthermore, in persons taking asymmetrical longer or shorter paretic steps than non-paretic, paretic and non-paretic stride lengths are likely to be the same. This suggests that investigation of foot placement relative to pelvis can reveal differential patterns of asymmetrical foot placement anterior or posterior to the pelvis and help understand the specific motor control in such asymmetrical sub-groups. Similarly, it is unclear whether there is any asymmetry in lateral foot placements relative to pelvis and whether it relates to step width post-stroke. Therefore, in this study patterns of foot placement relative to body were compared to step length asymmetry and step width to investigate the relation between stepping in a body reference frame to stepping relative to the other foot.

Furthermore, we expected that investigation of foot placement relative to body will provide insights into forward progression, weight support and dynamic stability during hemiparetic walking. Accurate foot placement in relation to the body during walking establishes a stable base for the body to progress forward [118, 132]; implying that foot placement during walking is closely related to forward progression. Furthermore, lateral balance of the body depends on the mediolateral foot placement [134] suggesting that identification of persons with hemiparesis with specific patterns of mediolateral foot placement can help determine their dynamic stability. Mediolateral foot placement relative to the body can also influence the weight shifted and supported on the legs since how far one places their foot relative to the body at ground contact will influence the extent of body displacement. Therefore, we also investigated the relation

between mediolateral foot placement relative to body and weight supported on the leg to identify persons with disturbances in weight shifting. Therefore, in this study, in addition to quantifying foot placement relative to body during hemiparetic walking, the relationship between foot placement relative to body and walking performance was explored. Walking performance was quantified using step length asymmetry patterns, paretic propulsion, weight support and a dynamic stability margin.

In summary, the purpose of this study was 1) to evaluate where the foot is placed relative to the body during walking in a post-stroke population and, 2) to understand how foot placement in the body reference frame relates to walking performance. Foot placement relative to body was calculated in the anterior-posterior and medial-lateral directions during hemiparetic walking and compared to patterns of foot placement in a similarly-aged healthy population (serving as controls) walking at matched slow speeds. Inclusion of control subjects was deemed necessary to compare the foot placement patterns in the hemiparetic gait to that of healthy gait. We hypothesized that the measures of stepping performance (step lengths and step widths), forward progression, weight support and dynamic stability would relate to the asymmetry in foot placement relative to body such that investigation of foot placement in a body reference frame will provide insights into essential requirements of locomotion and help better evaluate asymmetric gait post-stroke.

Methods

Participants

Data were collected from thirty-nine participants with chronic hemiparesis (Age = 60.21 ± 12.32 years, 20 men, 19 left-side hemiparesis) and twenty age-matched healthy participants (Age = 66.15 ± 10.03 years, 4 men) at the VA-UF Human motor performance laboratory, VA Medical center at Gainesville Florida. Inclusion criteria for the participants with hemiparesis were:

hemiparesis secondary to a single onset unilateral stroke; ability to ambulate independently with or without an assistive device over 10 m on a level surface; ability to walk on a regular basis at least at home; absence of significant lower extremity joint pain and major sensory deficits; absence of significant lower limb contractures and no significant cardiovascular or respiratory symptoms contraindicative to walking. Participants from the study were excluded if they had any orthopedic or neurologic conditions in addition to stroke, had significant musculoskeletal problems other than stroke that limit hip and knee extension or ankle plantar flexion to neutral, or were unable to provide informed consent. All participants in the study signed a written informed consent and Institutional Review Board of University of Florida approved the protocol.

Procedures

Retro-reflective markers were attached to the participant to collect bilateral 3D kinematics using a 12 camera VICON motion analyses system. Markers were attached to the head, trunk, upper extremity, lower extremity and the feet. Clusters of reflective markers attached to rigid bodies were also located on the pelvis, bilateral thighs, shanks and feet (Figure 5-1). A fixed laboratory coordinate reference frame was created within the VICON system that was placed at the left corner of the laboratory.

At the beginning of the test session, controls and participants with hemiparesis walked for 2-3 trials across a 12 ft long instrumented mat (GaitRite) at their self-selected walking speeds to collect over ground spatiotemporal parameters of steps and estimate their overground walking speeds. Gaitrite is a valid instrument to measure spatiotemporal parameters during walking [179].

Subsequently, controls and participants with hemiparesis walked on an instrumented split-belt treadmill (TECMACHINE) for three trials at their self-selected treadmill walking speed. In case of participants with hemiparesis, the treadmill self-selected speeds were 10 – 30% slower

than the over ground self-selected speeds. Participants with hemiparesis completed three 30-second walking trials without use of an assistive device or ankle-foot orthosis. A safety harness mounted to the laboratory ceiling was worn across the shoulders and chest to protect the participants in the event that they loose balance. Note, that no bodyweight was offloaded by the harness. Additionally, a physical therapist closely guarded the participants as they walked over the treadmill (although no manual support was provided by the therapist). For healthy participants, treadmill speeds closely approximated their over ground speeds. Healthy participants, in addition, walked at fixed slow speeds on the treadmill for a single trial at 0.3 m/s, 0.6 m/s, and 0.9 m/s to provide control data at speeds matched to the slower speeds of the hemiparetic participants.

To optimize capture of steady state data on the treadmill, each subject walked for 10 s prior to each of the 30 s of data collection. Three-dimensional GRFs were measured from each half of the treadmill along with the kinematic data collection during the walking trials.

Data Analyses

Kinematic data: Raw kinematic data was low-pass filtered using a fourth-order zero-lag Butterworth filter with a 10 Hz cutoff frequency. The joint center and anatomical trajectories was fitted to an eight-segment musculoskeletal model generated using SIMM (MusculoGraphics, Inc.) consisting of a trunk (including the mass of the torso, head and arms), pelvis and legs for each subject (Figure 5-1 presents the SIMM model from an individual participants walking trial). Each lower extremity consisted of a thigh, shank and foot. The anthropometrics and inertial properties were based on that of de Leva [200]. Segmental center-of-mass (COM) calculations were used to calculate the whole body COM. In this study, specifically pelvis COM was used to reference the body.

Kinetic data: Three-dimensional GRFs were sampled at 2000 Hz. Ground reaction force data was low-pass filtered using a fourth-order zero-lag Butterworth filter with a 20 Hz cutoff frequency. The SIMM Motion Module (MusculoGraphics, Inc.) was used to perform a standard inverse dynamics analysis to determine the inter-segmental joint moments. Kinetic data (GRFs and joint moments) was normalized by subject body weight. Kinetic data was time normalized to 100% of the paretic leg gait cycle (paretic heel strike to paretic heel strike).

Calculation of study variables: All variables were calculated by averaging across all complete gait cycles of each of the three trials. The number of steps that are used to calculate the averages varied across subjects since they walked at different speeds.

Foot Placement and foot position variables: Anterior-Posterior (AP) and medial-lateral (ML) foot placement and position variables relative to pelvis were calculated for individual legs. The body was referenced at the pelvis COM (Figure 5-2). Calculation of initial foot placement (calculated at ground contact in the 1st double support phase) and terminal foot position (calculated as toe-off in the 2nd double support phase) in AP and ML direction is presented in Table 5-1. The term foot position was used to distinguish these variables from foot placement variables since the foot position variables describe how the body movement progressed in a specific positioning of the foot relative to the body. *Stepping relative to the other foot:* Step length and step widths were calculated for individual legs at the same instant as the variables calculated in the body reference frame (Table 5-1). *Stepping asymmetry, forward progression, weight support and Dynamic stability:* Step length asymmetry was quantified using a Paretic step ratio [194], (Table5-1). Asymmetry in the hemiparetic participants was characterized based on symmetry ranges calculated from similarly-aged healthy participants walking on the treadmill, as follows: ‘Longer paretic’ steps than non-paretic (PSR > 55), ‘Shorter paretic’ steps than non-

paretic (PSR < 45) and 'Symmetric' step lengths (45 ≤ PSR ≤ 55). Forward progression was quantified using paretic propulsion [76], (Table 5-1). Weight support was quantified by the average vertical force supported on individual legs during the stance phases (Table 5-1). Dynamic stability was evaluated by a Dynamic Stability Margin (DSM) similar to earlier studies [201, 202], (Table 5-1). Specifically, we used the DSM that we calculated at the instance at which the xCOM reached its maximum value in 1st double-support (DSMmax).

Statistical Analyses

For control subjects, right-left foot placements and positions were similar at all speeds (i.e., no between-leg asymmetry), in both AP and ML direction relative to pelvis ($p < .001$). Therefore, only control (left) leg was used in the comparisons with participants post-stroke. To compare foot placement and position relative to the body between controls and participants with hemiparesis, a 3 (leg: paretic, non-paretic, control) x 2 (phase: foot-strike, foot-off) Mixed ANOVA was conducted. Median foot placements of control participants at varying speeds were compared to participants with hemiparesis walking at similar range of speeds in the statistical analyses. Controls participants walked at 3 varying slow speeds: 0.3 m/s, 0.6 m/s and 0.9 m/s. Therefore, the comparisons of hemiparetic participants were made for those persons walking less than equal to these speed ranges. Note that each speed group consisted of different hemiparetic participants walking at their self-selected speeds. Since median foot placements were used in the analyses, number of controls was matched to that of the hemiparetic in each speed group. Pearson correlations were conducted to evaluate the relationships between foot placements, positions and walking performance measures. Quadratic relations were additionally explored for each of the relationships but it did not improve the explanatory power significantly in any relationship, therefore linear relationships have been presented.

Results

Quantifying Foot Placement Relative to the Pelvis

For control subjects, right-left foot placements and positions were similar at all speeds (i.e., no between-leg asymmetry), in both AP and ML direction relative to pelvis ($p < .001$). Therefore, the left leg was used as the control leg in the comparisons with participants post-stroke.

AP direction: In control participants, foot placement was anterior at ground contact (GC) and foot position posterior at toe-off (TO) relative to pelvis, with the anterior placement showing greater excursion than posterior at all the varying speeds (40 – 80 mm, $p < .001$), (Figure 5-3a). The greater anterior excursion of the foot at GC than posterior at TO relative to pelvis is operationally termed as between-phase asymmetry in this study to differentiate from between-leg asymmetry. In participants with hemiparesis, the pattern of paretic and non-paretic foot placement varied with their walking speeds.

Foot placement in slow walkers (≤ 0.3 m/s, n = 22) and controls walking at matched-speeds: Both between-leg and between-phase asymmetry was observed in those hemiparetic participants walking slowly. Median foot placements were anterior at GC and posterior at TO, with the paretic foot being more anterior and less posterior than non-paretic ($p=.003$). There was increased inter-subject variability in posterior paretic foot position at TO such that at least half the participants in this group never positioned their foot posterior to the pelvis (Figure 5-3a). The control foot showed greater excursion than both the paretic and non-paretic feet even at matched slow speed ($p<.001$). However, the difference in anterior foot placement between control and paretic feet was small (~30 mm).

Foot placement in Moderate (> 0.3m/s – 0.6 m/s, n = 12) and Fast walkers (> 0.6 m/s – 0.9m/s, n = 5) and controls walking at matched-speeds: For participants walking at moderate

(0.3 – 0.6 m/s) and fast (0.6 – 0.9 m/s) speeds there was no between-leg asymmetry but between-phase asymmetry was apparent (GC – TO relative to pelvis = 6 – 10 cm), ($p<.01$), Figure 5-3a. Further, for the fast walkers foot placement excursions were similar to that of Controls ($p<.001$), excepting paretic anterior foot placement that was reduced.

ML direction: There was no between-phase asymmetry in foot placements in controls and participants with hemiparesis (i.e., same ML distance at GC and TO). Therefore, lateral foot placement at GC is presented. In participants with hemiparesis walking slow and at moderate speeds, the paretic foot was placed lateral to pelvis compared to both non-paretic (difference range = 30 – 70 mm) and speed-matched control (difference range = 49 - 74 mm) foot placement ($p < .001$), (Figure 5-3b). Additionally, Paretic foot was lateral most compared to non-paretic in the slow walkers. In the fast walkers (speed 0.6 – 0.9 m/s), paretic foot was significantly lateral to the body only relative to speed-matched control foot placement and not non-paretic foot placement ($p< .01$), (Figure 5-3b).

Relationship between Anterior-Posterior Foot Placements Relative to Pelvis, Step Length Asymmetry and Paretic Propulsion

Between-leg asymmetry (anterior foot placement relative to pelvis) and step length asymmetry: Anterior foot placement asymmetry was associated with step length asymmetry ($r = .756$, $p< .001$), suggesting that persons taking asymmetrical step lengths also place their feet asymmetrically relative to pelvis (Figure 5-4). Nonetheless, asymmetry ranges in the two reference frames differed. Note that persons severely asymmetric in the global reference frame (PSR > 90%) showed similar patterns in both the reference frames, however persons showing mild to moderate step length asymmetry (PSR = 60 – 80%) placed their paretic and non-paretic feet more symmetrically with respect to the pelvis (asymmetry range in body reference frame = 55 – 60%), (Figure 5-4).

Anterior-posterior foot placement relative to pelvis and step length asymmetry: Paretic foot placement at TO and non-paretic foot placement at GC strongly correlated with step length asymmetry (PSR) even after controlling for walking speed (Figure 5-5). Note in Figure 5-5, participants taking relatively long paretic steps (PSR > 70%) positioned their paretic foot anterior (opposite to the expected pattern) at TO. Similarly, participants' taking relatively shorter paretic steps (PSR ≤ 40%) positioned their paretic foot much less posteriorly at TO relative to the symmetric group.

Between-leg asymmetry and paretic propulsion: Asymmetrical anterior foot placements relative to pelvis and step length asymmetry were each negatively associated to paretic propulsion ($r = -.584$ and $r = -.520$, respectively), suggesting that both between-leg anterior foot placement asymmetry in body reference frame and step length asymmetry related to percent propulsion generated by the paretic leg.

Relationship between Medial-Lateral Foot Placements Relative to Pelvis, Step Widths, Paretic Weight Support and Dynamic Stability Margin

Lateral foot placement asymmetry relative to pelvis, step widths and percent weight supported on the paretic leg: There was no difference in paretic and non-paretic step widths during hemiparetic walking ($p = .732$) and no relation between step width and either percent weight borne on the paretic leg ($r = .244$, $p = .135$) or lateral foot placements ($r = .235$, $p = .150$). However, lateral foot placement was asymmetric and was negatively associated to the percent weight supported on the paretic leg (Figure 5-6).

Lateral foot placement asymmetry relative to pelvis, step widths and dynamic stability margins: Pearson correlations controlling for walking speed revealed that Lateral foot placement asymmetry was positively associated to paretic DSMmax ($r = .548$, $p < .001$); suggesting that the wider paretic foot placement relative to pelvis than non-paretic foot

placement is related to the wider paretic stability margin. Step width was positively associated with both paretic and non-paretic DSMmax ($r = .520$, $p=.001$ and $r=.360$, $p= .024$; respectively) suggesting that overall a wider step was related to greater stability margin on both paretic and non-paretic legs.

Discussion

The results of this study show that foot placement in a body reference frame is asymmetric in AP and ML directions during hemiparetic walking and this asymmetry related to hemiparetic walking performance.

Anterior-Posterior Foot Placement Relative to Pelvis and its Relationship to Step Length Asymmetry and Forward Progression

Between-phase asymmetry: Overall, relative to pelvis the foot was placed more anterior at GC and less posterior at TO in both controls and participants with hemiparesis at all speeds. Further, participants post-stroke showed greater between-phase asymmetry in the paretic leg than non-paretic legs and also greater asymmetry than controls walking at matched slow speeds. The excursions of the foot were also greater in AP direction for controls even at the matched slow speeds indicating that slower walking speeds alone cannot explain the AP asymmetry in the hemipaertic participants. Further, note that paretic posterior foot position at TO specifically showed increased inter-subject variability (See Figure 5-3a, speeds ≤ 0.3 m/s) suggests that there were persons who never positioned their paretic foot posterior to the pelvis and others who positioned it posterior and yet these two groups were walking at similarly slow walking speeds.

Further, we correlated the posterior foot position at TO to step length asymmetry because we hypothesized that the inter-subject variability in posterior foot position would be well explained by relating the AP foot placements to step length asymmetry and could further help explain the motor control impairments in the asymmetrical groups. Reduced posterior paretic

foot position at TO and anterior non-paretic foot placement at GC relative to pelvis strongly correlated with step length asymmetry. Specifically, in persons taking 'Longer paretic' steps than non-paretic (PSR > 55%), the paretic posterior foot position was either reduced or anterior relative to the pelvis suggesting that paretic leg orientation at pre-swing phase was opposite than that expected (posterior positioning) in this phase. Therefore, while these persons with 'Longer paretic' steps had good stepping ability evidenced by the greater paretic anterior foot placement relative to pelvis than non-paretic (see Figure 5-4), an inability to achieve a more posterior paretic foot position at TO suggests impaired paretic leg extension in the 'Longer paretic' group. A flexed leg orientation at TO versus extended in the paretic pre-swing phase can reduce the mechanical advantage of an extended extremity to propel the trunk forward. This observation is supported by the evidence that the 'Longer paretic' group shows reduced paretic propulsion [194].

In support of our explanation, we would expect that in the 'Shorter paretic' group the posterior paretic foot position would be relatively greater compared to the 'Longer paretic' group since these persons show good paretic propulsion. While all participants in the Shorter paretic group positioned their paretic foot posterior relative to pelvis (Figure 5-5), in the severely asymmetric (PSR < 40%), the posterior foot position relative to pelvis was much reduced similar to some persons in the 'Longer paretic' group. Therefore, we additionally investigated their foot position relative to the trunk COM to evaluate if compensatory trunk lean might be the mechanism utilized to propel the body forward. Relative to the trunk, paretic foot position was much posterior at TO (showing similar excursion as anterior non-paretic foot placement at GC). Since the foot placement relative to trunk depends not only on where the foot is placed but where the trunk is positioned, it is possible that at least some of these persons in the 'Shorter paretic'

group generate good body forward progression during the paretic pre-swing phase by flexing the trunk forward and thereby creating a mechanically advantageous position for the propulsive ground reaction forces generated from the paretic leg to propel the body forward. Therefore, by investigating the foot placement relative to the trunk (in addition to pelvis), compensatory strategies could be diagnosed.

Between-leg asymmetry in anterior foot placements: We also correlated anterior foot placement asymmetry to step length asymmetry (Figure 5-4) to understand the relation between foot placement in a body reference frame to stepping relative to the other foot. Overall, stepping asymmetry in the two reference frames were correlated (for example, persons taking Longer paretic steps relative to non-paretic were also placing their paretic foot more anterior relative to pelvis). However, the symmetry range in the body reference frame was narrower than step length symmetry range and some persons who were asymmetric taking Longer paretic step lengths than non-paretic (PSR = 55 -65%) were symmetric in the body reference frame. Interestingly, others who were taking symmetrical step lengths were actually asymmetric relative to pelvis taking shorter paretic steps than non-paretic. The ability to place the foot further anterior to the pelvis suggests good stepping ability and the observation that the borderline symmetric persons actually took shorter paretic steps relative to pelvis suggests their impaired paretic stepping ability. Therefore, we suggest that since some persons [mildly asymmetric (longer paretic) and symmetric] step lengths changed their asymmetry in a translating body reference frame, additional investigation of foot placement pattern relative to pelvis might be necessary to classify them as symmetric or asymmetric.

In summary, by relating the foot placement and position relative to the pelvis to step length asymmetry we are able to propose some underlying motor control mechanisms in the

asymmetrical groups. 'Longer paretic' group showed poor forward body progression likely due to altered initial conditions of the leg in their pre-swing phase, whereas the 'Shorter paretic' group showed poor stepping ability due to the inability to place the paretic foot further anterior to the pelvis. Further, the good forward progression at least in some participants in the 'Shorter paretic' group might be compensatory from leaning the trunk forward in the pre-swing phase of their paretic gait cycle.

Medial-Lateral Foot Placement Relative to Pelvis and its Relationship to Weight Supported on Paretic Leg and Dynamic Stability Margin

There was no difference in ML foot placement at GC and TO (i.e. no between-phase asymmetry). However, between-legs, the paretic foot was placed wider relative to pelvis compared to non-paretic and speed-matched control in participants with hemiparesis walking at slow and moderate speeds. Further, this lateral foot placement asymmetry between-leg decreased with increasing walking speed during hemiparetic walking (Figure 5-3b). Furthermore, we found that the lateral foot placement asymmetry was strongly correlated with both weight support and paretic dynamic stability margin. We quantified the percent weight supported on paretic leg as the percentage of Vertical Ground reaction force on the paretic leg during stance compared to the both legs. Overall paretic leg supported lesser weight compared to non-paretic evidenced by the lower percentages than 50 (Figure 5-6). This weight-bearing asymmetry is consistently reported in the post-stroke population [19, 154]. However, the relationship between lateral foot placement and weight supported on the legs has not yet been reported in this population.

While we expected that weight supported on the leg would be related to lateral foot placement, our results specifically show that the asymmetry in lateral foot placement relative to pelvis (i.e., wider paretic steps relative to pelvis than non-paretic) related to the weight supported on the paretic leg during stance. On the other hand, step widths were unrelated to the paretic

weight support suggesting that investigation of lateral foot placement asymmetry relative to pelvis specifically revealed the impaired paretic leg weight support in persons taking wider paretic steps relative to body. Therefore, we suggest that close observation of the lateral paretic foot placement relative to the body in comparison to lateral non-paretic foot placement can provide useful insights to the clinicians regarding the amount of weight supported on the paretic leg.

Moreover, we found that the lateral foot placement asymmetry specifically related to the dynamic stability margin on the paretic side. We calculated the dynamic stability margin specifically for each foot (at initial contact) as the ML distance between xCOM and foot COM at the instance when xCOM reached its maximum value in 1st double-support (Table 5-1). Foot placements at heel-strike have earlier shown to be a linear function of COM displacement and velocity and shown to be the most important factor defining stable gaits [116, 117, 134] suggesting the importance of foot placement to dynamic stability. Specifically, in this study we chose to use DSMmax since it physiologically relates to the point of maximum instability. We also calculated DSM at other instances: first instance of ground-contact (DSM-GC) and at every 20% of 1st double-support. Evaluation of DSM at every 20% of the gait cycle revealed that there was no significant difference between DSM values as the 1st double-support phase progressed suggesting that the DSM doesn't change substantially across the double-support phase. Further, DSM-GC and DSMmax revealed the same pattern relative to foot placement, although correlations were stronger for DSMmax and foot placement. Note that, we did not use the entire area enclosed between foot placements (i.e., base of support) to define the DSM since we wanted to investigate how foot placement on individual legs affected the margin established at each foot placement. We found that wider paretic foot placement relative to pelvis than non-paretic

specifically related to wider stability margin established on the paretic side. However, there was no relation between the lateral foot placement asymmetry and the non-paretic stability margin suggesting that the wider paretic foot placement relative to non-paretic may specifically establish a wider stability margin at paretic foot placement. As expected, step width related to both paretic and non-paretic stability margins indicating that a wider step in general related to a wider stability margin. A wider margin specifically on one side can predispose lateral instability since the COM was closer to the margin of the base of support on one side than other. Since there was no relation between step width and lateral foot placement asymmetry, persons taking wider steps may have a wider or narrower margin on the paretic side relative to non-paretic and it may be difficult to identify those persons with lateral instability. Further, while the wider step on the paretic side related to decrease weight support it increased the stability margin on the paretic side indicating that ML asymmetry might be compensatory to increase the dynamic stability. Therefore, we suggest that lateral foot placement relative to pelvis is a useful outcome measure to quantify both paretic leg weight support and dynamic stability.

Limitations

There are some limitations in this study. The current study was conducted over the treadmill and foot placement relative to body might differ overground. Nonetheless, it is expected that while absolute foot placement (in isolation) might be altered (i.e., longer versus shorter step lengths) while walking overground, its relation to the body movements remain consistent suggesting the functional relevance of foot placement relative to body. This hypothesis could be tested in future studies by exploring the step-by-step variability in foot placement relative to body and whether this changes from treadmill to overground walking. Hip and knee kinematics were not explored in the current study. Since the foot is under multi-segmental and end-point control, it is expected that the foot kinematics relative to body will be

the end result of different combinations of hip and knee kinematics such that investigation of foot kinematics relative to body might be a relatively easy method to understand post-stroke walking from a control model viewpoint.

Conclusions

Post-stroke gait is asymmetric when quantifying foot placement relative to pelvis in both AP and ML directions. We suggest that for the AP direction, characterizing stepping relative to the other foot (i.e., step length) was appropriate as outcomes to quantify asymmetrical performance and forward progression because stepping relative to the other foot and stepping in a body reference frame were associated. Nonetheless, since some borderline asymmetrical (PSR = 55 – 65%) and other symmetrical persons show differing performance in the translating reference frame, it may be additionally useful to investigate foot placement relative to pelvis in these persons. Further, we could understand motor control mechanisms in the asymmetrical sub-groups (impaired initial conditions in the 'Longer paretic' group versus impaired stepping ability in the 'Shorter paretic' group) by investigating the foot position patterns relative to pelvis. For the ML direction, we suggest using foot placement relative to pelvis as an outcome to characterize hemiparetic performance. A wider foot placement of the paretic foot relative to pelvis than non-paretic can quantify the reduced weight supported on the paretic leg and lateral instability. Overall, we were able to better evaluate asymmetrical performance during hemiparetic walking by investigating foot placement and position in a body reference frame that was not revealed when investigating stepping relative to other foot. Therefore, we suggest that biomechanical analyses quantifying stepping performance in impaired populations should investigate foot placement in a body reference frame (especially in the ML direction) to understand motor control during gait.

Table 5-1. Definition of study variables

Foot placement and position relative to body
Anterior-Posterior
AP Foot placement relative to pelvis: Distance in AP direction from foot COM to pelvis COM at the initial foot placement (ground-contact in the 1st double-support).
AP Foot position relative to pelvis: Distance in AP direction from foot COM to pelvis COM at the terminal foot position (toe-off in the 2nd double-support phase).
Medial-Lateral
ML Foot placement relative to pelvis: Distance in ML direction from foot COM to pelvis COM at the initial foot placement (ground-contact in the 1st double-support).
ML Foot position relative to pelvis: Distance in AP direction from foot COM to pelvis COM at the terminal foot position (toe-off in the 2nd double-support phase).
Stepping relative to the other foot
Step length: Distance in AP direction from leading mid-foot to trailing mid-foot at the the initial foot placement (ground-contact in the 1st double-support).
Step width: Distance in ML direction from leading mid-foot to trailing mid-foot at the the initial foot placement (ground-contact in the 1st double-support).
Walking performance measures
Step length asymmetry: calculated as a Paretic step ratio (PSR = Paretic step length / (Paretic + Non-paretic step length) and expressed as a percentage.
Paretic propulsion: Paretic propulsive impulse / (Paretic + Non-paretic propulsive impulse).
Percent weight supported on the paretic leg: Paretic average vertical force / (Paretic + Non-paretic average vertical force) expressed as a percentage.
Dynamic stability margin: ML Distance between the extrapolated COM (xCOM = COM displacement + COM velocity) and foot COM.

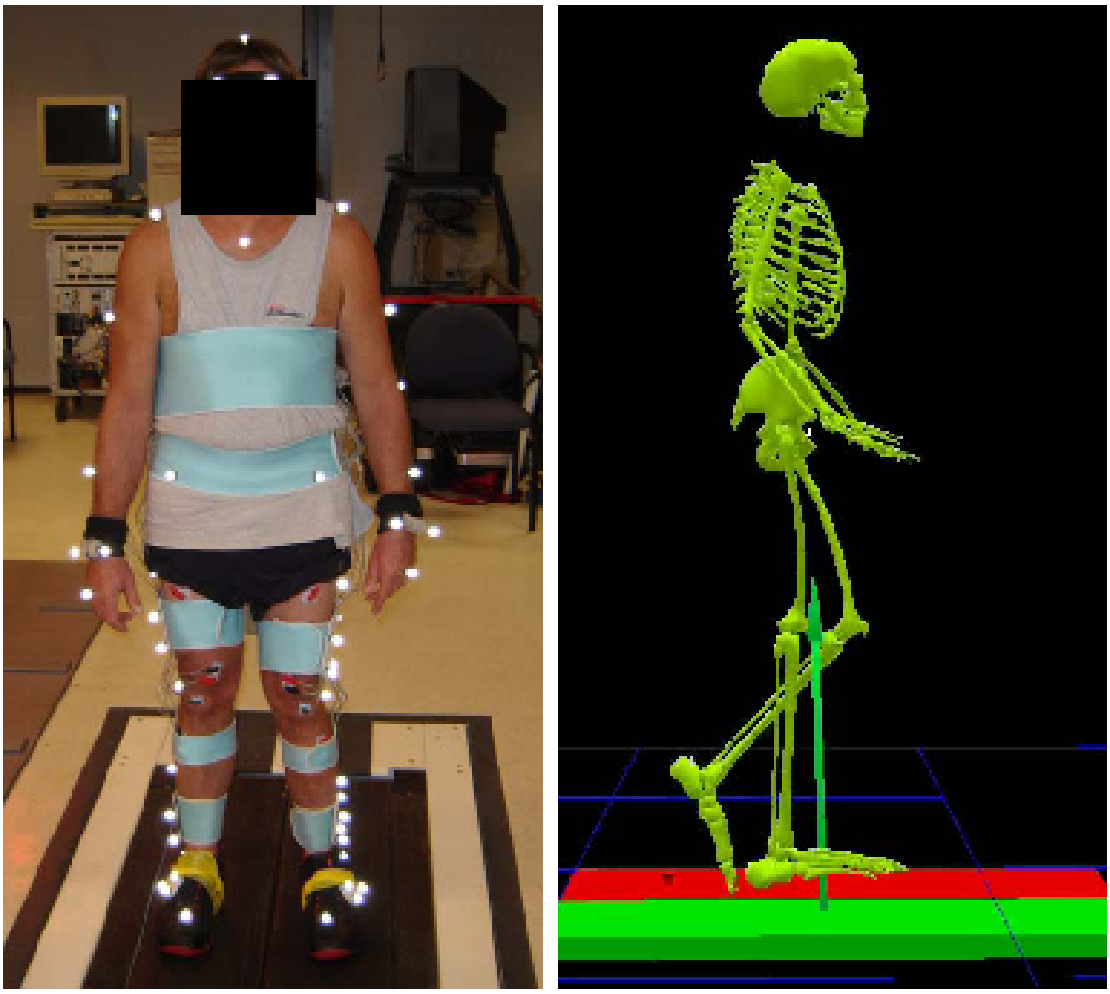


Figure 5-1. Illustration of marker positions for kinematic data collection and the SIMM model generated

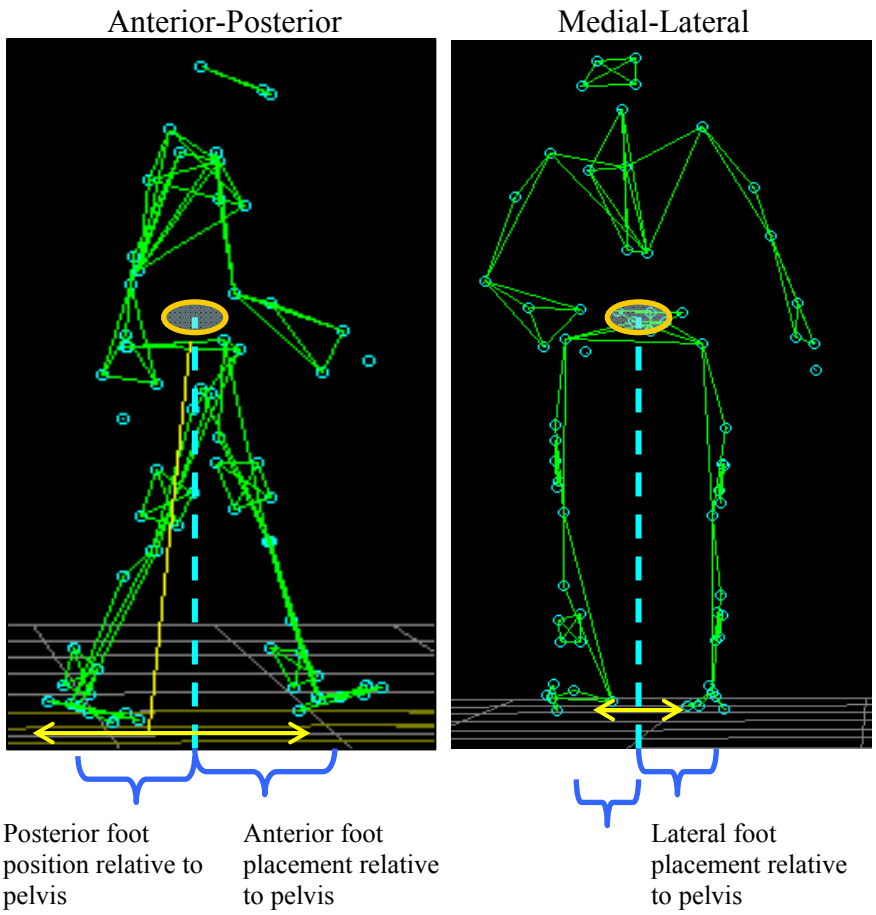
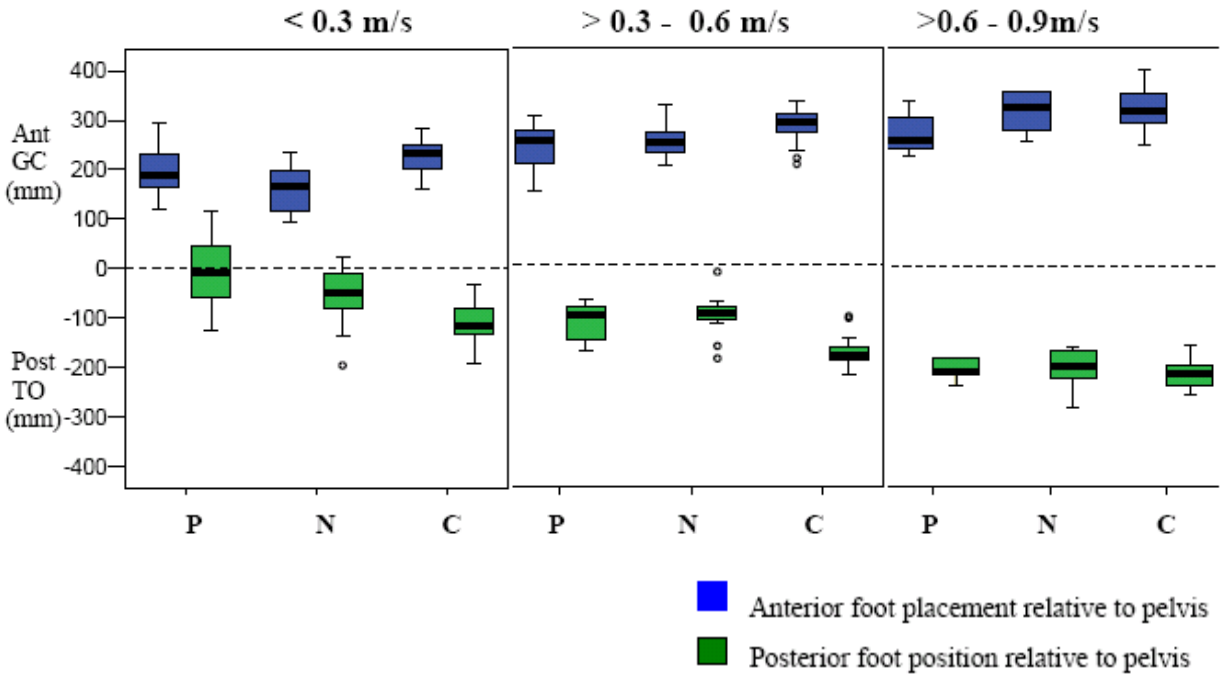


Figure 5-2. Calculation of anterior-posterior and medial-lateral foot placements relative to pelvis

This figure presents the foot placement and position variables calculated relative to pelvis. These variables were calculated as the AP and ML distance between foot COM and pelvis COM. Note, the yellow arrows present the step length and step width calculated relative to other foot.

a. Anterior-Posterior foot placement relative to pelvis



b. Medial-Lateral foot placement relative to pelvis

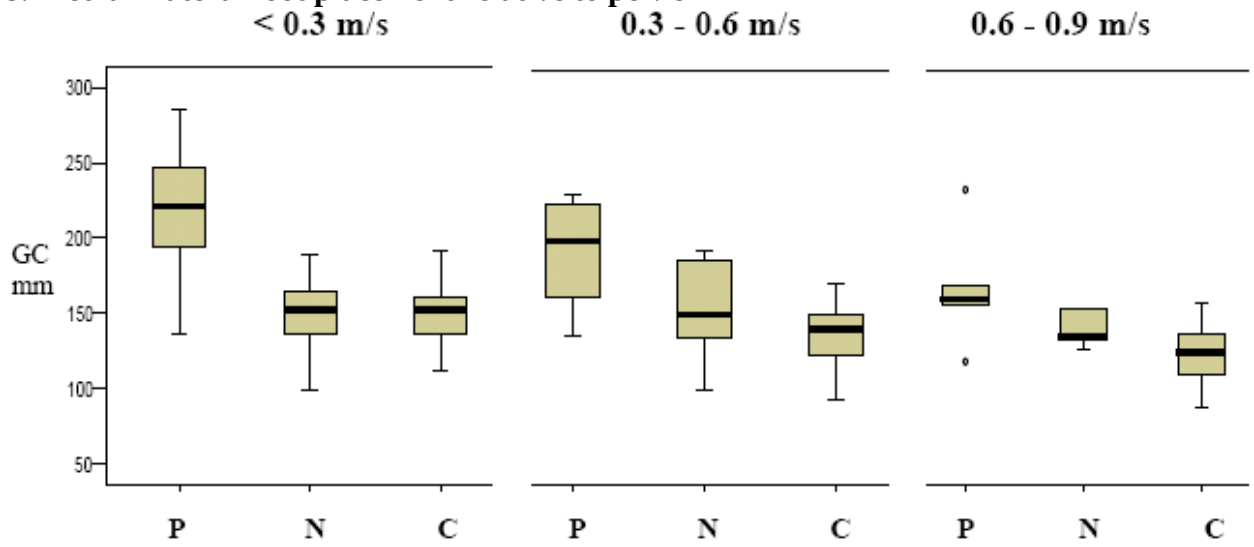


Figure 5-3. Foot placement relative to pelvis during hemiparetic and healthy gait

Abbreviations: GC – Ground contact at 1st double-support (initial foot placement), TO – Toe-off at 2nd double-support (terminal foot position), P- Paretic foot, N-Non-paretic foot, C-Control foot. The box plots indicate the range in the data. The central horizontal line is the median of the sample. The length of the box indicates the inter-quartile range with the upper and lower boundaries of the box indicating the upper and lower quartile, respectively. Circles represent sample values that statistically indicate outlier or extreme values (by SPSS software).

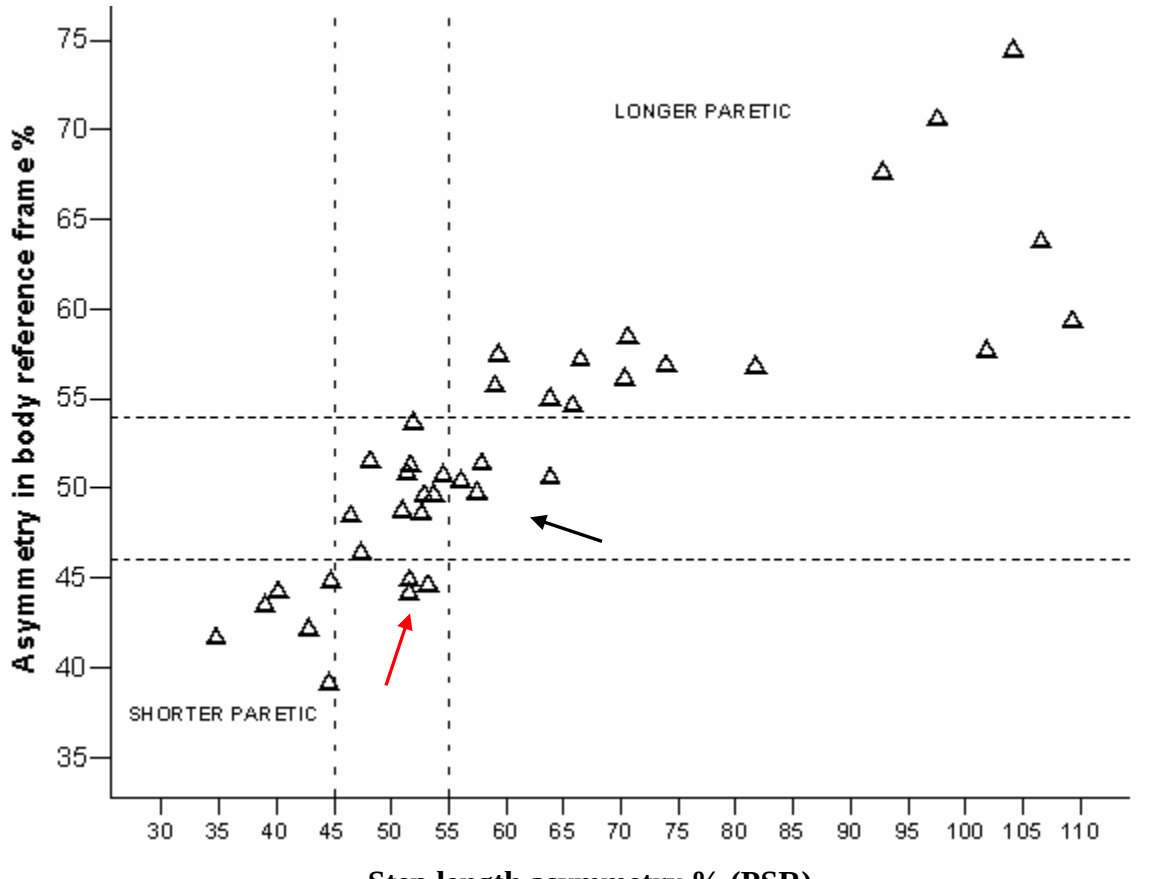


Figure 5-4. Relationship between anterior foot placement asymmetry relative to pelvis and step length asymmetry in participants with hemiparesis

This figure shows the relationship between asymmetry in foot placements relative to pelvis (body reference frame) and step length asymmetry (relative to the other foot). The vertical dashed lines indicate the step length symmetry ranges ($45 \leq PSR \leq 55$) and the horizontal dashed lines indicate symmetry ranges in the body reference frame ($46 \leq PSR \leq 54$) calculated similarly from healthy controls. Persons above the range take "Longer paretic" steps than non-paretic and those below the range take "Shorter paretic" steps than non-paretic. Note that, four persons (black arrow) taking longer paretic than non-paretic step lengths placed their paretic and non-paretic feet symmetric with respect to the pelvis. On the other hand, three persons (red arrow) who were taking symmetric step lengths place their paretic foot closer to pelvis than non-paretic (i.e. shorter paretic steps in the body reference frame).

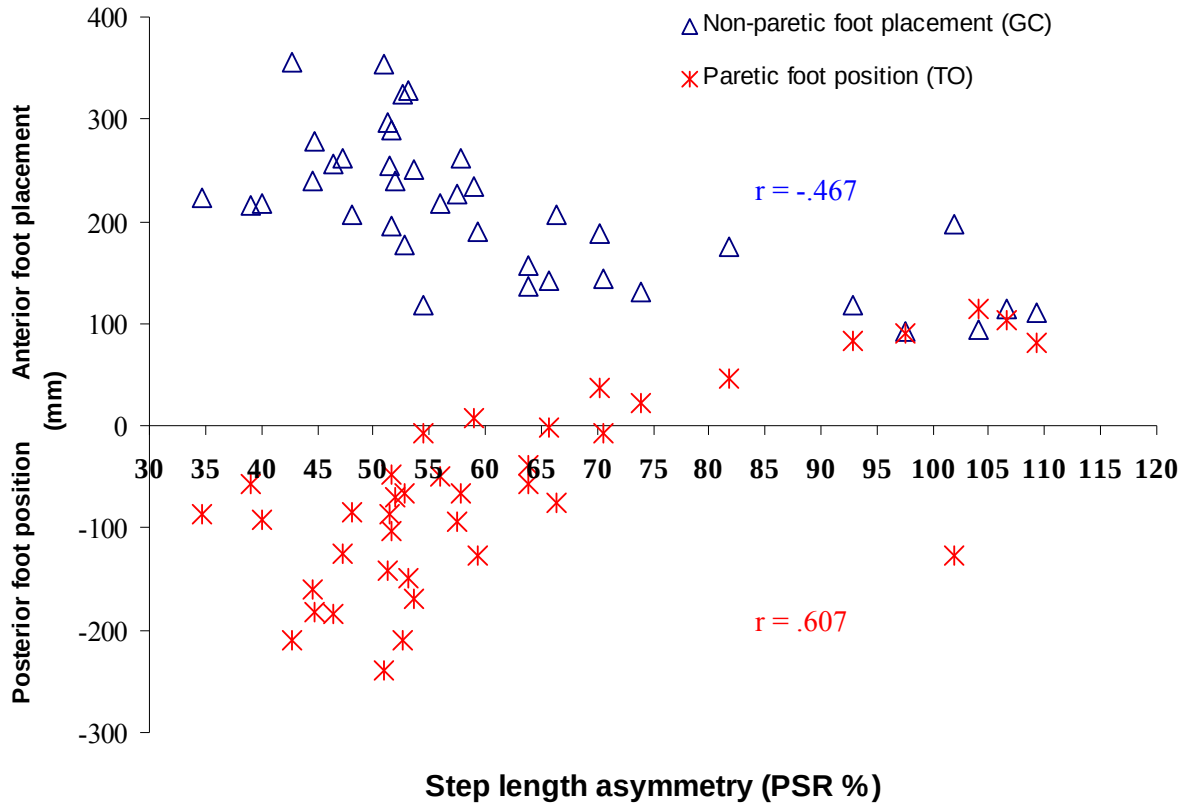


Figure 5-5. Relationship between step length asymmetry and anterior-posterior foot placement relative to pelvis in participants with hemiparesis

This figure shows the relationship between step length asymmetry and between-phase asymmetry in foot placements relative to pelvis. The step length symmetry ranges ($45 \leq PSR \leq 55$) were calculated similarly from healthy controls. Note the posterior paretic foot position in persons taking "Longer paretic" steps ($PSR > 55$). Some persons with severe asymmetry ($PSR > 70\%$) never position their paretic foot posterior to the pelvis in this phase of their gait cycle (2nd double support). Similarly, severely asymmetric persons taking "Shorter paretic" steps ($PSR < 40\%$) place paretic foot much anterior to pelvis than posterior at toe-off. Also, compare the paretic posterior and non-paretic anterior placement relative to pelvis between the symmetric and asymmetric persons.

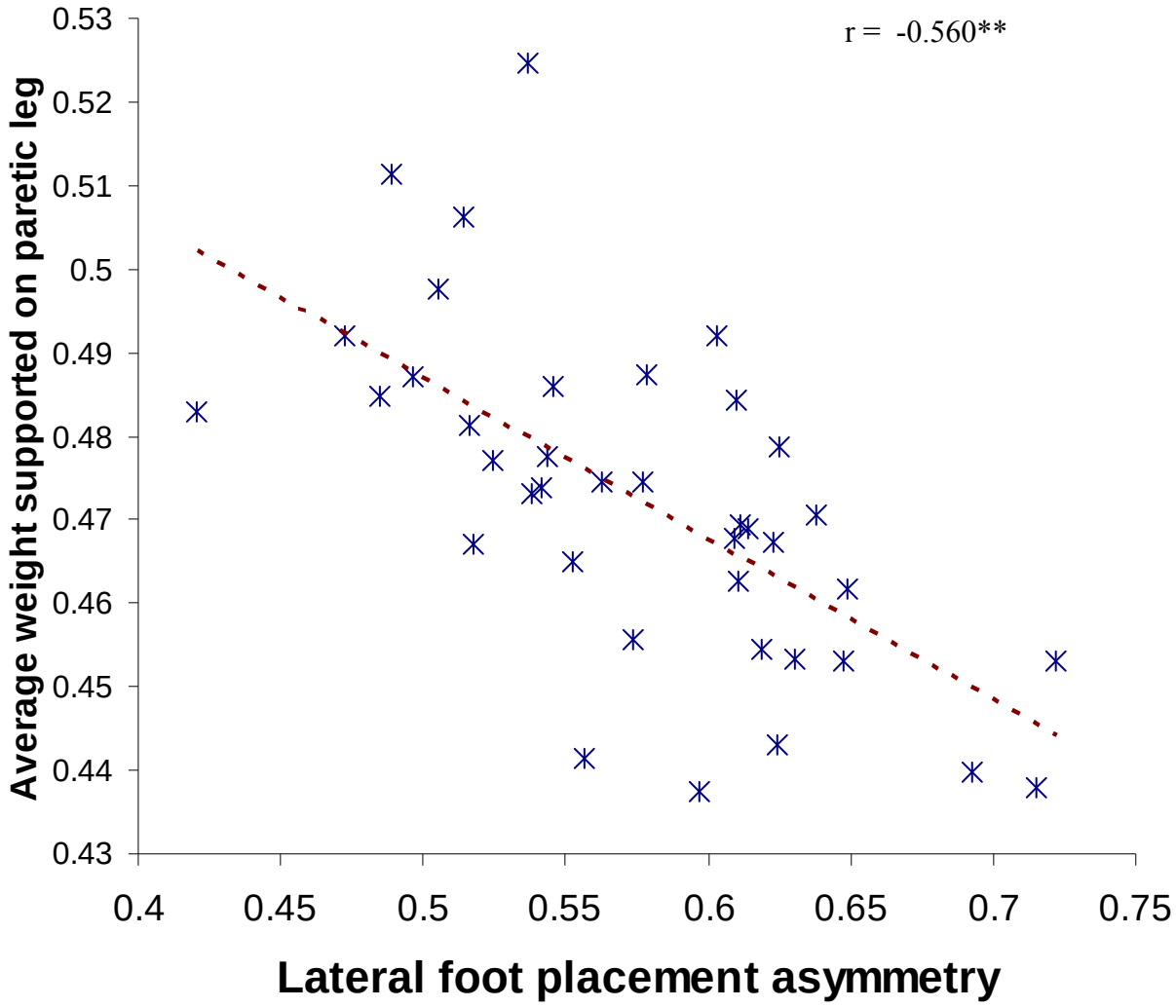


Figure 5-6. Relationship between paretic and non-paretic lateral foot placement asymmetry relative to pelvis and percent weight supported on the paretic leg

Note that greater lateral foot placement asymmetry (i.e., paretic foot placed wider relative to pelvis than non-paretic foot placement relative to pelvis) lesser the weight supported on the paretic leg during the stance phase.

CHAPTER 6
EVALUATION OF STEP LENGTH GENERATION DURING POST-STROKE HEMIPARETIC WALKING USING A NOVEL METHODOLOGY OF STEP-BY-STEP VARIABILITY IN GAIT DATA

Introduction

While hemiparetic walking is asymmetric, the underlying mechanisms responsible for step length generation during walking are not clearly understood in this population. Step lengths are reported to be shorter, asymmetric and variable from step to step during hemiparetic walking compared to healthy walking [5, 18, 69, 198]. Shorter step lengths are commonly associated to the slower walking speeds post-stroke [53]. Nonetheless, it is likely that underlying sensorimotor and muscular impairments post-stroke directly influence the attained step lengths beyond changes due to the slower walking speeds. In particular, step lengths are also asymmetric during hemiparetic walking with the direction of asymmetry being inconsistent. Reports suggest that there are a greater number of persons taking longer paretic steps than non-paretic and few take relatively shorter paretic steps than non-paretic [69, 194]. A sub-category of persons also walk with relatively symmetric steps [194]. The inter-subject variability in step length asymmetry suggests that the underlying neuromotor mechanisms controlling paretic step lengths could vary within these sub-groups of the hemiparetic population. Furthermore, there is considerable variability in step lengths on a step-by-step basis within individual stroke participants, with greater step-to-step variability reported in the more severe participants (unpublished data). This suggests that evaluation of the neuromotor mechanisms controlling step length generation needs to account for the subject-specific step-by-step variability in step length generation. Therefore, in this study, mechanisms of step length generation during hemiparetic walking were evaluated using a novel methodology that incorporates this within-subjects step-by-step variability in step lengths.

Several potential contributing factors are likely to affect attained step lengths. Current evidence indicates that step length generation can be influenced primarily by the: 1) initial conditions at the initiation of swing phase of the ipsilateral leg, 2) biomechanics of the early (primarily accelerating) and late swing phase (primarily decelerating) of the ipsilateral leg, and 3) contralateral stance leg ground reaction forces during its early and late single-support phase (usually braking and propulsive, respectively, in healthy subjects), occurring simultaneously with the ipsilateral swing phase.

Initial conditions of the leg (e.g. kinematics, kinetics and muscle activity in the pre-swing phase) are suggested to define the resulting swing leg trajectory [173]. For instance, dynamic simulations of swing phase in healthy gait performed in the absence of muscle joint torques approximated normal knee kinematics by selecting the initial angular velocities and positions alone [164, 203], implying that the initial angular velocity is an important determinant of kinematics during swing. Initial conditions prior to swing also provide the energy to the swinging leg [204, 205]. Therefore, the strong influence of initial conditions on the ipsilateral swing phase of the gait cycle, can indirectly affect where the leg is placed at the end of the swing phase. Nonetheless, while the initial conditions of the leg prior to swing phase should be partially predictive of swing phase characteristics, swing phase muscle activity is likely to either augment the leg flexion during swing (presumably by leg flexors) or decelerate the leg to terminate the swing (e.g., hamstrings muscle activity). Therefore, events occurring specifically during swing phase of the gait cycle can independently control the eventual step length. In support, Varraine et al. (2003) showed that when healthy participants were asked to voluntarily modulate their step lengths, they lengthened the time the leg was held in the swing phase [182]. Consequently, they proposed that controlling the swinging leg trajectory by altering the swing phase parameters

enabled the healthy participants to step longer. Therefore, while initial conditions prior to swing phase can indirectly control step lengths (by its influence on the swing phase), events during the swing phase present potentially direct mechanisms that can control step lengths. Lastly, contralateral stance leg ground reaction forces occurring at the same instance as ipsilateral swing phase can influence step length generation. Recent work showed a strong negative relationship between propulsive force asymmetry and step length asymmetry that highlights the causal relationships between ground reaction forces and step lengths [194]. In particular, the finding that persons who take longer paretic steps than non-paretic generate lesser paretic leg propulsion; suggests that the greater compensatory non-paretic leg propulsion might be causing the trunk to progress further forward as the paretic leg is stepping, thereby resulting in the relatively longer paretic steps. Overall, current literature suggests several potential indirect and direct mechanisms related to the initial conditions of the leg, swing phase and contralateral stance phase that might influence step length generation.

In this study, we selected kinematic and kinetic measures that independently describe each of these phases of the hemiparetic gait cycle. Leg orientation at toe-off, pelvis velocity at toe-off and ankle joint center velocity at toe-off determined the initial state of the leg and body before the leg begins to swing. Hip Impulse in early and late swing and corresponding contralateral Anterior-posterior (AP) ground reaction force (GRF) impulses were chosen to represent the events in the ipsilateral swing phase and corresponding contralateral stance phase, respectively. We hypothesized that the kinematic and kinetic measures that describe each of these phases of the hemiparetic gait cycle would relate to step lengths and explain step length variability within-subjects. In particular, we hypothesized that contralateral AP GRF impulses would be a strong predictor of step length variability in majority of persons. Whereas, hip impulse in ipsilateral

swing will be a strong predictor of step length variability in persons taking longer paretic steps than non-paretic. Regression models were built for individual subjects using gait data from each step because we wanted to evaluate the kinematic and kinetic parameters that best predicted the step length variability within-subjects. In summary, the purpose of this study was to comprehensively evaluate the underlying mechanisms of step length generation during hemiparetic gait by determining the predictors that explain the step length (step-by-step) variability within-subjects.

Methods

Participants

Participants were a convenience sample of thirty-eight persons with chronic hemiparesis (Average Age = 60.21 ± 12.32 years, 20 men, 19 left-side hemiparesis) and twenty similarly-aged healthy participants (Age = 66.15 ± 10.03 years, 4 men) evaluated at the VA-UF Human motor performance laboratory, VA Medical center at Gainesville Florida. Table 6-1 presents the individual subject characteristics for the hemiparetic participants. Healthy subjects' data were used to calculate the step length symmetry ranges for the hemiparetic participants. Inclusion criteria for the participants were: hemiparesis secondary to a single onset unilateral stroke; ability to ambulate independently with or without an assistive device over 10 m on a level surface; ability to walk on a regular basis at least at home; absence of significant lower extremity joint pain and major sensory deficits; absence of significant lower limb contractures and no significant cardiovascular or respiratory symptoms contraindicative to walking. Participants from the study were excluded if they had any orthopedic or neurologic conditions in addition to stroke, had significant musculoskeletal problems other than stroke that limit hip and knee extension or ankle plantar flexion to neutral, or were unable to provide informed consent. All participants in the

study signed a written informed consent and Institutional Review Board of University of Florida approved the protocol.

Procedures

Retro-reflective markers were attached to the participant to collect bilateral 3D kinematics using a 12 camera VICON motion analyses system. Markers were attached to the head (top, left, right, front and back), trunk (C7, T10, clavicle, sternum and right scapula), upper extremity (bilateral shoulders, elbows and wrists), lower extremity (bilateral knees and ankles) and the feet (tip of the toes, left and right side of forefoot, midpoint of forefoot and calcaneus). Clusters of reflective markers attached to rigid bodies were also located on the pelvis, bilateral thighs, shanks and feet. A fixed laboratory coordinate reference frame was created within the VICON system that was placed at the left corner of the laboratory.

At the beginning of the test session, controls and participants with hemiparesis walked for 2-3 trials across a 12 ft long instrumented mat (GaitRite) at their self-selected walking speeds to collect over ground spatiotemporal parameters of steps and estimate their overground walking speeds.

Subsequently, participants with hemiparesis walked on an instrumented split-belt treadmill (TECMACHINE) for three trials at their self-selected treadmill walking speed. The treadmill self-selected speeds were 10 – 30% slower than that over ground for participants with hemiparesis. All participants completed three 30-second walking trials without use of an assistive device or ankle-foot orthosis. A safety harness mounted to the laboratory ceiling was worn across the shoulders and chest to protect the participants in the event that they loose balance. Note, that no bodyweight was offloaded by the harness. Additionally, a physical therapist closely guarded the participants as they walked over the treadmill (although no manual support was provided by the therapist). To optimize capture of steady state data on the treadmill,

each subject walked for 10 s prior to each of the 30 s of data collection. Three-dimensional Ground Reaction Forces (GRFs) were measured from each half of the treadmill along with kinematic data that were collected during the walking trials. Refer to Figure 6-1 showing a participant walking on the treadmill as kinematic and kinetic data were collected.

Data Analyses

Kinematic data: Raw kinematic data was low-pass filtered using a fourth-order zero-lag Butterworth filter with a 10 Hz cutoff frequency. The joint center and anatomical trajectories was fitted to an eight-segment musculoskeletal model generated using SIMM (MusculoGraphics, Inc.) consisting of a trunk (including the mass of the torso, head and arms), pelvis and legs for each subject. Each lower extremity consisted of a thigh, shank and foot. The anthropometrics and inertial properties were based on that of de Leva [200]. Joint positions and velocities were calculated within the SIMM Motion Module. The kinematic variable calculated in this study included the Leg orientation at toe-off that was defined as the angle between the vector from the pelvis COM to the foot COM and vertical. Leg orientation is negative when the foot is posterior to the pelvis (Figure 6-2).

Kinetic data: Three-dimensional GRFs were sampled at 2000 Hz. Ground reaction force data was low-pass filtered using a fourth-order zero-lag Butterworth filter with a 20 Hz cutoff frequency. The SIMM Motion Module (MusculoGraphics, Inc.) was used to perform a standard inverse dynamics analysis to determine the inter-segmental joint moments. Kinetic data (GRFs and joint moments) were normalized by subject body weight and to 100% of the paretic leg gait cycle (paretic heel strike to paretic heel strike). Kinetic data included the pelvis velocity at toe-off, ankle joint center velocity at toe-off, Hip impulse and AP impulse (Figure 6-2). Hip impulse was calculated as the time integral of the hip moment during early swing and late swing. Positive moment indicated flexor moments and negative moments indicated extensor moments. AP

impulse was calculated as the time integral of the AP GRF during the single-support phase of the contralateral leg occurring at the same instance as the ipsilateral swing phase (Figure 6-2). Positive impulses denoted propulsive (anterior) impulse and negative impulses denoted braking (posterior) impulse.

Sub-division of the gait cycle: The stance phase of the gait cycle was subdivided into Bins to correspond with: the first double support phase following heel strike (Bin1), the first 50% of single leg stance (Bin 2), the second 50% of single leg stance (Bin 3) and the second double support phase during paretic pre-swing (Bin 4). Bins 1 through 4 were defined from GRF records. Additionally, the swing phase was sub-divided into two phases: the first 50% of swing phase (Bin 5) and the second 50% of swing phase (Bin 6). The last instance of Bin 4 of stance phase was toe-off and the initial condition variables were calculated at this instance. The early and late hip impulses were calculated in Bin 5 and Bin 6 of swing phase. The early and late contralateral AP impulses were calculated in Bin 2 and Bin 3 of stance phase.

Statistical Analyses

Stepwise Regression: Stepwise model-building techniques for regression designs with a single dependent variable are described elsewhere [206]. The basic procedures involve (1) identifying an initial model, (2) iteratively stepping, that is, repeatedly altering the model at the previous step by adding or removing a predictor variable in accordance with the 'stepping criteria', and (3) terminating the search when stepping is no longer possible given the 'stepping criteria', or when a specified maximum number of steps has been reached.

In this study, step length was the dependent variable and the selected kinematic and kinetic variables were the independent variables. A stepwise regression model was used to select critical predictors (from the 7 hypothesized variables) that could account for the variability in step lengths for individual participants and indicate how much variance in step length was explained

by these predictors (indicated by the R2 values). Two regression models were built for each participant's data to predict paretic and non-paretic step length variability each. Therefore, overall 76 regression models were built for the 38 study participants. The step-wise regression was conducted by pooling individual steps from all trials together for individual participants. Note that for each participant different numbers of steps were analyzed (see Table 6-2 for the minimum number of steps analyzed) since each participant walked at their self-selected speed at a specific cadence. Only data from good steps of the participant were utilized (i.e., in the event that there was a crossover of a step these data were deleted for this step and the corresponding step). For some participants, there was a difference of 2-3 steps between paretic and non-paretic legs that depended on which leg took the first and last step.

The 'stepping criteria' for this study was a significant relationship between the predictor variable and step length at $p < .05$. The chosen predictors could best explain the generated step lengths since they covaried (related) with step lengths on a step-by-step basis. That is; greater or lesser value of the critical predictor variable that was chosen related to longer or shorter step length in an individual participant and therefore explained the variability in step length generation in an individual participant. Entry p value for the stepwise regression model was set at less than 0.05 and removal p value was set at less than 0.10.

Determination of step length asymmetry: Step length asymmetry groups were determined based on the overground asymmetrical patterns in healthy control participants using a Paretic step ratio [PSR = Paretic step length/(Paretic + Non-paretic step length)] and expressed as a percentage [194]. Asymmetry in the hemiparetic participants was characterized based on symmetry ranges calculated from similarly-aged healthy participants, as follows: 'Longer

paretic' steps than non-paretic (PSR > 52.5), 'Shorter paretic' steps than non-paretic (PSR < 47.5) and 'Symmetric' step lengths (47.5 ≤ PSR ≤ 52.5).

Results

Average gait characteristics for individual participants with hemiparesis are presented in Table 6-2. There was no difference in step-by-step variability between paretic and non-paretic step lengths ($p = .752$). Majority of participants had paretic and non-paretic step length variability less than 5 cm (Figure 6-3).

In general, greater variability was explained for the non-paretic step lengths than paretic for individual participants (compare the difference in R2 values for individual participants in Tables 6-3 and 6-4). The explanatory power of the models was also unrelated to the absolute magnitude of step-by-step variability (standard deviation) in paretic and non-paretic step lengths ($p = .233$). There were 2 participants for whom no predictor variables could explain the paretic step length variability at $p < .05$. In comparison, there was no participant with unexplained non-paretic step length variability.

Predictors of Step Length Variability and the Differences in Selected Predictor Variables across the Asymmetrical Sub-Groups

Shorter paretic group

For the paretic step length models, non-paretic AP impulse in late swing (APImp LS) was the most commonly selected variable related to paretic step lengths (Table 6-3). Further, it showed a positive association with step lengths wherever it was chosen. However, the non-paretic APImp LS occurring during paretic stepping were braking forces for several participants in this group (see Figure 6-4). Note that Paretic Hip Impulse in early and late swing was not selected as a predictor variable related to paretic step length variability in this group of participants.

For the non-paretic step length models, paretic APImp LS was the most commonly selected variable related to non-paretic step lengths (Table 6-4). The paretic APImp LS occurring during non-paretic stepping was propulsive in this phase. Unlike for the paretic step length models, non-paretic Hip Impulse in early and late swing showed a significant positive relation to non-paretic step lengths in 3/6 participants. Specifically, the non-paretic hip impulse was flexor in these participants.

Symmetric group

For the paretic step length models, non-paretic APImp LS (in 7/10 participants) and paretic leg orientation at toe-off (in 6/10 participants) were the most commonly selected variables related to variability in individual participants. While non-paretic APImp LS was positively related to paretic step lengths (Table 6-3), paretic LO was negatively related to step lengths. Further, non-paretic APImp LS was propulsive in all but one participant (H12) and paretic leg orientation was extension in all participants.

For the non-paretic step length models, non-paretic ankle joint center (AJC) velocity at toe-off (7/10 participants), non-paretic Hip Impulse in early swing and paretic APImp LS (6/10 participants) were most commonly selected variables that related to non-paretic step length variability. Further, all these variables were positively related to non-paretic step lengths. Specifically, hip impulse was flexor in early swing and paretic AP impulse was primarily propulsive in late swing.

Longer paretic group

For the paretic step length models, paretic hip impulse in early swing and non-paretic AP impulse in early swing (APImp ES) were the most commonly selected variables that related to paretic step length variability (12/22 participants selected these variables). Note that, APImp in this group was more commonly selected in the early swing than late compared to the Shorter and

Symmetric persons where APImp LS was more commonly selected (Table 6-3). However, paretic hip impulse in early swing showed a stronger relation with paretic step length in participants in this group compared to those selectively in the Shorter paretic group (indicated in Table 6-3 by significant relations between, also refer to Figure 6-5).

Both paretic hip impulse in early swing and non-paretic APImp ES were positively related to paretic step lengths (Figure 6-5). Note that paretic hip impulse in early swing was flexor in all participants. Non-paretic APImp ES was primarily propulsive in this phase in majority of the participants (only 2/22 showed entirely braking in this phase compared to most participants in Shorter paretic group who showed braking forces, Figure 6-4).

For the non-paretic step length models, paretic APImp LS and non-paretic hip impulse in early swing were most commonly selected as predictors that related to non-paretic step length variability (15/22 participants showed significant relations for both of these variables). Both predictors were positively related to non-paretic step lengths. Non-paretic hip impulse was primarily flexor. Paretic APImp LS was primarily braking in 10/22 participants where it showed a significant relationship. Non-paretic AJC velocity at toe-off (14/22 participants) was also among the commonly selected predictors that related to non-paretic step length variability.

Discussion

The purpose of this study was to comprehensively evaluate the mechanisms underlying step length generation during hemiparetic gait by determining those variables that strongly relate to step length variability within-subjects. While recent work suggests potential mechanisms that might be related to step lengths during hemiparetic gait [194], there has been no systematic investigation to understand mechanisms underlying step length generation during hemiparetic walking at a self-selected pace. To improve walking outcomes after a hemiparetic stroke, rehabilitation therapists commonly focus on increasing the step lengths. Determining the most

significant predictors that relate to step-by-step variability in step lengths post-stroke, can provide insights on those parameters that can be targeted to change step lengths (i.e., increase step lengths or even reduce step length asymmetry).

Overall, the range of variance explained by the regression models suggested that our hypotheses that the events occurring during a) the initial conditions prior to ipsilateral swing phase, b) ipsilateral swing phase, and c) contralateral stance phase relates to step lengths. Our current methodology (of individual subject regression models) enabled us to determine several mechanisms underlying step length generation and also helped us to delineate differential mechanisms in sub-groups of this asymmetrical population. Specifically, within-subject analyses as used in our study is specific to our study question where we want to understand kinematic and kinetic variables that relate to step lengths in individual participants.

Contralateral Stance Leg Ground Reaction Force (AP Impulse during Ipsilateral Swing) is a Significant Predictor of Step Length Variability

In majority of participants, contralateral AP impulse (propulsion mostly) occurring at the same instance as ipsilateral swing was the most commonly selected variable that was related positively to step length variability, suggesting that an increase in the contralateral AP impulse resulted in a longer ipsilateral step length in individual participants (Table 6-3, 6-4). However, sub-groups showing asymmetrical performance showed a difference in selection of either contralateral AP impulse during its early versus late phase. In persons taking 'Symmetric' and 'Shorter paretic' steps, non-paretic AP impulse during late non-paretic single-support phase related strongly to paretic step lengths. Contrarily, in persons taking 'Longer paretic' steps several persons showed a strong relation between non-paretic AP impulse in the early phase and paretic step lengths (Figure 6-5). Further, since compared to the 'Shorter paretic' and 'Symmetric' group, persons in this group primarily showed non-paretic leg propulsion in its

early stance phase, it is likely that persons taking longer paretic steps show additional increased non-paretic propulsive impulse that begins sooner in the gait cycle (Figure 6-4). This greater non-paretic leg propulsive impulse might be propelling the trunk further forward early during paretic leg swing phase resulting in the paretic leg stepping longer than the non-paretic. Specifically, since impaired paretic leg propulsion can be compensated by increased non-paretic leg propulsion, persons in the 'Longer paretic' group show greater compensatory non-paretic leg propulsion to offset the reduced paretic leg propulsion [194]. Therefore, while increased non-paretic leg propulsion in general related to a longer step length; greater compensatory non-paretic leg propulsion that begins sooner resulted in a relatively longer paretic step than non-paretic in the 'Longer paretic' group. Further, note that compared to the non-paretic leg propulsion being selected more in early stance than late; the paretic leg propulsion is selected more in late stance than early and is also primarily braking in nature suggesting the reduced paretic leg propulsion in the 'Longer paretic' group.

Ipsilateral Hip Impulse in Early Swing is a Significant Predictor of Step Length Variability in Persons taking Longer Paretic than Non-Paretic Steps

Hip impulse during initial swing showed a strong relationship to step lengths. It was selected as a significant predictor explaining paretic step length variability specifically in the 'Longer paretic' group of participants and as an important predictor explaining non-paretic step length variability in majority of participants. Note that in participants taking 'Shorter paretic' steps, paretic hip impulse did not significantly explain paretic step length variability (Table 6-3). Whereas, in participants taking 'Longer paretic' steps and 'Symmertic' steps, hip impulse in initial swing was one of the important variables (Longer paretic > Symmetric) chosen by the regression models. Furthermore, the hip impulse was primarily flexor during early swing (Figure

6-4) suggesting that this flexor moment during early swing was pivotal to accelerating the leg in this phase such that the step is longer.

In particular, the weak relation between paretic flexor moment in early swing and step length suggests that persons taking 'Shorter paretic' steps might be at the limit of their paretic hip flexor moment generation (due to impaired hip flexor activity) such that it no longer correlates with step length on a step-by-step basis. Impaired paretic flexor activity shortens the paretic steps relative to non-paretic in the 'Shorter paretic' group. Note that, the non-paretic hip impulse during early swing showed a significant relationship to non-paretic step lengths in 3/6 participants further indicating the specific absence of relationship between paretic hip impulse and paretic step lengths in the 'Shorter paretic' group (refer to Table 6-4). Therefore, we propose that the impaired paretic hip moment in early swing shortens the paretic step length resulting in asymmetrical shorter paretic steps than non-paretic in this sub-group of participants.

Note that while hip flexor moment in initial swing was consistently positively related to step lengths, direction of relationship between hip moments in late swing and step lengths was inconsistent. Hip moments during late swing can both accelerate and decelerate the leg swing Flexor moments (from uniarticular hip flexors) during late swing can hold the leg longer in swing such that the leg steps longer, whereas extensor moments (often from biarticular muscles like hamstrings and gluteus maximus [53]) during late swing can terminate the swing phase such that the leg steps shorter. Interestingly, most participants taking Shorter paretic steps showed paretic hip extensor moments, whereas 19/22 (H4 showed flexor activity) taking Longer paretic steps primarily showed hip flexor moments even in late swing phase (Figure 6-4). This observation further suggests the greater flexor related activity in the 'Longer paretic' group compared to the 'Shorter paretic' group. Further evaluation of EMG activity in individual

participants and similar model building using EMG activity to explain step length variability can help implicate the specific muscles responsible for these observed kinetic relationships.

Ankle-Joint Center Velocity at Toe-Off is a Significant Predictor of Non-Paretic Step Length Variability

Ankle joint center velocity at toe-off was significantly positively related to non-paretic step length variability in majority of participants, suggesting that greater AJC velocity at toe-off accelerates the foot forward into swing. Contrarily, AJC velocity was not as commonly selected to explain paretic step length variability. AJC velocity at toe-off will be affected by ankle excursion (plantarflexion) at toe-off, ankle muscle paresis, spasticity, increased antagonist coactivation and increased passive stiffness [207]. This indicates that the paretic impairments commonly observed in majority of participants post-stroke can affect the AJC angular velocity at toe-off. Therefore, paretic AJC velocity at toe-off was not as strongly related to step lengths since majority of hemiparetic participants might be at the limit of their paretic AJC velocity (due to impaired capacity). It is also possible that while there is little variation in AJC velocity within-subjects, there would be considerable variation between-subjects and would contribute significantly to between-subject variation in step lengths.

Leg Orientation at Toe-Off and Pelvis Velocity at Toe-Off and their Contribution to Explaining Step Length Variability

Leg orientation at toe-off was the second most commonly selected predictor variable that was significantly related to paretic step lengths in participants taking ‘Symmetric’ steps. Note that, while it is expected that leg orientation would be negative at toe-off, there were seven hemiparetic participants (all taking Longer paretic steps than non-paretic) with the leg oriented anterior to pelvis in this phase (i.e., showing positive values for paretic leg orientation for all steps that they walked) and four others who showed positive values for at least half of the steps that they took. Posterior leg orientation implies an extended leg at toe-off. Selection of paretic

leg orientation specifically in the symmetric group suggests that these persons might be attaining appropriate leg extension that would acts to direct the ground reaction forces appropriately to propel the trunk forward at the terminal stance phase. Pelvis velocity at toe-off was significantly related to step lengths only in few participants; suggesting that the velocity of the body during the initial conditions prior to ipsilateral swing was not a strong predictor of step lengths.

Within-Subjects Regression Models

The within-subjects regression methodology as used in our study is different from conventionally used methodology where average data for individual subjects are used to build one regression model. Within-subject analyses as used in our study are specific to our study question where we want to determine parameters that influence the step length variability in individual participants. Since each participant walks at their self-selected speed and at a pre-determined cadence that differs across participants, grouping all subjects' data would confound the results if we want to determine those predictors that significantly relate to step-by-step variability in step lengths. For instance, when we averaged the data for individual subjects and ran one regression model to understand paretic step length generation between-participants, the predictor variable that related to between-subjects were leg orientation at toe-off that was negatively related to step length and hip impulse in early swing that was positively related to step lengths. This result suggests that longer or shorter paretic step length between-participants related to how their leg was oriented at toe-off and hip impulse in early swing. While this information is valuable to understand why some persons take longer steps than others, in order to effect changes in individual patients' performances, it would be more essential to understand which predictor variables resulted in an increase or decrease in step length.

Such an approach of individual-subject analyses has been highlighted in earlier work [163]. Olney et al. (1994) in their attempt to understand the most critical variables that predict

walking speed suggested that the regression analysis done on the subjects deviations from their averages (i.e., the within-subject analyses) is more appropriate if the aim were to understand what variables can affect individual persons performance. They suggested that their within-subjects regression analyses were appropriate because it shows that the walking speed of a subject increases with appropriate changes in the chosen predictor variables. They also acknowledged that on the other hand between-subject analyses were more appropriate if the aim were to determine those variables that predict the walking speeds of different subjects. The individual-subject regression analyses as used in our study accounted for the step-by-step variability in the initial conditions, swing phase and contralateral stance phase mechanisms that control step lengths and therefore, provide strong evidence for mechanisms of step length generation.

Limitations

Gait variables are expected to be correlated. This quality of the variables is described as "multicollinearity." Although multicollinearity can be a problem in some regression modeling, it does not interfere in our study because 1) we tried to select relatively independent variables; 2) we only had 7 predictor variables, and 3) Stepwise procedures mitigate against the retention of highly correlated predictor variables. It is possible that the step-by-step variability in step lengths was directly related to the explanatory power of the models; suggesting that increased or decreased model variance could be simply due to increased or decreased step variability. Nonetheless, we found that there was no significant relationship between the step-by-step variability in step lengths and the variance explained by both the paretic and non-paretic regression models indicating the model's specificity in selecting those variables that best related to paretic step lengths. Further, extrinsic sources of error might have increased the step variability (i.e., differences in marker placement from subject to subject). However, since we

used a within-subjects design these sources of variation were minimal. It is possible that there are other predictors that relate to step length variability (e.g. knee and ankle moments during initial conditions and during swing, pelvic displacements specifically at the initial conditions prior to swing). Studies are warranted to systematically consider other predictors that might relate to step length variability.

Conclusions

Use of an individual-subjects design incorporating step-by-step variability in gait data helped us to determine the specific mechanisms that relate to step length generation. Based on chosen predictors that relate to step lengths, we are able to propose specific mechanisms that affect step length generation. Knowledge of these mechanisms, in turn, highlights rehabilitation strategies that can be targeted specifically to affect step lengths and improve walking outcomes post-stroke. In the sub-group of persons taking 'Longer paretic' steps than non-paretic, we suggest that improving paretic leg propulsion would increase the non-paretic step length that would reduce their asymmetry. On the other hand, in the sub-group taking 'Shorter paretic' steps than non-paretic, improving the paretic hip flexor activity could lengthen the paretic steps and reduce their asymmetry. Future studies should evaluate EMG muscle activity and its relation to step length variability to support and further explain the current proposed mechanisms underlying step length generation during hemiparetic walking.

Table 6-1. Subject characteristics

ID	Paretic side	Gender	Age (years)	Time since stroke (mos)	Fugl-meyer grading
H01	Right	M	49		moderate
H02	Left	M	52	94	severe
H03	Left	F	54	18	moderate
H04	Right	F	45	11	moderate
H05	Left	M	74	26	moderate
H06	Right	M	70	62	moderate
H07	Right	M	35	11	moderate
H08	Left	M	48	55	moderate
H09	Right	M	72	31	moderate
H10	Right	M	61	116	mild
H11	Right	F	77	127	mild
H12	Left	M	63	114	moderate
H13	Right	M	81	29	moderate
H14	Left	M	69	20	mild
H15	Right	F	68	100	mild
H16	Right	F	61	65	severe
H17	Left	M	40	104	moderate
H18	Right	M	60	58	mild
H19	Left	F	36	12	moderate
H20	Left	F	45	10	moderate
H21	Right	F	74	7	mild
H22	Left	F	70	21	severe
H23	Left	F	79	36	moderate
H24	Left	M	55	37	moderate
H25	Left	M	53	25	severe
H26	Left	M	61	62	moderate
H27	Left	M	55	11	moderate
H28	Left	M	45	9	moderate
H29	Left	F	54	76	severe
H30	Left	M	53	11	moderate
H31	Left	F	61	8	severe
H32	Left	M	63	90	severe
H33	Right	F	40	43	moderate
H34	Right	F	53	67	moderate
H35	Left	M	72	64	moderate
H36	Left	F	46	95	moderate
H37	Left	M	60	101	mild
H38	Right	M	72	86	severe

Note that Fugl-meyer grading is based on scores from lower-exremity fugl-meyer scale (synergy portion only – maximum score = 22). Severe ≤ 14, Moderate = 15 – 18, Mild ≥ 19.

Table 6-2. Average gait characteristics for individual participants

Sub ID	Min # of Steps	Speed (m/s)	Cadence (steps/ min)	P SL (m)	N SL (cm)	P SwT (s)	N SwT (s)
H01	65	0.70	97	0.38	0.43	0.55	0.24
H02	40	0.20	66	0.13	0.21	0.38	0.29
H03	54	0.25	83	0.17	0.16	0.40	0.22
H04	65	0.80	96	0.47	0.42	0.46	0.28
H05	53	0.30	84	0.21	0.19	0.32	0.25
H06	52	0.15	81	0.12	0.10	0.30	0.14
H07	26	0.15	45	0.22	0.16	0.64	0.25
H08	42	0.35	68	0.31	0.29	0.58	0.28
H09	58	0.15	66	0.21	0.05	0.45	0.13
H10	64	0.50	96	0.28	0.31	0.43	0.32
H11	76	0.15	116	0.36	-0.03	0.26	0.17
H12	52	0.40	83	0.32	0.23	0.46	0.24
H13	49	0.70	106	0.33	0.40	0.34	0.34
H14	31	0.20	72	0.22	0.09	0.45	0.24
H15	20	0.15	60	0.26	0.02	0.60	0.27
H16	50	0.15	88	0.20	0.005	0.43	0.22
H17	19	0.20	50	0.32	0.22	0.60	0.35
H18	49	0.80	107	0.36	0.48	0.30	0.31
H19	35	0.20	85	0.18	0.06	0.55	0.15
H20	58	0.30	68	0.14	0.11	0.50	0.34
H21	41	0.40	91	0.22	0.27	0.32	0.30
H22	32	0.15	55	0.20	0.10	0.33	0.21
H23	53	0.20	64	0.23	0.13	0.26	0.21
H24	71	0.30	79	0.17	0.26	0.28	0.27
H25	62	0.45	92	0.29	0.27	0.34	0.26
H26	52	0.30	79	0.30	0.12	0.27	0.20
H27	61	0.55	91	0.31	0.27	0.49	0.26
H28	50	0.20	76	0.19	0.11	0.46	0.22
H29	51	0.45	123	0.14	0.27	0.27	0.20
H30	45	0.20	107	0.15	-0.009	0.38	0.13
H31	45	0.30	72	0.26	0.20	0.43	0.23
H32	40	0.20	88	0.30	-0.001	0.43	0.13
H33	44	0.40	67	0.34	0.29	0.58	0.33
H34	57	0.35	84	0.24	0.26	0.34	0.24
H35	46	0.40	70	0.35	0.33	0.39	0.27
H36	49	0.40	75	0.40	0.20	0.46	0.38
H37	62	0.90	96	0.53	0.51	0.37	0.39
H38	57	0.40	88	0.28	0.26	0.35	0.24

Note that min # of steps indicates the minimum number of good (paretic or non-paretic) steps that were used for data analyses for each participant pooling all trials together. Abbreviations: P – Paretic; N – Non-paretic; Cad – Cadence; SL – Step length; SwT – Swing time.

Table 6-3. Regression models for individual participants to predict paretic step length variability

			PARETIC INITIAL CONDITIONS AT TOE-OFF			PARETIC SWING		NON-PARETIC STANCE		MODEL VARIANCE
Sub ID	Speed m/s	PSR %	LO	PV	AJC	Hip Imp ES	Hip Imp LS	cAPImp ES	cAPImp LS	R2 p<.05
H29	0.45	36.6	-2		-3				1	0.67
H02	0.20	39.8								nr
H18	0.80	43.3							1	0.26
H21	0.40	44.2								nr
H13	0.70	44.3						-2	1	0.57
H01	0.70	47.3	-3		4			2	1	0.60
H10	0.50	47.8	-3		2				1	0.48
H34	0.35	48.1	-2					1		0.38
H08	0.35	49.3				1		2		0.33
H20	0.30	49.8					-1			0.08
H12	0.40	50.8		4	3	1	2		5	0.72
H37	0.90	50.8	-4		3		-1		2	0.65
H03	0.25	51.6	2		4	3			1	0.81
H25	0.45	51.6	-2		3				1	0.68
H35	0.40	51.6	-4	3			2		1	0.51
H38	0.40	52.5		2					1	0.28
H05	0.30	53.1	-3		5	4	1	6	2	0.68
H04	0.80	53.3	-2	-5		1	4	3		0.62
H06	0.15	53.8				1		2		0.79
H33	0.40	54.6				1				0.13
H27	0.55	55.6		3			1	2		0.47
H31	0.30	55.6				2		1		0.50
H17	0.20	58.4			2				1	0.41
H07	0.15	60.4							1	0.15
H28	0.20	63.7	3	-2		1				0.53
H23	0.20	63.8				1	2			0.43
H14	0.20	67.7							1	0.61
H36	0.40	67.7	-3				-4	1	2	0.68
H26	0.30	69.1				2			1	0.36
H22	0.15	70.7	2	4			-3	1		0.59
H24	0.30	71.0			2	3	4	1		0.42
H19	0.20	71.2	-3					1	2	0.37
H09	0.15	81.1	4			2	-3		1	0.48
H16	0.15	96.3				1				0.20
H15	0.15	99.8				2		1		0.80
H32	0.20	101.1	1	2					3	0.53
H30	0.20	102.7						1		0.53
H11	0.15	192.7			2	1	-4	3		0.76

Numbering (1, 2 ,3) indicates the order in which the predictors were chosen for the individual participants. 'nr' indicates no result.Blue represents participants taking 'Shorter paretic' steps, Green represents participants taking 'Symmetric' steps and Red represents participants taking 'Longer paretic' steps (PSR > 52.5). Abbreviations: Sub ID – Subject; PSR – Paretic Step ratio; LO – Leg orientation at toe-off; PV – Pelvis velocity at toe-off; AJC – Ankle joint center velocity at toe-off; Hip Imp ES – Hip Impulse during Early (1st 50%) swing; Hip Imp LS – Hip Impulse during late (2nd 50%) swing; cAPImp ES – Contralateral AP impulse during ipsilateral early swing; cAPImp LS – Contralateral AP impulse during ipsilateral late swing.

Table 6-4. Regression models for individual participants predicting non-paretic step length variability

			NON- PARETIC INITIAL CONDITIONS AT TOE-OFF			NON-PARETIC SWING		PARETIC STANCE		MODEL VARIANCE
Sub ID	Speed m/s	PSR %	LO	PV	AJC	HImp IS	HImp LS	cAPImp IS	cAPImp LS	R2 p<.05
H29	0.45	36.6		-5	2	3		4	1	0.79
H02	0.20	39.8	-2						1	0.83
H18	0.80	43.3					-2	-3	1	0.47
H21	0.40	44.2	-3			1		2		0.47
H13	0.70	44.3		-2		4	3		1	0.77
H01	0.70	47.3	-2		5		4	3	1	0.72
H10	0.50	47.8			2	3		1	4	0.55
H34	0.35	48.1	-3				2	1		0.35
H08	0.35	49.3		-5	2	3	4		1	0.65
H20	0.30	49.8					-1			0.15
H12	0.40	50.8			2				1	0.84
H37	0.90	50.8	-2		3				1	0.44
H03	0.25	51.6			2	3	4		1	0.89
H25	0.45	51.6	-2		4	3		1		0.54
H35	0.40	51.6	-1			2				0.35
H38	0.40	52.5		-2	1	3				0.59
H05	0.30	53.1			2	1			3	0.79
H04	0.80	53.3	-1		3			2	4	0.51
H06	0.15	53.8			2	3		1		0.82
H33	0.40	54.6		4	2	1			3	0.71
H27	0.55	55.6	-3	-2			4		1	0.76
H31	0.30	55.6			2	3		4	1	0.60
H17	0.20	58.4	-2						1	0.56
H07	0.15	60.4				2			1	0.48
H28	0.20	63.7			4	3	-2		1	0.70
H23	0.20	63.8					-1			0.14
H14	0.20	67.7	-2						1	0.78
H36	0.40	67.7	-2				1	3		0.48
H26	0.30	69.1			2	3			1	0.81
H22	0.15	70.7				2	-1	3		0.81
H24	0.30	71.0		-4	1	2	5	3		0.67
H19	0.20	71.2			2	3		1		0.81
H09	0.15	81.1			3		-2		1	0.65
H16	0.15	96.3			2	3			1	0.86
H15	0.15	99.8	-3			1			2	0.81
H32	0.20	101.1			3	2			1	0.67
H30	0.20	102.7				1	-3	2		0.51
H11	0.15	192.7			2	3		4	1	0.76

Numbering (1, 2 ,3) indicates the order in which the predictors were chosen for the individual participants. 'nr' indicates no result.Blue represents participants taking 'Shorter paretic' steps, Green represents participants taking 'Symmetric' steps and Red represents participants taking 'Longer paretic' steps (PSR > 52.5). Abbreviations: Sub ID – Subject; PSR – Paretic Step ratio; LO – Leg orientation at toe-off; PV – Pelvis velocity at toe-off; AJC – Ankle joint center velocity at toe-off; Hip Imp ES – Hip Impulse during Early (1st 50%) swing; Hip Imp LS – Hip Impulse during late (2nd 50%) swing; cAPImp ES – Contralateral AP impulse during ipsilateral early swing; cAPImp LS – Contralateral AP impulse during ipsilateral late swing.

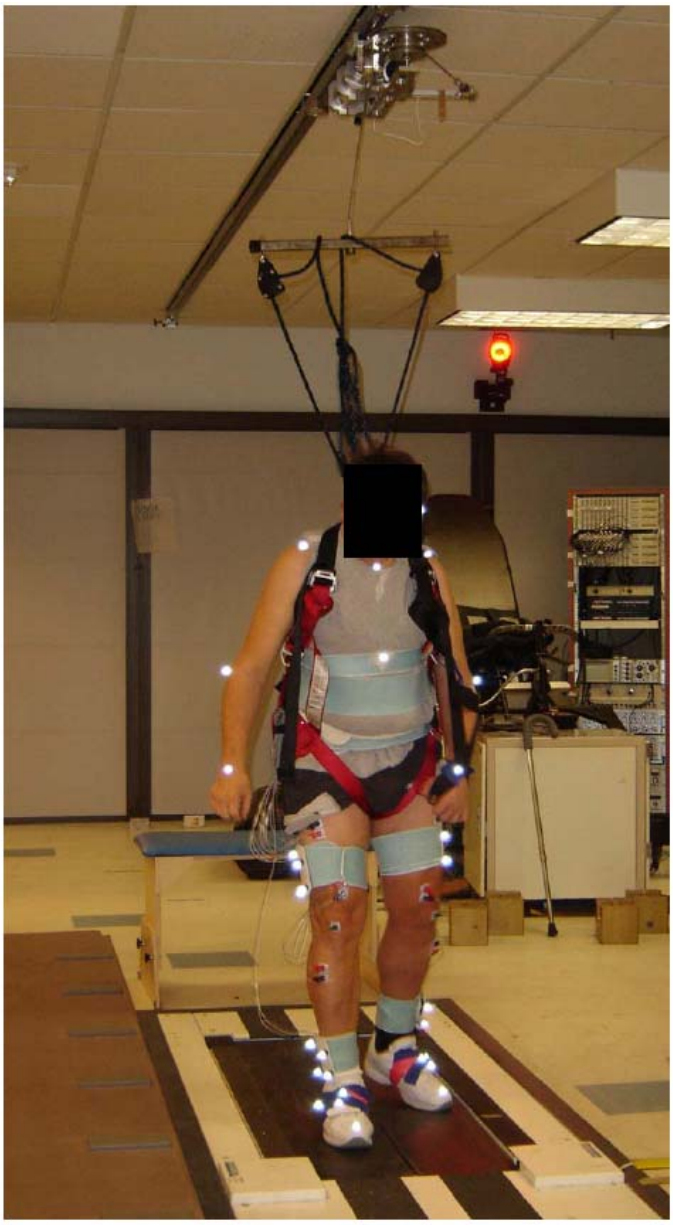


Figure 6-1.An individual participant walking on the split belt treadmill as kinematic, kinetic and EMG data were recorded

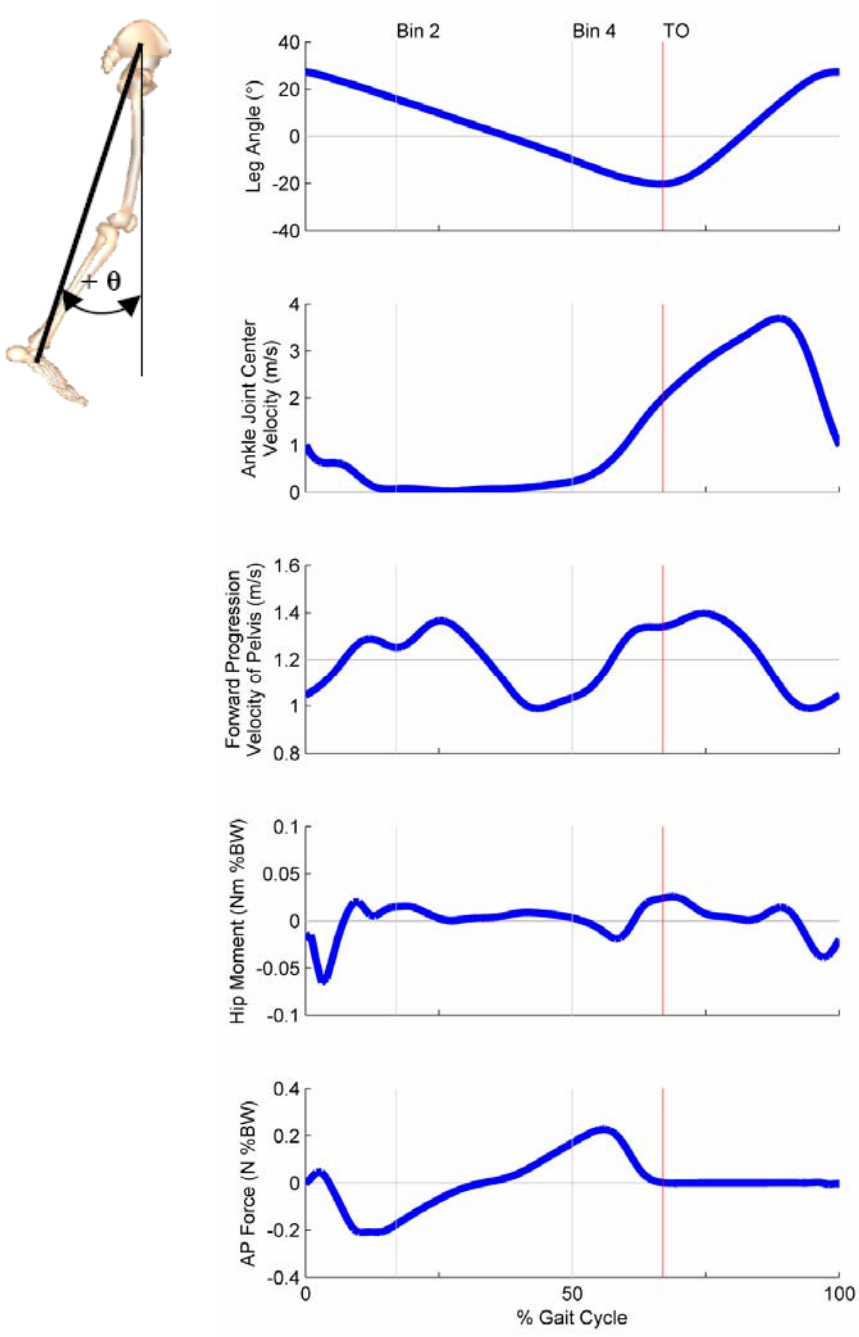


Figure 6-2. Illustration of variables used in the study

Kinematic and Kinetic profile is illustrated from one step during walking for an individual participant. Note that all variables are normalized to the gait cycle. Red line indicates toe-off. Leg orientation, Pelvis velocity and Ankle joint center velocity calculated at toe-off were the initial condition variables. Hip impulse and Contralateral AP impulse during initial (1st 50%) and late (2nd 50%) swing were the swing phase variables calculated. Note that the impulse is calculated as the area under force curve. Raw profile for the Hip moment and AP GRF are not presented. The AP impulse presented in the figure is from the contralateral leg that is occurring at the same instance as the ipsilateral swing phase.

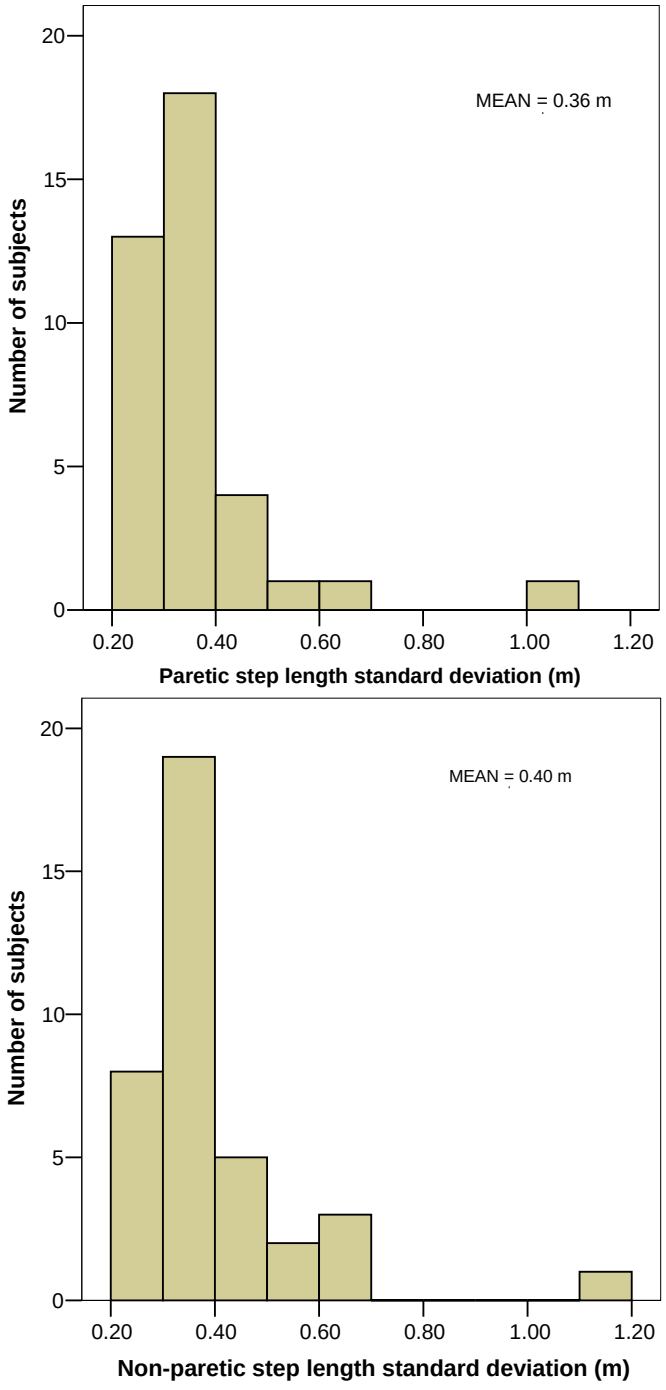


Figure 6-3. Frequency distribution of step-to-step variability in step lengths.

A. Non-paretic AP Impulse

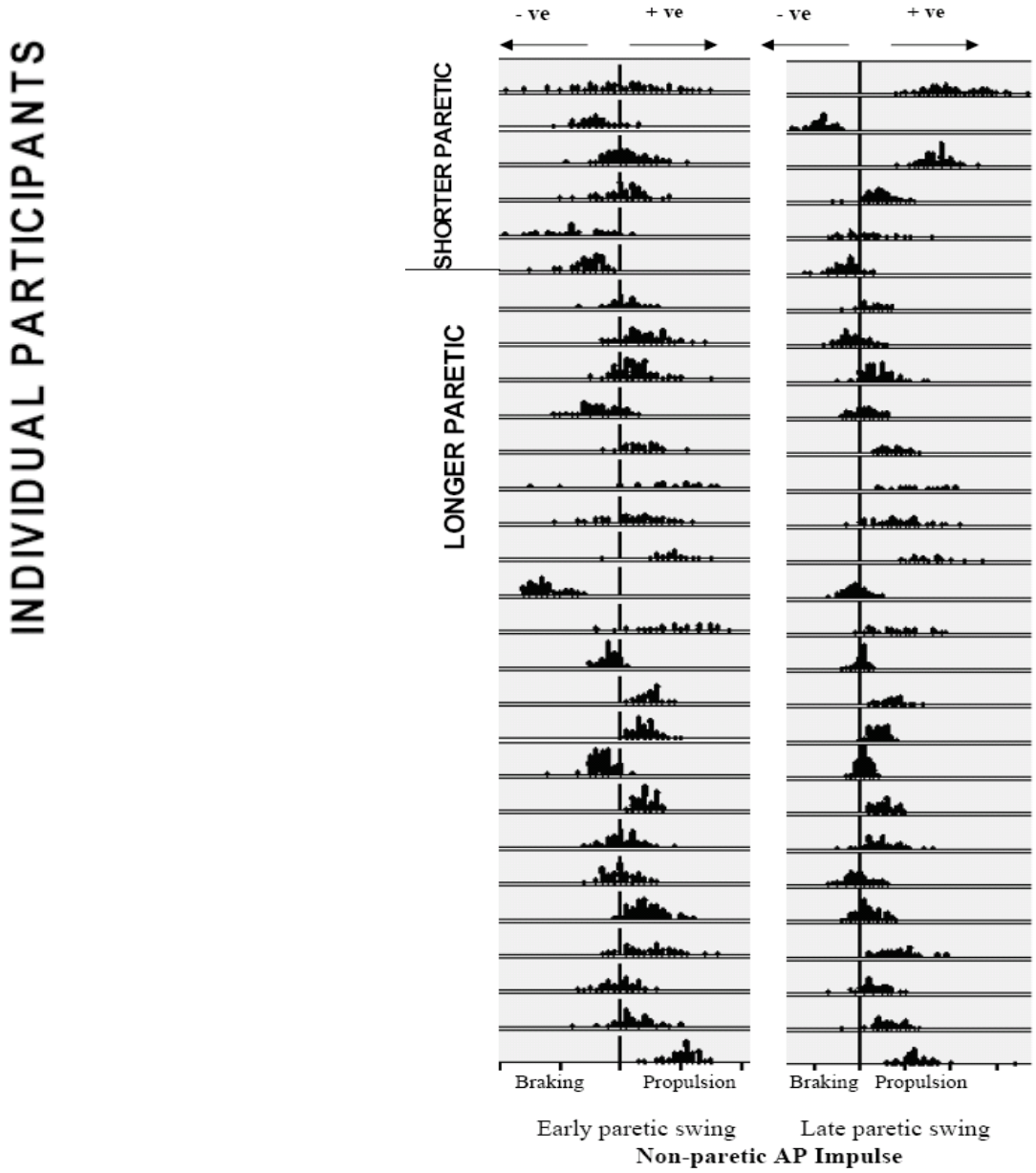


Figure 6-4. Non-paretic AP impulse and Paretic hip impulse during paretic stepping in the asymmetrical sub-groups

B. Hip Impulse

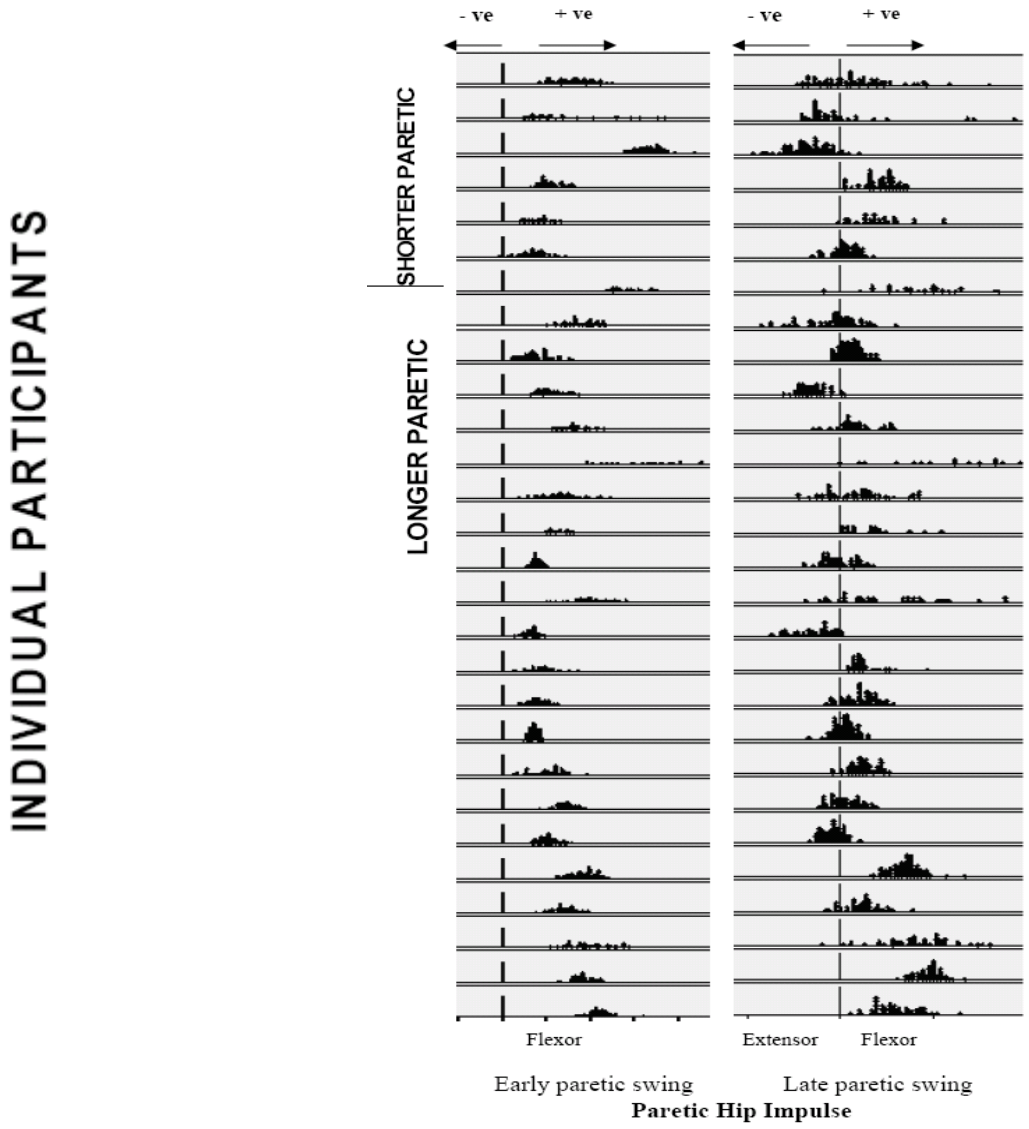
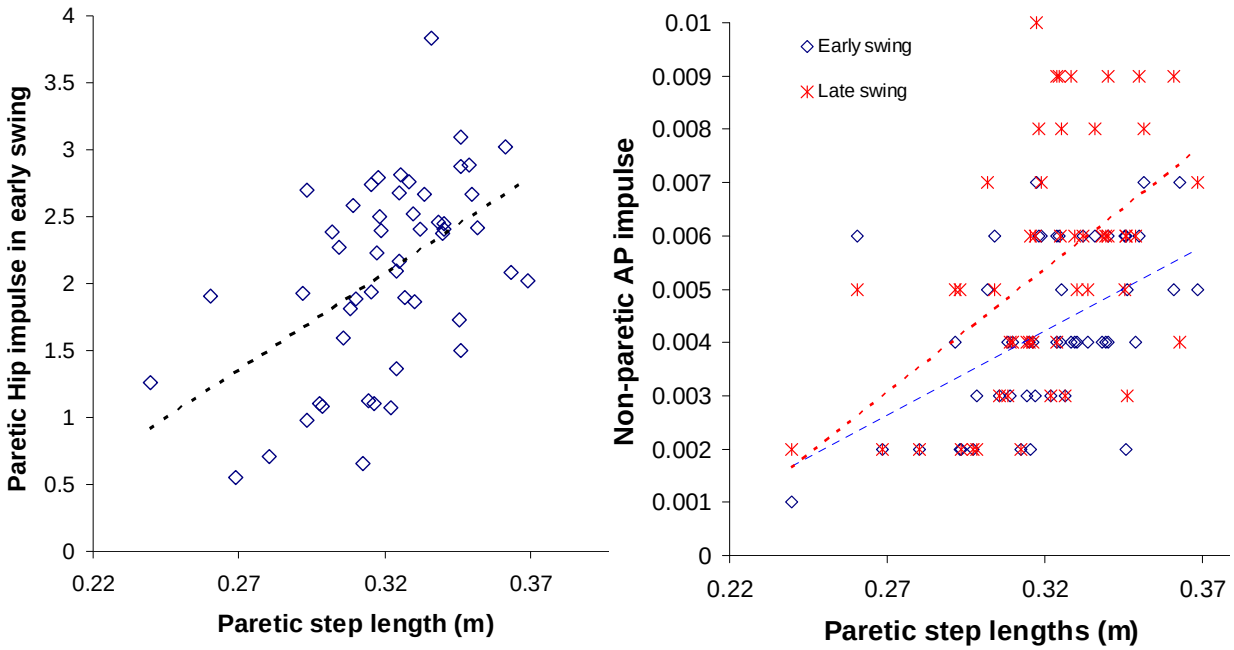


Figure 6-4. Continued.

This figure presents the non-paretic AP impulse and hip impulse from individual steps for the asymmetrical sub-groups (n = 28; 22 taking longer paretic steps and 6 taking shorter paretic steps). Each row of data is a simple dot plot of the AP impulse or hip impulse for individual participants. The participants are lined based on the ascending order of the asymmetry (note that symmetric persons have not been presented here). For AP impulse, positive values on the right represent propulsive forces and negative values on the left represent braking forces. For Hip impulse, positive values on the right represent flexor moment and negative values represent positive moments.
Note that the non-paretic AP impulse in early swing is primarily propulsive for the Longer paretic persons compared to several shorter paretic persons showing braking forces. Similarly, the non-paretic AP impulse in late swing is propulsive for most participants taking longer paretic steps compared to 3 participants in the Shorter paretic group showing more braking. Paretic hip impulse was flexor in early swing for most participants. Conversely, hip impulse was extensor for at least some participants in the late swing. Participants taking shorter paretic steps showed extensor moments (4/6) while the most asymmetric longer paretic persons (bottom rows) showed primarily flexor moments.



PSR – 69.8
Speed – 0.30 m/s

Figure 6-5. Relationship between paretic hip impulse in early swing, non-paretic AP impulse in early and late swing in an individual participant taking longer paretic steps than non-paretic

CHAPTER 7
CONCLUSIONS: INTEGRATING THE FINDINGS

The asymmetrical nature of hemiparetic gait is extensively documented [12-15]. Yet, there is considerable disagreement in the literature regarding the clinical relevance of evaluating asymmetrical gait post-stroke. This disagreement stems from two conflicting suggestions in the literature. Some authors report that gait symmetry should be an ideal objective in walking rehabilitation post-stroke due to the apparently asymmetrical nature of hemiparetic walking [12, 208, 209]. While others highlight that since compensatory strategies can promote functional performance, normalization of movement patterns should not be the primary focus of gait rehabilitation [5, 18]. We suggest that, while gait symmetry need not be the primary focus for walking rehabilitation, gait asymmetry should be evaluated to identify the unique paretic leg motor deficits and to understand how the paretic leg impairments might limit functional walking performance.

This dissertation aimed at quantifying gait asymmetry, understanding its relationship to walking performance and investigating mechanisms underlying gait asymmetry. Specifically, stepping asymmetry was investigated since spatiotemporal characteristics of steps are the final outcomes of all events occurring in the gait cycle. Further, since the stepping measures can be easily recorded, transition of our results to clinical settings can be easily facilitated.

Step Length Asymmetry during Hemiparetic Walking

While temporal asymmetry is well understood [13], step length asymmetry is not well quantified and its relation to hemiparetic walking performance is unclear. Therefore, in study one of this dissertation, we primarily quantified the step length asymmetry patterns during hemiparetic walking and proposed some underlying mechanisms in the asymmetrical sub-groups. We were able to quantify the step length asymmetry patterns using a step length

asymmetry ratio and by relating the step length asymmetry to propulsive forces asymmetry. Our results also showed that while step length asymmetry related to hemiparetic severity, it did not limit the attained walking speed highlighting other compensatory strategies to achieve the functional walking speeds. Our findings also suggested differing motor control mechanisms (i.e., ability to change speeds, weight supported on paretic leg) in persons showing different step length asymmetry patterns. Future work is encouraged to understand the specific sensorimotor impairments related to the step length asymmetry patterns and evaluate whether walking rehabilitation directed at these impairments can improve walking outcomes post-stroke.

Step Variability during Hemiparetic Walking

Study two of this dissertation investigated step variability to determine how gait variability might relate to asymmetrical walking post-stroke and whether measures of variability can be used as markers of impaired performance. Gait variability is shown to be strongly associated with motor and balance control during walking and suggested to be a quantifiable biomechanical marker to evaluates walking impairments [77]. Our results revealed that increased variability in most spatiotemporal characteristics and reduced width variability related to poor performance outcomes (severe hemiparesis, asymmetrical gait and poor balance). Specifically, results showed an asymmetry in swing and pre-swing time step variability in participants with the most impaired performance. There was no difference in step length variability between paretic and non-paretic steps, suggesting that the step length asymmetry patterns are relatively consistent from step to step during hemiparetic walking. Specifically since step variability is shown to strongly predict the risk for falling [98, 103], the finding that the asymmetrical groups showed greater step variability than the symmetrical persons suggests the dynamic balance impairments in these persons. Future research is warranted to determine specific thresholds of step variability to further validate the use of variability measures as performance markers.

Asymmetrical Stepping in a Body Reference Frame Post-Stroke

In study three of this dissertation, stepping asymmetry was quantified in a body reference frame. Literature shows that asymmetry in steps and asymmetry in body movements are investigated in isolation [11, 199]. Nonetheless, since foot placement (during stepping) closely relates to body movements, it might be essential to investigate asymmetry in foot placement relative to body (rather than in isolation). Our results showed that post-stroke anterior-posterior and medial-lateral foot placements were asymmetrical relative to body and that this asymmetry related to step length asymmetry but not step widths. Wider paretic foot placement relative to pelvis than non-paretic also related to reduced paretic leg weight support and lateral instability, encouraging the clinical utility of medial-lateral foot placement relative to pelvis. Since foot placement relative to body can be associated to forward progression and dynamic balance [132, 133], investigation of stepping asymmetry in a body reference frame also helped us propose underlying mechanisms of stepping asymmetry. Future studies are encouraged to evaluate the underlying kinematics of hip and knee to develop an integrated understanding of stepping in the asymmetrical groups.

Step Length Generation during Hemiparetic Walking

In study four of this dissertation, mechanisms of step length generation during hemiparetic walking were evaluated using a novel methodology that incorporated the step-by-step variability in step lengths. Literature suggests several potential indirect and direct mechanisms relates to step length [173, 182, 194] but there is no clear consensus on predictors that relate to step length generation. Results revealed that contralateral anterior-posterior and hip impulses during swing explained the step length variability in majority of participants. Relation of these predictors to step lengths differed based on step length asymmetry patterns, implying differential mechanisms of step length generation across the sub-groups of persons showing differing step length

asymmetries. Compared to persons taking shorter paretic steps or symmetric steps, persons taking longer paretic steps showed early stance phase non-paretic leg propulsion, suggesting increased non-paretic propulsive impulse beginning sooner in the gait cycle. In participants taking shorter paretic steps, paretic hip impulse in initial swing was not strongly related to paretic step lengths unlike persons in other groups for whom hip flexor impulse was a strong predictor of paretic step lengths. Based on these observations, we suggest that in persons taking longer paretic steps than non-paretic, improving paretic leg propulsion would increase the non-paretic step length and reduce their asymmetry. While in persons taking shorter paretic steps than non-paretic, improving the paretic hip flexor activity could lengthen the paretic steps and reduce their asymmetry. Future studies should evaluate EMG muscle activity and its relation to step length variability to support and further explain the current proposed mechanisms underlying step length generation in the step length asymmetry groups.

Summary

Our findings suggest that stepping asymmetry is a clinically relevant measure to evaluate paretic leg performance post-stroke. We propose step length asymmetry, asymmetry in swing and pre-swing time step variability and medial-lateral foot placement relative to the pelvis as outcomes of asymmetrical walking performance. Our findings also suggest differential motor control mechanisms in persons walking with differing step length asymmetry patterns.

Results of this dissertation highlight the need to evaluate gait asymmetry post-stroke which would, in turn, enable us to differentiate physiological restitution from compensation during walking. Clinically, specificity in evaluation would help to tailor locomotor retraining specifically to address the root causes of impaired ambulation for each individual.

APPENDIX A
LOWER EXREMITY FUGL-MEYER SCALE

TEST	ITEM	SCORING CRITERIA
1. Reflex activity	Achilles Patellar (KNEE FLEXION)	0-No reflex activity can be elicited 2-Reflex activity can be elicited
2. Within synergy		
a. Flexor synergy (in supine) (FLEXOR SYN)	Hip flexion (FLEXOR SYN – HIP FLEX) Knee flexion (FLEXOR SYN – KNEE FLEX) Ankle dorsiflexion (FLEXOR SYN – ANKLE DF)	0-cannot be performed at all 1-partial motion 2-full motion
b. Extensor synergy (in sidelying) (EXTENSOR SYN)	Hip extension (EXTENSOR SYN – HIP EXT) Hip adduction (EXTENSOR SYN – HIP ADD) Knee extension (EXTENSOR SYN – KNEE EXT) Ankle plantarflexion (EXTR SYN – ANKLE PF)	0-no motion 1-weak motion 2-almost full strength comapred to normal
3. Movement combining synergies (in sitting: knees free of chair) (COMBINE SYN)	Knee flexion beyond 90 (COMBINE SYN – KNEE FLEX)	0-no active motion 1-from slightly extended position, knee can be flexed, but not beyond 90 2-knee flexion beyond 90
	Ankle dorsiflexion (COMBINE SYN – ANKLE DF)	0-no active flexion 1-incomplete active flexion 2-normal dorsiflexion
4. Movement out of synergy (in standing, hip at 0) (OUT OF SYN)	Knee flexion (OUT OF SYN – KNEE FLEX)	0-knee cnnot flex without hip flexion 1-knee begins flexion without hip flexion, but does not reach to 90, or hip flexes during motion 2-full motion as described
	Ankle dorsiflexion (OUT OF SYN – ANKLE DF)	0-active motion 1-partial motion 2-full motion
5. Normal reflexes (sitting)	Knee flexors Patellar Achilles	0-at least 2 of the 3 phasic reflexes are markedly hyperactive 1-one reflex is hyperactive, or at least 2 reflexes are lively 2-no more than one reflex is lively and none are hyperactive
6. Coordination/speed (supine : heel to opposite knee; 5 repititions in rapid succession)	Tremor	0-marked tremor 1-slight tremor 2-no tremor
	Dysmetria	0-pronounced or unsystematic dysmetria 1-slight or systematic dysmetria 2-no dysmetria
	Speed	0-acticity is more than 6 seconds longer than unaffected side 1-(2-5) seconds longer than unaffected side 2-less than 2 seconds difference

APPENDIX B
DYNAMIC GAIT INDEX SCALE

Gait level surface _____
Instructions: Walk at your normal speed from here to the next mark (20')
Grading: Mark the lowest category that applies.
(3) Normal: Walks 20', no assistive devices, good sped, no evidence for imbalance, normal gait pattern
(2) Mild Impairment: Walks 20', uses assistive devices, slower speed, mild gait deviations.
(1) Moderate Impairment: Walks 20', slow speed, abnormal gait pattern, evidence for imbalance.
(0) Severe Impairment: Cannot walk 20' without assistance, severe gait deviations or imbalance.

Change in gait speed _____
Instructions: Begin walking at your normal pace (for 5'), when I tell you "go," walk as fast as you can (for 5'). When I tell you "slow," walk as slowly as you can (for 5').
Grading: Mark the lowest category that applies.
(3) Normal: Able to smoothly change walking speed without loss of balance or gait deviation. Shows a significant difference in walking speeds between normal, fast and slow speeds.
(2) Mild Impairment: Is able to change speed but demonstrates mild gait deviations, or not gait deviations but unable to achieve a significant change in velocity, or uses an assistive device.
(1) Moderate Impairment: Makes only minor adjustments to walking speed, or accomplishes a change in speed with significant gait deviations, or changes speed but has significant gait deviations, or changes speed but loses balance but is able to recover and continue walking.
(0) Severe Impairment: Cannot change speeds, or loses balance and has to reach for wall or be caught.

Gait with horizontal head turns _____
Instructions: Begin walking at your normal pace. When I tell you to "look right," keep walking straight, but turn your head to the right. Keep looking to the right until I tell you, "look left," then keep walking straight and turn your head to the left. Keep your head to the left until I tell you "look straight," then keep walking straight, but return your head to the center.
Grading: Mark the lowest category that applies.
(3) Normal: Performs head turns smoothly with no change in gait.
(2) Mild Impairment: Performs head turns smoothly with slight change in gait velocity, i.e., minor disruption to smooth gait path or uses walking aid.
(1) Moderate Impairment: Performs head turns with moderate change in gait velocity, slows down, staggers but recovers, can continue to walk.
(0) Severe Impairment: Performs task with severe disruption of gait, i.e., staggers outside 15" path, loses balance, stops, reaches for wall.

Gait with vertical head turns _____
Instructions: Begin walking at your normal pace. When I tell you to "look up," keep walking straight, but tip your head up. Keep looking up until I tell you, "look down," then keep walking straight and tip your head down. Keep your head down until I tell you "look straight," then keep walking straight, but return your head to the center.
Grading: Mark the lowest category that applies.
(3) Normal: Performs head turns smoothly with no change in gait.
(2) Mild Impairment: Performs head turns smoothly with slight change in gait velocity, i.e., minor disruption to smooth gait path or uses walking aid.
(1) Moderate Impairment: Performs head turns with moderate change in gait velocity, slows down, staggers but recovers, can continue to walk.
(0) Severe Impairment: Performs task with severe disruption of gait, i.e., staggers outside 15" path, loses balance, stops, reaches for wall.

Dynamic Gait Index continued….

Gait and pivot turn _____
Instructions: Begin walking at your normal pace. When I tell you, "turn and stop," turn as quickly as you can to face the opposite direction and stop.
Grading: Mark the lowest category that applies.
(3) Normal: Pivot turns safely within 3 seconds and stops quickly with no loss of balance.
(2) Mild Impairment: Pivot turns safely in > 3 seconds and stops with no loss of balance.
(1) Moderate Impairment: Turns slowly, requires verbal cueing, requires several small steps to catch balance following turn and stop.
(0) Severe Impairment: Cannot turn safely, requires assistance to turn and stop.

Step over obstacle ____
Instructions: Begin walking at your normal speed. When you come to the shoebox, step over it, not around it, and keep walking.
Grading: Mark the lowest category that applies.
(3) Normal: Is able to step over the box without changing gait speed, no evidence of imbalance.
(2) Mild Impairment: Is able to step over box, but must slow down and adjust steps to clear box safely.
(1) Moderate Impairment: Is able to step over box but must stop, then step over. May require verbal cueing.
(0) Severe Impairment: Cannot perform without assistance.

Step around obstacles _____
Instructions: Begin walking at normal speed. When you come to the first cone (about 6' away), walk around the right side of it. When you come to the second cone (6' past first cone), walk around it to the left.
Grading: Mark the lowest category that applies.
(3) Normal: Is able to walk around cones safely without changing gait speed; no evidence of imbalance.
(2) Mild Impairment: Is able to step around both cones, but must slow down and adjust steps to clear cones.
(1) Moderate Impairment: Is able to clear cones but must significantly slow, speed to accomplish task, or requires verbal cueing.
(0) Severe Impairment: Unable to clear cones, walks into one or both cones, or requires physical assistance.

Steps _____
Instructions: Walk up these stairs as you would at home, i.e., using the railing if necessary. At the top, turn around and walk down.
Grading: Mark the lowest category that applies.
(3) Normal: Alternating feet, no rail.
(2) Mild Impairment: Alternating feet, must use rail.
(1) Moderate Impairment: Two feet to a stair, must use rail.
(0) Severe Impairment: Cannot do safely.

TOTAL SCORE: ___ / 24

Adapted from: Herdman SJ. ***Vestibular Rehabilitation***. 2nd ed. Philadelphia, PA: F.A.Davis Co; 20

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BIOGRAPHICAL SKETCH

Chitra Lakshmi K.Balasubramanian received her Bachelor in Physical Therapy from College of Allied Health Sciences, Manipal Academy of Higher Education, Karnataka, India. She worked for one year imparting physical therapy to adults and children with disabilities in New Delhi, India.

Her strong interest in an interdisciplinary approach to rehabilitation encouraged her to pursue the Rehabilitation Science Doctoral program at University of Florida. She was funded by the Alumni fellowship for four years of her graduate education and from an National Institutes of Health research grant for the final eight months of her doctoral education. Her research employed biomechanical measures to understand asymmetrical nature of walking in persons who have had a stroke and she was guided under the expert tutelage of Dr. Steve Kautz.

In the near future, Chitra plans to use her doctoral education to actively pursue teaching and research in neurologically impaired populations. She is specifically interested in developing a scientific framework that will aid in designing efficient rehabilitation strategies to improve walking function. In the long-term, she plans to return to India and aims to establish a strong foundation for rehabilitation education and related research and facilitate development of a common academic structure in rehabilitation.