# NON-PARETIC LEG PERFORMANCE IN HEMIPARETIC WALKING: LOADING ASYMMETRIES, COMPENSATORY MECHANISMS AND RESPONSIVENESS TO LOCOMOTOR TRAINING

By

BHAVANA RAJA

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To my Parents who taught me how to dream and my Husband for helping me fulfill those dreams.

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| | |
|---|---|
| BWS | Body Weight Support |
| BWSTT | Body weight supported treadmill training |
| CNS | Central Nervous System |
| CPG | Central Pattern Generator |
| EMG | Electromyography |
| GM | Gluteus Medius |
| GRF | Ground Reaction Forces |
| LL/LU | Limb loading and unloading |
| LH | Lateral Hamstrings |
| LT | Locomotor Training |
| MG | Medial Gastrocnemius |
| MH | Medial Hamstrings |
| NDT | Neuro-Development Therapy |
| PNF | Propioceptive Neuromuscular Facilitation |
| RF | Rectus Femoris |
| SCI | Spinal Cord Injury |
| SO | Soleus |
| TA | Tibiablis Anterior |
| VM | Vastus Medialis |

Abstract of Dissertation Presented to the Graduate School
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# NON-PARETIC LEG PERFORMANCE IN HEMIPARETIC WALKING: LOADING ASYMMETRIES, COMPENSATORY MECHANISMS AND RESPONSIVENESS TO LOCOMOTOR TRAINING

By

Bhavana Raja

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Walking requires appropriate integration of sensorimotor input to coordinate both the legs. Altered walking post-stroke is traditionally considered a unilateral motor control problem of the paretic leg. The non-paretic leg is generally considered unaffected and left unattended during rehabilitation. The purpose of this dissertation was to understand the performance of the non-paretic leg during walking. To this end three studies were conducted. We investigated loading asymmetries (a major source of afferent input), quantified compensatory mechanisms (a major component of motor output) and investigated the responsiveness of those compensatory mechanisms to locomotor training. Participants with chronic hemiparesis and similarly aged healthy control subjects walked over an instrumented (with force plates) treadmill as kinematic, kinetic and electromyographic data were collected.

In the first study, limb loading and unloading during walking was quantified. Results of the analysis revealed limb loading and unloading was asymmetric between the paretic and the non-paretic leg and significantly different from healthy control subjects walking at matched speeds. Limb loading and unloading was related to knee

angle and leg angle. Individuals post-stroke make several adjustments in the loading and unloading pattern to maintain a steady walking state. We suggest promoting symmetry in limb loading and unloading while training kinematic changes might promote better gait pattern.

In the second study, the changes in the motor pattern of the non-paretic leg were quantified and evaluated as to whether they represented compensatory mechanisms or impairments. Results demonstrated that the muscle activity of the non-paretic leg differed from healthy control leg walking at matched speeds and that most of the changes appeared to be compensatory in nature. Some of these compensations are simply increased output of the usual dominant power bursts of a healthy leg, while others are novel adaptations in the non-paretic leg that appear to aid in the production of additional propulsion by the paretic leg.

In the third study of this dissertation, we investigated the response of the non-paretic leg motor pattern to a task-specific locomotor training, i.e. body weight supported treadmill training. Walking speed was increased post-training and the increase in walking speed was associated with significant changes in the non-paretic leg muscle activity. The results of our analysis revealed mixed response to LT post-stroke. The muscle activity of non-paretic leg was significantly normalized in some individuals walking post-LT. Nevertheless, there were some significant increases in the muscle activity in the non-paretic leg post-LT when compared to healthy control subjects walking at matched speeds. Most notably, participants that walked at slower speeds before LT, but acquired moderate walking speed post-LT exhibited the greatest increases in muscle activity. This suggests that, in addition to the changes that were

occurring in the paretic leg, the non-paretic leg appears to have contributed to the increased walking speed through increases in one or more compensatory mechanisms.

# CHAPTER 1
# LITERATURE REVIEW

## Introductory Statements

In the United States approximately 795000 new or recurrent strokes occur each year, with a surviving cohort of 6.5 million people.[1] Stroke is the primary cause of adult disability.[2, 3] The residual functioning deficits involve cognitive, communicative, visual and sensorimotor systems.[4] Motor impairments, characterized by hemiparesis or the weakness of one half of the body are reported in 75% of individuals after stroke.[4] The sensorimotor deficits include, weakness, impaired selective motor control, spasticity, and proprioceptive deficits that interfere with normal adult walking.[5]

Inability to walk independently after stroke is also associated with poor coordination and inadequate proprioceptive input to both the legs. Proprioception from the load receptors signals the body about the body's center of mass during locomotion.[6] Therefore, they facilitate balance during standing, quasi-static positions and walking. Loss of proprioception after stroke might result in asymmetric loading and unloading.

While asymmetry in loading and unloading is well documented in static and quasi-static tasks[7-10], there is little information about asymmetry in loading during walking. One of the primary reasons regarding the importance of investigating asymmetric loading during walking is because it is characterized by acceleration of the body segments.[7] Thus, unlike static and quasi-static postures the center of mass is mostly outside the base of support, which makes walking more unstable. Furthermore, walking is a cyclic task in which loading of one limb is simultaneously associated with the unloading of the other. Nonetheless, there is no specific information about loading of paretic and non-paretic leg during steady state walking in individuals post-stroke.

Alterations in walking ability post-stroke are primarily considered to be related to paretic leg performance. Therefore, most of the rehabilitation and research studies focus on the paretic leg, while assuming the non-paretic leg is unaffected; considering it to primarily be compensating for the impairments of the paretic leg. Recent research studies suggest performance is poor in bilateral task as opposed to unilateral tasks. For example, pedaling studies suggested the deteriorated bilateral performance could be attributed to the detrimental influence of the non-paretic leg on the paretic leg is performance.[11] This strongly indicates bilateral involvement post-stroke and therefore the need to assess and treat both the paretic and the non-paretic side, to facilitate better performance post-stroke.

With our increased understanding of the neural mechanisms of disease and recovery there has been increased information related to the role of bilateral neural structures and the potential effects of unilateral injury on limbs of both sides is better understood. Several recent studies have emphasized the importance of including the non-paretic leg in the evaluation and rehabilitation post-stroke[12, 13], but no study has specifically evaluated the non-paretic leg, or reported its response to task-specific rehabilitation training.

The literature review of this dissertation provides the background information relevant to the specific aims of this dissertation. It first broadly discusses alterations in hemiparetic gait in individuals walking after stroke. Secondly it presents the importance of limb loading (LL) during walking. Third, it overviews the information related to the involvement of non-paretic extremity after stroke, relevant to the second study. Finally it describes literature relevant to walking rehabilitation after stroke, the traditional and

modern day concepts with emphasis on the task-specific locomotor training (relevant to study 3).

## Stroke: Definition, Incidence

**Definition.** Stroke is defined as a condition characterized by rapidly developing symptoms and signs of a focal brain lesion, with symptoms lasting for more than 24 hours or interrupted by death, with no apparent cause other than of vascular origin.[14]

**Incidence and squeal.** Stroke is one of the most debilitating central nervous system conditions resulting in long term disability. Every year approximately 795,000 people are affected by stroke with a surviving cohort of 6.1 million individuals.[1] Over 75% of people affected by stroke have limitation in mobility related to walking.[4] Furthermore, only less than 50% of individuals achieve independent community walking status.[15] Of those individuals who recover the ability to walk, many are still disabled by slow walking speeds and limited endurance. Residual motor weakness, abnormal movement synergies and spasticity result in altered gait patterns and contribute to poor balance, risk for falls and increased energy expenditure.[16] Most of the research and clinical work identifies the impairments in walking after stroke as unilateral motor control problems of the paretic leg; however, a detailed investigation suggests that there is bilateral involvement of the limbs after stroke, as discussed in the studies of this dissertation.

## Pathophysiology of Stroke

The pathophysiological basis of stroke leading to various impairments can be either occlusive (due to obstruction of a blood vessel by a thrombus or an embolus) or hemorrhagic (due to rupture of a blood vessel) usually on one side of the brain. Insufficiency of blood supply is termed as ischemia which deprives brain tissue of

oxygen and glucose and prevents the removal of toxic metabolites. The decrease in blood flow results in the death of neurons and other cellular elements. Hemorrhagic stroke may occur at the brain surface or intraparenchymal causing a blood clot or haematoma within cerebral hemisphere or brainstem or in the cerebellum. The types and degrees of disability that follow a stroke depend upon multiple factors such as location and size of brain lesion, severity of the lesion, degree of spontaneous recovery, and the duration of stroke onset.[17, 18]

## Characteristics of Hemiparetic Gait

**Phases of the Gait Cycle.** Walking function may be quantified in terms of 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 same limb.[19] Each gait cycle is primarily divided into a stance phase (as when one limb is on the ground) and swing phase (as when the same limb is swinging with no contact on the ground).[19]

Stance and Swing phases of a gait cycle can be further sub-divided into different phases. Figure 1-1 presents the detailed description of the sub-divisions of a gait cycle. For the purpose of the studies of this dissertation these phases of gait cycle have been identified as different regions, also identified in figure 1-1.

## Biomechanical Characteristics of Hemiparetic Gait

Hemiparesis following stroke is characterized by a multitude of sensorimotor problems, marked by weakness, impaired selective motor control, spasticity and proprioceptive deficits that interfere with invariant features of normal adult gait.[5] Following a stroke, walking ability often plays a major role in a patient's discharge to either home or an assisted living environment. Therefore, ability to walk independently with efficient velocity and endurance is one of the major goals for rehabilitation after

stroke.[20] In order to successfully provide walking rehabilitation, it is important to understand and discuss the differences between hemiparetic and healthy adults' gait. Biomechanical measures provide an opportunity to investigate the control processes during walking post-stroke. This section aims to review the biomechanical characteristics (kinematics, kinetics and EMG) of hemiparetic gait with focus on both the paretic and non-paretic leg characteristics.

**Kinematic Characteristics**

Kinematic variables include linear and angular positions, their displacements and time derivatives.[21] Individuals post stroke display altered kinematics in sagittal plane with peak joint displacements or excursions at the hip, knee and ankle[22-24] in both the legs altered relative to healthy subjects walking. Common kinematic deviations of the paretic lower limb in the sagittal plane include ankle plantar flexion being increased at initial contact[12, 24] and mid swing and decreased at toe-off [25] (figure 1-2a). In addition, paretic leg knee flexion is increased at initial contact[25] and reduced during swing phase[26, 27] (figure 1-2b) while peak paretic hip extension is reduced at toe-off and peak paretic hip flexion is increased during swing phase as compared to healthy similarly aged individuals walking at self-selected or matched speeds[25, 28] (figure 1-2c). In the horizontal plane, individuals with hemiparesis demonstrate abnormally large external rotation at the hip joint on the paretic leg throughout the gait cycle.[26] Furthermore, some authors reported an abnormal abduction and inversion at the paretic hip and ankle in the frontal plane in individuals post-stroke.[12]

In addition to those in the paretic leg, some abnormal joint motions have also been reported on the non-paretic side. Hip extension at toe-off is reduced (figure 1-2c) and knee flexion is maintained at 15-30 degrees during the stance phase and reaches a

peak during the swing phase that is greater than that of the paretic leg ( approx. 20 degrees) and the healthy controls ( 35- 43 degrees)[29] (figure 1-2b).

The kinematic variables, primarily the knee and ankle angle have been employed by two studies to group the participants based on the similarity of their characteristics.[29, 30] Identification of patterns of gait dysfunction for patients following stroke have been used to construct a classification system to guide intervention. Relation of the gait patterns to the underlying impairments would allow clinicians to target rehabilitation strategies to the individual's needs.[30] De Quervain et al.[29] used observational gait analysis and Mulroy et al.[30] used non-hierarchial cluster analysis technique for this purpose. The groups defined by Mulroy et al.[30] classified individuals post-stroke into four groups based on their speed and knee and ankle angle characteristics. Individuals in group 1 were fast walkers characterized by decreased knee extension in mid-stance and adequate dorsiflexion in swing. Group 2 had intermediate velocity with greater knee flexion in mid-stance (as compared to group 1) and adequate dorsiflexion in swing, Group 3 had a very slow velocity and excessive knee flexion in mid-stance and inadequate dorsiflexion in swing, while group 4 also had a very slow gait velocity with a motion pattern characterized by knee hyperextension in mid-stance and inadequate dorsiflexion in swing.[30]

**Kinetic Characteristics**

Kinetic variables include forces, joint moments, powers and work. They are a direct consequence of joint moments generated by the muscles about a joint.[31] These variables cannot be measured directly (except forces), but can be calculated using the principles of inverse dynamics.[32] More than one type of kinematic and kinetic gait pattern have been identified across subjects post-stroke, indicating that persons with

stroke use different strategies to achieve the goal of walking.[12] Furthermore, kinetic variables explain the cause of the movement pattern observed at different joints.

**Ground reaction forces (GRF)**

GRF are recorded using force plates. GRF are derived from the Newton's third law of motion, which states that for every action there is an equal and opposite reaction.[33] In the study of human motion, this law indicates that all surfaces provide a reaction force and that the individual is acted upon by that force when he/she is in contact with that surface.[33] In human motion analysis, GRF is measured as a vector. This vector comprises of three orthogonal components: the anterior-posterior, the medial-lateral and the vertical component (figure 1-3a). Extrapolation of these forces in the proximal and distal direction by employing a free body diagram allows for the calculation of subsequent joint reaction forces and is the basis for the inverse dynamics approach of human motion analysis.[33] These calculations establish bases for the determination of joint moments, power and thus work, as discussed later.

**Vertical ground reaction forces.** Vertical GRF have been used to assess the weight bearing or "Loading" ability in static and quasi-static tasks.[7, 9, 10] In normal human locomotion, a typical vertical GRF curve is an M-shaped, double peaked curve separated by a valley. However, following a stroke the M-shaped pattern is distorted (figure 1-3b). Three different patterns of vertical GRF are typically observed in the paretic leg: double peaked similar to the normal M-shaped curve, single peak in mid-stance and a plateau with no discernable peak.[34, 35]

The measures derived from GRF provide a means of quantifying bilateral symmetry of LL and limb unloading (LU). Individuals with hemiparesis are characterized by asymmetric LL and LU. Hemiparetic subjects do not put as much weight on their

paretic leg as on the non-paretic leg.[36-41] Since walking requires the full body weight to be borne on either leg during single limb support phase, therefore, learning to load and unload the paretic leg is important for balance and gait training in stroke patients.[42] Studies have shown that individuals with stroke have difficulty leaning their body as far as possible in all planes.[43, 44] In addition, when shifting weight from a two-legged stance to one-legged stance or when stepping on stairs of various heights stroke patients show most difficulty in transferring weight to the paretic leg.[7, 45, 46] Some studies have reported that the loading of the non-paretic leg is difficult too, which could be explained as either due to subtle neuromuscular impairments ipsilateral to the brain lesion or to a reduced ability to control weight shift toward the non-paretic side using the leg and hip muscles of the paretic side.[10, 37, 45] This suggests that the speed and precision of the weight bearing of both the paretic and the non-paretic leg are affected by stroke and thus the stroke patients need more time to shift weight to the paretic leg than to the non-paretic leg.[8]

LL asymmetry and difficulties with actively redistributing weight while standing have been considered major contributors to disordered hemiparetic gait.[35, 47, 48] The ability to initiate and control voluntary weight shifts toward either leg is a pre-requisite for independent walking. Hence, the prevailing clinical assumption suggests practicing self-generated weight shifts in the frontal plane within the base of support would be essential ability to train and monitor these patients.[39, 46, 49] However, a recent investigation did not support the view point that a reduction in the LL asymmetry during static or quasi-static posture would result in improved hemiparetic gait. They concluded that the people, who want to learn to walk, must be trained to walk.[40] Therefore, the treatment strategies

based on the static analysis of posture and balance function may not be appropriate for dynamic activities, like walking, in which significant acceleration of body segments are occurring. Similarly, gait analysis may provide clinicians with an improved understanding of specific problems that cause instability and falls. This is the underlying viewpoint of the first study, in which we aim to investigate LL and LU and its relation to various walking parameters post-stroke.

**Neurophysiology of LL and LU.** The significance of LL and LU has been studied and reported both in animal and human literature. LL has been regarded as an important afferent input which assists in generating a stepping pattern in absence of supraspinal input.[50, 51] These afferent inputs are interpreted in the lumbar motorneuron pool in humans.[52] Furthermore, Dobkin et al.[51] and Dietz et al.[53, 54] suggested that the human lumbosacral spinal cord can generate a step-like oscillating EMG pattern when adequate and appropriate sensory information associated with weight bearing is provided. In addition, Harkema et al.[52] showed that the EMG activity in the lower leg muscles of spinal cord injury (SCI) person is affected by the level of the load carried on the soles during treadmill stepping, implying that the mean EMG amplitude is related to peak load rather than the peak stretch/ stretch loading. This suggests that the LL and LU are important components of walking and therefore, need to be assessed and quantified adequately during walking to facilitate better understanding of the hemiparetic gait, which might help us design better rehabilitation techniques.

**Non-neurological changes due to asymmetric loading in individuals with hemiparesis after stroke.** Hemiparesis following stroke is not a unilateral motor control problem as often considered and reported, but affects both the paretic and the non-

paretic sides. The difference in the performance of the paretic and the non-paretic limb can be either: 1) functional (or compensatory), 2) due to the neurological damage caused by the stroke or 3) structural or musculo-skeletal, due to disuse or the asymmetrical use of the paretic and the non-paretic legs.

Asymmetry is one of the most important characteristic features of individuals walking post-stroke. Additionally, asymmetry in LL is also reported in individuals with amputations, poliomyelitis etc. There is ample evidence in individuals post-amputation that suggests osteo-arthritis of the joints in the intact limb (e.g. knee[55] and hip[56]) because of the changes in the direction of the ground reaction forces and greater LL.[57] Similarly, subjects with poliomyelitis have asymmetrical disease, with greater forces transmitted across the unaffected limb resulting in greater symptoms on the unaffected leg as compared to the affected leg.[58] Similarly, we hypothesize in the first study of this dissertation that there will be increased magnitude of LL on the non-paretic leg post-stroke, which could thus result in some biomechanical and structural changes. Thereafter, in subsequent studies we will investigate the performance of the non-paretic leg and importance of including the non-paretic leg as a part of the rehabilitation regime post-stroke to achieve maximum benefit.

**Joint moments and power**

The magnitude of power bursts are smaller on the paretic side as compared to the non-paretic side, and smaller in both the limbs as compared to healthy subjects walking at their comfortable speed.[23]

Recent research suggests that the power generated at the hip and ankle joints in the paretic and the non-paretic leg in individuals with hemiparesis is significantly related to gait speed.[12, 13, 59] Additionally, there are some abnormal patterns of power

generation in the paretic and non-paretic legs of individuals with stroke (figure 1-4a, b, c). In particular, peak power[12, 13] and positive work[60] of the hip (pull-off) and ankle (push-off) and negative knee power during pre-swing and initial swing on both sides have been shown to be positively related to gait speed in subjects with stroke. Furthermore, Kim et al.[12] reported an extra power burst in the frontal plane at the hip joint; and a very different pattern of power generation at the knee in the transverse plane and ankle in the frontal plane of the paretic leg as compared to the control subjects. These studies do report the abnormalities in the power generation or work done in the non-paretic leg but do not consider either the influence of walking speed [60] or do not use a precise quantification method.[12]

In a seminal work Olney et al.[60] reported that the non-paretic limb is responsible for performing a greater proportion of work at all speeds, but the work done by the non-paretic leg is less than the work done by the healthy controls at their self-selected speeds. Furthermore, more negative work is done by the non-paretic knee in the later part of stance phase of walking. This indicates that the energy absorption occurs in the knee extensors, while the knee is flexing[12]. Furthermore, Olney et al.[13] demonstrated in their regression model that the ankle and hip joint positive power of the non-paretic leg were significantly related to walking speed.[12]

The inverse dynamics analysis reflects only the net result of active and passive muscle forces acting around the joint. However, it is noteworthy that the contribution from the passive structures (non-muscle fibers) around the paretic ankle joint in hemiparetic individuals is much greater than the healthy controls (2-49 % as opposed to 5.9 to 6.5%).[61] The increased passive stiffness may compensate for the reduced

moment output due to the weakness of the paretic ankle.[61] Therefore, kinetic variables alone do not provide detailed insight into the underlying pathophysiological mechanism in locomotor function post stroke, but kinetic analysis in combination with the electrophysiological techniques e.g. electromyography (EMG) may be able to provide greater understanding of the hemiparetic gait.[62]

**Electromyography**

Walking involves a series of tasks which are accomplished by muscle activity of adequate magnitude and duration which can be recorded as electrical signals from electrodes placed on the skin. This technique of evaluating and recording the electrical activity produced by muscle is called the EMG.[63] However, following a stroke the neuromuscular control of walking undergoes radical changes which include muscle weakness on the body side opposite to the lesion resulting in changes in spatial and temporal organization of muscle activity.[64]

One of the most important impairments after central nervous system injury is the inability to generate normal levels of muscle force.[65] This lack of force generation influences the success of the individuals trying to accomplish many tasks, like walking. Gait in subjects after stroke is associated with abnormal muscle activation patterns (magnitude and timing). However there are large inter individual differences in the EMG of individuals post-stroke. The commonly reported deviations include; a. premature activity of the paretic calf muscles during preswing and initial stance[66, 67], b. prolonged stance activity of the hamstrings and quadriceps muscles in both the paretic[24, 68, 69] and the non-paretic leg[69, 70], c. the absence of activity in the paretic ankle dorsiflexors during the late swing and early stance phase.[66]

In a seminal work relating to EMG in individuals post-stroke, Knutsson and Richards[24] provided an objective criterion for identification of muscle activation patterns during gait. They attempted to classify EMG patterns across subjects with stroke into 3 types based on the examination of timing and amplitude of the muscle activation on the paretic side. Type I (spastic) was characterized by a premature activation of the calf muscle during the stance phase. Type II (paretic) showed abnormally low levels of activation in most muscle groups and Type III displayed co-activation that spread to several or all muscle groups. Thereafter, Shiavi et al.[69] applied this classification to both the paretic and the non-paretic side post stroke. They examined bilateral changes in EMG activation taking place with motor recovery post-stroke from 1 to 10 weeks, with follow-up ranging from 6–24 months. They reported an abnormal synergy exhibited by the contralateral limb (non-paretic) was almost always a co-contraction.

## Interlimb Coordination and Need for Studying the Non-paretic Leg

Humans and other primate and non-primate animals are capable of coordinating various limb movements to accomplish a task. A remarkable spatio-temporal coordination is evident in spite of the large differences in the inertial characteristics of the effectors involved. This suggests the existence of some basic coordination principles that apply across widely different cooperative ensembles.[71]

### Evidence in Animal Literature

Interlimb coordination is one of the principal components of effective locomotion. This suggests that the movements of the limbs producing gait must be linked to each other to maintain equilibrium. Evidence from animal literature suggests that the contraction of muscles on one side of the body leads to an increase in excitability of the contralateral homologous motor pathways.[72] For example, Rossignol et al.[72]

demonstrated in spinal cats that continuously imposed flexion on one hind limb blocked the rhythm on both sides, suggesting that the rhythmic activity in one limb is influenced by the manipulation of the other leg. This conveys that the spinal cord contains some mechanisms for interlimb coupling. Similarly, the reduction of input on one side produces deficits on the contralateral side. For example, Guiliani et al.[73] showed unilateral deafferentation in spinal cats disrupted both ipsilateral and contralateral stepping. In addition to the spinal cord, brainstem and other sub-cortical structures are believed to play an important role in coordination. In experiments in cats with hemisected brainstem it was shown that when one hind limb is prevented from moving, the other hind limb continued its stepping behavior while rhythmic alternating bursts persisted in the stationary leg.[74, 75] This suggests that the influence of sensorimotor state of one limb over the other and signifies the role of the spinal and the supra spinal structures in walking.

**Evidence in Human Literature**

**Evidence in upper extremity literature**

Following unilateral stroke, neurological symptoms are mainly expressed on the side contralateral to the damaged hemisphere. However, careful investigation reveals that the side ipsilateral to the lesion also shows various deficits, such as impaired manual dexterity[76-78], muscular weakness[79-81], and reduction of sensory-motor function.[76, 79, 82] Significant differences have also been reported between healthy control and non-paretic hand for fine and gross manual dexterity, motor coordination, global performance for two of four tasks, and thumb kinesthesia.[76]

Colebatch and Gandevia[79] found that the unilateral damage to motor cortex or its projection produced weakness in the ipsilesional limbs. Ipsilateral deficits in hemiparetic

subjects might be due to the interruption of the ipsilateral projection of the corticospinal tract. Although most of the corticospinal fibers decussate in the medulla, a significant proportion remains uncrossed and forms the ventral corticospinal tract.[83] In addition, it is possible that a lesion in one hemisphere resulting from a vascular cause interrupts corticobulbar and corticoreticular projections and consequently affects subcortical structures involved in motor control.[84] Therefore, this suggests that the integrity of these descending pathways is necessary to achieve some motor performance on the non-paretic side.

**Evidence in lower extremity**

The regulation of human walking requires a close coordination of muscle activation between the two legs. During the stance and swing phase of gait, both legs act in a cooperative manner, each limb affects the strength of muscle activation and the time-space behaviors of the other.[85] Moreover, from a functional perspective interlimb coordination is necessary to keep the body's center of gravity over the feet.[86]

Recently, Dietz et al.[87] demonstrated that unilateral stepping movements can lead to a patterned activation of the contralateral but rhythmically loaded leg in individuals post-SCI. They suggested that this influence of locomotor activity on the contralateral static leg is based on the interlimb coordination. However, the patterned activity in the non-moving leg was mostly restricted to the leg flexor muscles.[87] The preserved activation of the leg flexor, but strongly reduced EMG activity in the leg extensor muscles was suggested to be due to the well established differential control of leg flexor activity.[85, 88] During normal locomotion the leg extensor activity is modulated continuously by proprioceptive feedback during the stance phase. Therefore, in the

condition mentioned above, with a lack of roll off of the body over the standing leg the EMG activity is expected to be diminished.[86]

In addition to the evidence in the spinal cord literature, interesting evidence exists regarding the role of the non-paretic leg, post-stroke in coordination during pedaling.[11, 89] The pedaling literature post-stroke suggests that some level of impaired motor control in the paretic leg during bilateral pedaling is due to synchronous motor activity of the non-paretic leg and the interlimb afference associated with the non-paretic LL and movement.[89] It was also suggested that due to disruption of the normal task-dependent suppression of interlimb influences, the paretic motor pattern was more impaired in bilateral pedaling than in unilateral pedaling. The greater impairment of the paretic leg output could be attributed to the coupling of pattern generation of the two legs.[89] Furthermore the unilateral non-paretic leg pedaling activated a rhythmic locomotor pattern in the paretic leg of subjects with severe hemiparesis, whereas unilateral pedaling in non-disabled control subjects did not activate a contralateral, rhythmic locomotor pattern.[89] Additionally, the induced pattern in the paretic leg was nearly identical to the pattern observed in bilateral pedaling in the hemiparetic subjects. Therefore, they concluded that probably the interlimb influences are substantially responsible for the impaired motor pattern of the paretic leg during pedaling.[89] Thus, extrapolation of these results suggests that the therapeutic interventions targeting alterations of the sensorimotor state of the non-paretic leg may be highly effective in improving the motor output pattern of the paretic leg and thus facilitate better walking pattern.

### Evidence of the Role of Non-Paretic Leg in Walking

The role of the non-paretic leg has been identified in achieving faster speed in individuals walking post-stroke. Kim et al.[12] found a high correlation between the gait speed and the non-paretic leg saggital plane kinetics, particularly hip flexors at pull-off and ankle plantarflexors during stance and push-off. Similar results were supported by the regression models of Olney et al.[13], which included hip and ankle power variables from both the paretic and the non-paretic side. Parvatneni et al.[59] also demonstrated a significant correlation of the non-paretic hip extensor power in individuals walking faster after stroke. Several other studies have shown that the muscle strength in the non-paretic leg is highly correlated with gait speed.[90, 91] These studies support the contribution of the non-paretic leg performance in the gait of persons with stroke. The high correlation between the gait speed and selected variables non-paretic leg, the performance of the non-paretic leg itself may be an important contributing factor to walking ability and justify the inclusion of the non-paretic leg in training.[12] Therefore, the intervention should not focus on training any single weak link, but should target the parameters on both the sides. Therefore, the evidence from the above mentioned studies strongly supports the idea and need to study the non-paretic leg in individuals walking post-stroke.

## Post-stroke Rehabilitation

### Traditional Gait Rehabilitation Therapies

Until the emergence of the evidence of the spinal modulation of walking pattern in humans, rehabilitation of walking post-stroke followed the hierarchical model of motor control.[92] This theoretical model hypothesizes that the central nervous system (CNS) is hard-wired and irreparable. Furthermore, an injury to the CNS was deemed permanent

and irreversible. Therefore, the rehabilitation strategies relied on compensatory strategies for non-remediable impairments and deficits. The aim of rehabilitation was thus to achieve successful completion of the task while the quality of movement was a secondary aim.

In late 1950s and 1960s neurofacilitation techniques were developed. These techniques primarily focused on retraining motor control by facilitation or inhibition of movement patterns. According to these techniques, desirable movement patterns should be facilitated while massed patterns or synergies should be inhibited.[93] According to proponents of these techniques, the stereotypical movements result from a neurological disease and are not functional. Neurofacilitation techniques include Bobath approach, the Brunnstorm approach, and Propioceptive Neuromuscular Facilitation (PNF).

The Neurodevelopmental Technique (NDT) established by Bobath [94] assumes that an abnormal postural reflex activity is the major cause of dysfunction, and therefore a significant proportion of therapy time involves inhibiting spasticity and other abnormal responses. In the Brunnstrom technique, synergistic movements are used to strengthen and practice single movements.[95] PNF techniques consist of assisted isometric and isotonic leg flexion-extension exercises, which are thought to improve strength and control of leg musculature in preparation for walking.[96] If a patient is unable to successfully maintain/ achieve a posture, the therapist would provide/instruct to use assistive devices and braces or suggest greater reliance on the non-paretic side. These strategies would assist the individual in resuming almost pre-injury levels of mobility

using remaining physical strengths and external aids, but is not equivalent to recovery or restoration of pre-injury capabilities.[97]

**Activity Based Gait Rehabilitation Therapies**

Over recent years, there has been a shift in focus from the nervous system being hard-wired to it being malleable.[98] This property of the nervous system led to the understanding of the ability of the neural systems to undergo plastic changes (neuroplasticity). Neuroplasticity is the mechanism by which the brain encodes experience and learns new behaviors. It is also the mechanism by which the damaged brain relearns lost behavior in response to rehabilitation, referred to as activity dependent plasticity.[99] This ability of the nervous system to change and learn presents strong potential for the resumption of functional activities after a nervous system injury if the training conditions are optimized or are task-specific, thus providing rehabilitation strategies alternative to the traditional approach.

**Evidence of neuroplasticity following therapy from animal literature**

Plautz et al.[100] trained adult squirrel monkeys to perform a motor task that required pellet retrievals from a well (small/ large) to induce repetitive use of a limited set of distal forelimb movements. Detailed analysis of the motor behavior of the monkeys indicated that when monkeys were trained to retrieve food pellets from a small well, requiring skilled use of the digits, progressive increments in motor performance were seen. On the contrary, monkeys trained to retrieve pellets from a larger food well did not acquire any additional skill. Comparisons between pre-training and post-training maps of cortical movement representations revealed no task-related changes in the cortical area devoted to the hand in the large well group. Movement specific changes in the small well group were large and consistent, and corresponded to the actual movement

kinematics of the task.[100] They concluded that repetitive motor activity alone does not produce functional reorganization of cortical maps. However, skilled activity practice is a prerequisite factor in driving representational plasticity in motor cortex.[100]

Furthermore, several critical insights into the mammalian neural mechanisms controlling stepping have also been made. It was discovered that the adult cats with low thoracic spinal transection could recover hindlimb stepping by walking on a moving treadmill belt when given trunk support, assistance in paw placement and optimal sensory input.[50] Furthermore, Forssberg et al.[101, 102] and Grillner et al.[103] showed that the kinematics and EMG of spinal kittens were similar to normal cats and the spinalized kittens possessed the ability to adapt to various speeds of the treadmill. This emphasizes the importance of the peripheral afferent signals in adapting the locomotor pattern to external conditions. Along these lines of work it was shown that the age of spinalization and the amount of training in the treadmill had important effects in the locomotor pattern.[104, 105] Animals spinalized at 2 weeks of age had a much better locomotor performance than those spinalized at 12 weeks of age. Training in 12 week old cats had an observable effect on weight bearing during locomotion. On the other hand Barbeau and Rossignol[50] demonstrated that appropriate training strategies (duration and intensity) could produce stepping recovery in adult cats as well. Adult cats spinalized at a lower thoracic level were trained in the treadmill environment two/three times per week. Over periods of several weeks up to one year of training deficits in balance and volitional movements remained. However, cats remarkably exhibited the ability to maintain hindquarter weight support and to generate rhythmic, coordinated stepping with overall similar joint excursions and EMG.[106, 107] The neural networks

capable of generating rhythmic motor activity in the absence of sensory feedback are termed Central Pattern Generators (CPG).

Another remarkable effect of treadmill walking in the spinalized cats appears to be the specificity of training. This implies that in order to learn how to walk, walking needs to be practiced and the neuromotor skill learned during walking does not translate to performance of other tasks e.g. standing. For example Hodgson et al.[106] randomized spinalized cats (lower thoracic) into untrained, standing-trained and treadmill trained groups one month post-transection. All the cats were given 2-3 months of training based on the groups they were placed. Following stand training, cats could maintain a standing posture for an extended period of time without any stimulation to their tails; however the cats in this group could not step; except for a couple of cats which could take few uncoordinated steps at speeds below 0.2 m/s. On the other hand, the cats in the step trained group could step at faster speeds (0.62 m/s) but required maximal stimulation at their tails to maintain standing. Follow-up experiments included the stand or step training just one week after transaction and continued for six to eight months, then crossed the cats to the other training group. After the cross-over the cats learned to perform the original tasks, i.e. if they learned to step before switching to the stand training, they had difficulty stepping, but could stand well. This emphasizes the effect of the specificity of training. Following the strong evidence of the ability of retraining walking post- spinal cord transaction in cats, the task specific therapy was employed in individuals post-stroke or spinal cord injury.

**Evidence from human literature**

Neurorehabilitation is increasingly taking into account novel scientific findings from the basic science. The gait strategies have been recently improved by providing

treadmill training with partial body weight support (BWS) combined with enforced stepping. This method is derived from information gained from animal studies showing that the adult spinal cord can recover a near normal walking pattern after a period of interactive locomotor training (LT) on a treadmill in which weight support for the hind quarters is provided, hence facilitating stepping on a treadmill.[50] On the basis of this evidence body weight supported treadmill training (BWSTT) has been employed in individuals post- spinal cord injury[108-110], stroke[111, 112], Parkinson's disease[113], Cerebral Palsy[114] and Down's syndrome.[115]

Several studies have been conducted in individuals post-stroke to test the effectiveness of the BWSTT as a method of rehabilitation of walking.[116, 117] Researchers have compared the ability of the patients following BWSTT with conventional physiotherapy techniques[112, 118], (example: NDT, PNF) or aggressive bracing techniques.[119] Hesse et al.[112] conducted a study in which they compared NDT with BWSTT. The study was carried out as an ABA single case study with seven patients. The first three week phase (A-phase) consisted of 30 min treadmill training each workday. The subsequent three week (B-phase) consisted of 45 min physiotherapy sessions daily followed by another A-phase. The gait parameters were reported to improve only during the A-phases.

BWSTT in chronic stroke individuals improves balance and gait ability. Individuals' post-LT walk at higher speeds associated with improved stride length, stance and swing time, initial and terminal double limb support phases.[116, 120-122] Additionally, the scores from the Rivermead motor assessment or stroke rehabilitation assessment of motor recovery[111, 123] were also improved post-LT. LT also improved cardiovascular status of

these individuals by improving their oxygen uptake.[117] However, there is no consensus regarding the effectiveness of BWSTT as a rehabilitation technique for individuals post-stroke. This could primarily be due to the lack of consensus regarding rules to administer and control this therapy. For example, it is important to note that most of the studies mentioned above focused on the performance of the paretic leg alone by employing a trainer to guide the movement of the paretic leg and a trainer on the trunk to facilitate weight shift and maintain upright posture. Moreover, the outcome measures reported were either reported for the paretic leg (stance time, double limb support time) or were focused at both the paretic and the non-paretic leg performance, or accomplishment of the task, for example gait speed, functional ambulation scores, or Rivermead motor scores. The non-paretic leg has been largely ignored through these studies and to our knowledge no study has included the non-paretic leg in the treatment protocol (except some studies using robotic devices for rehabilitation of walking) however, no study has characterized for the changes in the non-paretic leg following LT.

Walking is a bilateral task, which requires a coordinated output from both the legs, and the performance of one leg largely influences the output of the other leg. Therefore to improve the overall outcome of the rehabilitation of walking post-stroke, both paretic and the non-paretic legs should be included in the rehabilitation protocols. Furthermore, assessment of the non-paretic leg performance post-LT would help us to discern if the locomotor training improves walking function by promoting recovery and thus reduced dependence on the non-paretic leg, or compensation by increased dependence on the non-paretic leg, or adaptation by development of a new and different motor control

pattern. This would further improve our understanding regarding the mechanisms underlying BWSTT as rehabilitation techniques.

**Relevance of the Reviewed Literature to the Studies of the Dissertation**

Asymmetry is one of the cardinal signs of walking post-stroke and has been often reported as one of the primary problem in successful accomplishment of certain tasks involving participation of both the limbs, e.g. walking. Asymmetry, i.e. greater contribution of the non-paretic leg, has often been reported without being considered to have any pathological importance. Asymmetry has been reported in spatio-temporal, kinematic and kinetic measures, but not much information is available regarding the asymmetry of kinetic variables during walking. Since LL and LU form an important part of the walking task and are reported to be affected by stroke, through the first study of this dissertation, we aim to investigate the magnitude, pattern and temporal aspects of LL and LU and assess asymmetry (if any) between the paretic and non-paretic leg LL and LU during waking. Since the kinetic parameters are the result of muscle activity, in the next two studies the analysis will concentrate primarily on the EMG and the secondary analysis will include relevant kinetic parameters.

Quantification of the muscle output of the non-paretic leg may provide greater insight into the motor control of walking post stroke. Furthermore, in the hemiparetic population it is still unclear how the non-paretic leg muscle activity may contribute to walking performance. There is some evidence of the compensatory mechanisms of the non-paretic leg aimed at assisting in achieving a steady walking state. However, there is no single large study to provide detailed information regarding these compensatory mechanisms used by the non-paretic leg. Therefore, investigation of the relationship between muscle activity and the biomechanical variables would help determine changes

in the non-paretic leg muscles in order to maintain a steady walking state. Thus, the second study of this dissertation aims to quantify the muscle activity of the non-paretic leg and determine its relation to biomechanical characteristics of hemiparetic gait.

Overall current evidence suggests that BWSTT is an efficient and effective rehabilitation technique to promote walking function in individuals with acute and chronic neurological injuries. However, there is limited or no quantitative information about the changes in the motor control of the non-paretic leg and it is unclear how the changes in the motor control of the non-paretic leg relate to the biomechanical parameters of walking performance. Therefore, the third study of this dissertation aimed at quantifying the response of the non-paretic leg muscle activity to locomotor training and explaining the relation to selected biomechanical measures of walking performance post stroke.

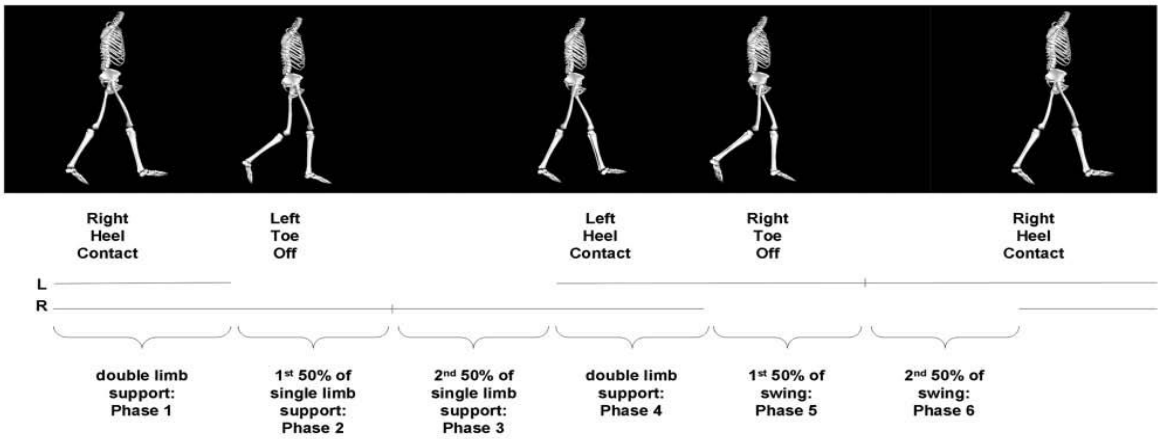
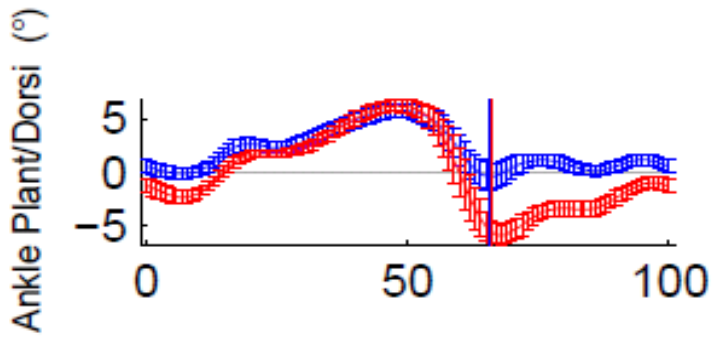
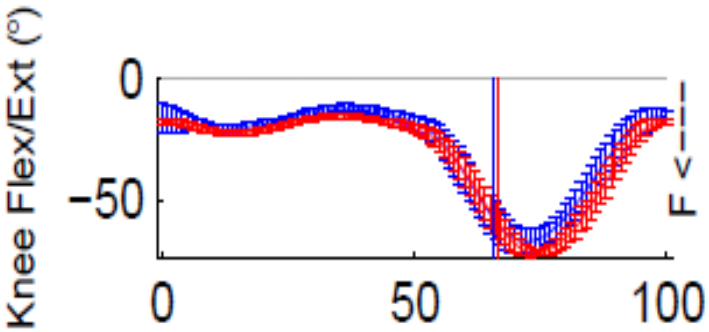



Figure 1-1. Represents different phases of the gait cycle. Phase 1 represents the first double limb support, phase 2 represents the first 50% of the single limb support phase, phase 3 is the second 50% of the single limb support phase, phase 4 represents the second double limb support phase and phase 5 and 6 represent the first and second 50% of the swing phase. (From: Bowden MG, Clark DJ, Kautz SA. Evaluation of Abnormal Synergy Patterns Poststroke: Relationship of the Fugl-Meyer Assessment to Hemiparetic Locomotion. Neurorehabil Neural Repair 2010 24(4) 328–337.)

a.




b.




c.

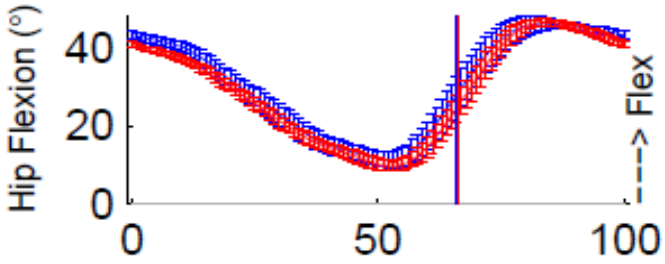
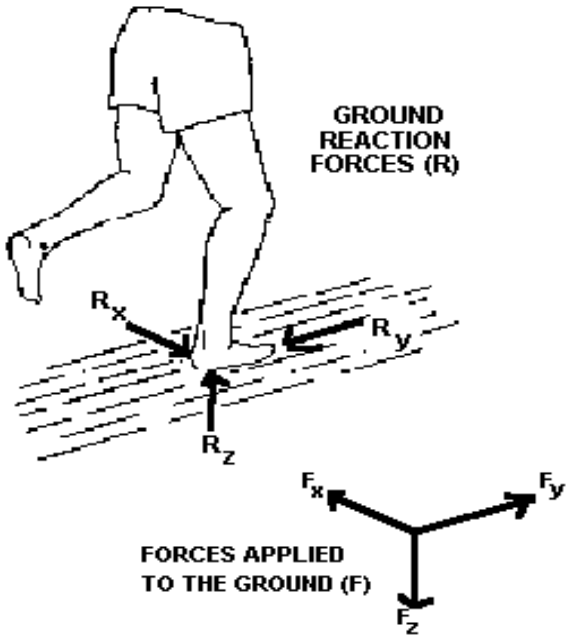



Figure 1-2. Represents hip, knee and ankle joint angles subject walking. a) ankle angle, b) knee angle and c) Hip angle in a healthy subject walking at self-selected speed. Blue and red represent each leg.

a.




b.

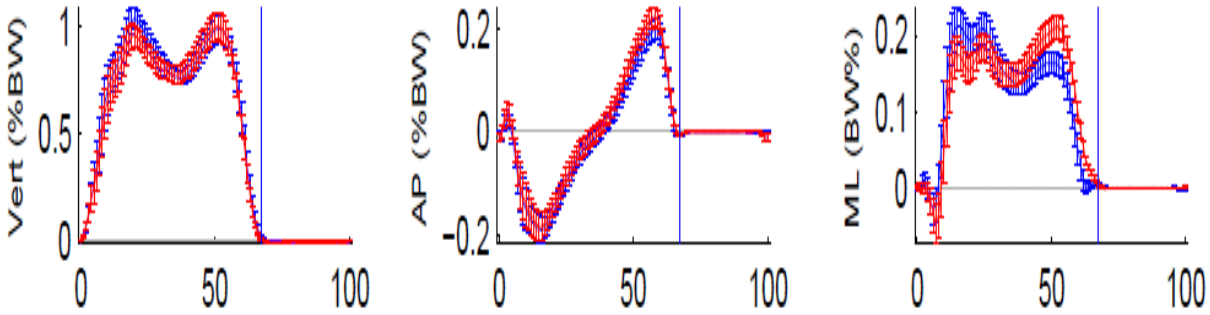
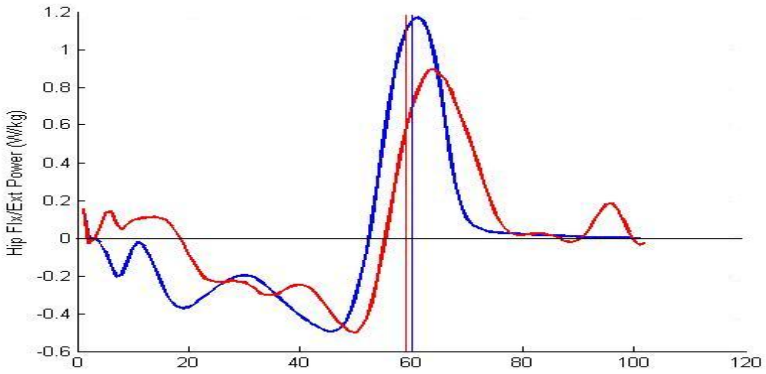



Figure 1-3. Represents the ground reaction forces. a) Figure illustrates the 3-D forces applied to the ground from the legs (Fx, Fy, Fz) and the measured Ground reaction forces (Rx, Ry, Rz). x corresponds to medial-lateral, y corresponds to anterior-lateral and z corresponds to the vertical component of the ground reaction forces. (Adapted from http://moon.ouhsc.edu/dthompso/gait/kinetics/GRFBKGND.HTM, assessed on 10.17.2010). b) Figure represents the vertical, anterior-posterior and medial-lateral ground reaction forces recorded from a healthy individual walking at self-selected walking speed. Red and blue plots represent ground reaction forces in two legs.

a.

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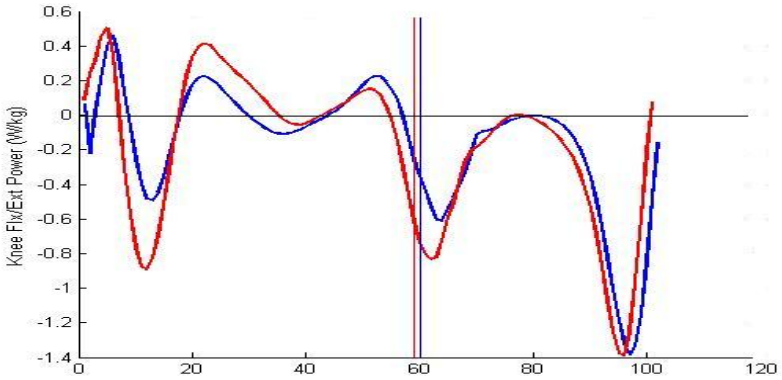


c.

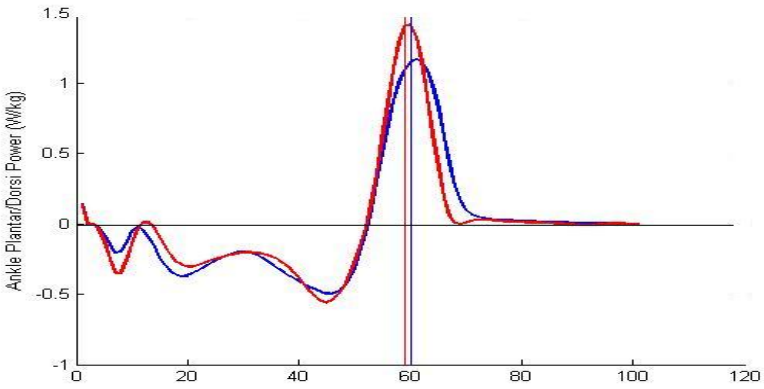


Figure 1-4. Represents power profiles in hip, knee and ankle subject walking at self-selected speed. a) Hip power, b) knee power and c) ankle power in a healthy subject walking at self-selected speed. Red and blue plots represent ground reaction forces in two legs.

# CHAPTER 2
# RATIONALE FOR VARIABLE SELECTION AND ANALYSIS

## Background

Hemiparesis post-stroke is characterized by asymmetry and incoordination between the paretic and the non-paretic leg, resulting in an altered walking pattern. Asymmetry has been reported in terms of spatio-temporal[31, 48], kinematic (joint angles)[27, 124], kinetic (GRF, power and work)[12, 59, 60] parameters and muscle activity (EMG).[69, 125, 126] Therefore, these variables form important components for describing the characteristics of movement.

GRF can be recorded directly on force plates and the vertical component of the GRF represents the LL and LU) ability. LL and LU has been reported in static and quasi-static positions in individuals' post-stroke.[7-10, 44] However, not much has been investigated with respect to LL and LU during walking. Walking involves movement of center of mass out of the base of support as opposed to static and quasi-static positions where center of mass is always within the base of support. Therefore, it is imperative to assess LL and LU ability in the task of walking.

### Hemiparesis: Unilateral vs. Bilateral

Hemiparesis post-stroke, as the name suggests is considered a unilateral motor control problem of the paretic leg, and clinicians and researchers generally consider the non-paretic leg as unaffected. Therefore any changes in the non-paretic leg output are considered a result of compensation for altered coordination of the paretic leg. For example, there is evidence in the literature that demonstrate significant correlation between the non-paretic hip, ankle joint power [59] and work [60] and walking performance

post-stroke signifying its role to accomplish the coordinative task of walking.[12, 13] On the other hand, some studies have indicated altered non-paretic leg muscle activity as compared to healthy control subjects, for example, e.g. Shiavi et al.[69] and Buurke et al.[126] reported changes in the EMG activity of the non-paretic leg and suggested they could be due to some residual impairment in the non-paretic leg after stroke.

**Evidence of Bilateral Involvement in Upper Extremity**

Evidence in the upper extremity literature supports bilateral limb involvement post-stroke. For example, a study demonstrated that the hemiplegic subjects were impaired in operating a steering wheel with their ipsilesional limb.[82] Brodal, a neuroscientist observed that his handwriting was less coordinated after a stroke in the internal capsule ipsilateral to his dominant hand.[127] Colebatch and Gandevia[79] found that unilateral damage to motor cortex or its descending projections produced weakness in the ipsilesional limb. These and other results[76, 128-131] suggest that damage to one hemisphere may have a bilateral effect on the control of movement.

**Causes of Bilateral Limb Involvement Following a Unilateral Lesion**

There is no consensus regarding the bilateral involvement post-stroke, but several neurophysiological mechanisms have been reported to identify involvement of both sides after stroke. Deficits in hemiparetic subjects might be due to the interruption of the ipsilateral projection of the corticospinal tract. Although most of the corticospinal fibers decussate in the medulla a significant number remain uncrossed.[83] Furthermore, the lesion in one hemisphere might interrupt corticobulbar and corticoreticular projections and consequently affect sub-cortical structures involved in motor control.[84] The presence of a bilateral component in motor control is supported by observations that a functional activation might have a bilateral expression in cortical areas.[132] The abnormal

ipsilateral descending output (from the contralesional hemisphere), associated with a unilateral cortical lesion, may contribute to the ipsilateral limbs abnormal function.[128, 130] Similar mechanisms might be responsible for the altered performance of the non-paretic leg. In addition, there might be greater contribution of the spinal structures in the control of lower extremity functioning as compared to the upper extremity.

## Goals of Studies of the Dissertation

Three studies were conducted as a part of this dissertation to evaluate and understand the motor control of hemiparetic gait and its response to task-specific therapy.

**Study1.** The first study of this dissertation attempts to investigate LL and LU asymmetry during walking post-stroke and its relation to functional walking status. We also examined how LL and LU asymmetry relates to some of the kinematic changes in the leg to maintain steady walking state.

**Study 2.** The analyses of the second study of this dissertation seek to inspect the changes in the muscle activity in the non-paretic leg and whether there is evidence to support that the non-paretic leg is affected after stroke. It is important to mention that the performance of the non-paretic leg will be evaluated during bilateral walking and not in an experimental condition specifically designed to examine it. Therefore, it would not be possible to explicitly delineate the abilities of the non-paretic leg. However, there has been little attention paid to the non-paretic leg performance, so this study fills the gap in our understanding.

**Study 3.** The third study of this dissertation will answer the question of whether the motor pattern of the non-paretic leg (identified in the second study) can be changed following a task-specific LT. We will evaluate if the pattern of the non-paretic leg

normalizes following the LT and if it appears to do so by reduced compensation or remediated impairment.

## Variables Used

This section discusses the variables selected for each study to accomplish the goals of analyses, benefits and limitations of the variables selected and other potential variables and why they were not selected for the analyses.

### Study 1

In this study we examine LL and LU asymmetry during walking and its relation to kinematic measures of the leg (knee angle and leg angle). LL and LU occur during the two double limb support phases of the gait cycle, (where one limb is being loaded and the other is being simultaneously unloaded). Therefore all the parameters of this study will be evaluated in these two regions only. The parameters of this study are discussed below.

#### Measure of limb loading and unloading

**Average normalized vertical GRF.** This measure will be used to capture the magnitude of LL and LU in the paretic and the non-paretic leg during the two double limb support phases while walking on an instrumented treadmill.

**Benefits.** GRF is based on Newton's third law of motion and is equal and opposite reaction of the supporting surface to the body.[33] For example, an individual standing on the ground exerts a force on it (i.e. body weight), simultaneously the ground exerts an equal and opposite force on the person, which represents the vertical GRF. An average value of vertical GRF takes into account all the points of the LL and LU phase of the vertical GRF curve. Additionally, normalizing the force value with each subject's own body weight removes confounds that people with greater body weight will

have greater GRF (load). This facilitates inter-subject comparisons. Vertical GRF value of 0.5 implies symmetric LL and LU, whereas a value < 0.5 suggests reduced loading (e.g., a lower amount of body weight supported) and a value > 0.5 means that there is greater LL.

**Limitations.** Average normalized vertical GRF might be affected by walking speed (because vertical GRF is greater during faster walking speeds[133]). To avoid this we grouped subjects based on their self-selected over ground walking speed (≤ 0.4m/s = Household ambulators, 0.4m/s-0.8m/s= Limited community ambulators, >0.8m/s= community ambulators).[15]

**Other related measures used in literature.** LL and LU has been reported in static and quasi-static tasks by using center of pressure displacement[8, 45, 134], peak vertical GRF[135], and the weight borne by each extremity as measured by force plates[136] and weight scales.[46] The center of pressure displacement and velocity provide spatial information of the vertical GRF instead of magnitude of LL and LU. Furthermore, physiologically the peak of LL occurs later than the LL defined for the first study. Therefore, the peak value has little significance for the purpose of our study.

**Timing of limb loading and limb unloading**

For the purpose of this study, timing is represented by “cross-over point”. The cross-over point is defined as the specific point in time when the two vertical GRF curves cross each other. It represents the percent gait cycle from the beginning of the double support phase until the majority of weight shifts from the leg in second double support period to the leading leg in first double support phase.

**Benefits.** Timing of LL and LU is an important parameter, because Dequervain et al.[29] suggested that the time spent in the pre-swing phase is an important predictor of

the walking speed of individuals post-stroke. Cross-over point quantifies the moment of shift of the predominance of the weight, irrespective of the total amount of weight transferred or the peak. It provides accurate and quantifiable information about the time (both as percentage of double limb support phase and absolute time in seconds) it takes to shift the predominance of weight. The cross-over time in the healthy control subjects (based on the preliminary analysis of study 1) is about 44±4.6% of the double limb support phase of the gait cycle. This means that at about 44% of the double limb support, the predominance of weight shifts from one leg to the other.

**Limitations**. It is an accurate measure, with potentially no major limitations. However, an instrumented split-belt treadmill is required to simultaneously record GRF from both legs during steady state walking.

**Other related measures used in literature**. Loading time has been quantified in the literature as time to peak vertical GRF[137, 138], and weight transfer time.[135] Time to peak vertical GRF quantifies the time it takes vertical GRF to attain its peak value. However since we desired to capture the moment of the shift of the predominance of weight from one leg to the other (which actually signifies the loading and unloading time), time to peak does not provide that information. Furthermore, owing to the altered LL and LU ability, individuals post-stroke may have more than one peak GRF or no specific peak (as seen in individuals with a plateau type LL).

A second measure used to report temporal characteristics of LL and LU is weight-time transfer.[135] It is defined as the duration from the first change in the vertical GRF data (change for either limb of more than 2% of body weight from the mean baseline measurement during quiet standing) to when the force beneath the leading limb reaches

zero, indicating that the limb has lost contact with the ground.[135] This is a good measure, but it represents duration (period) from the first change in the vertical GRF data, instead of quantifying the moment of weight shift as required in this study.

**Pattern of limb loading and limb unloading**

The pattern of LL/LU will be calculated from the shape of the vertical GRF curve. The shape of the vertical GRF curve will be quantified using the best fit curve for polynomial in JMP statistical software (SAS, Cary NC) for the values of the loading phase (heel strike to just the beginning of single limb support) and unloading phase (end of the single limb support to toe off) for each subject (highlighted with black circle in figure 2-1). A significant first order term will imply a linear slope, whereas a significant second order coefficient term will indicate the LL/LU profile has a curvature (positive coefficient is concave and negative coefficient is convex).

**Benefits**. It is a simple and elegant method of quantifying the shape of the curves to understand the pattern of LL and LU. Quantification of the LL and LU pattern will reveal variability in performance of each individual which thus will facilitate classifying individuals post-stroke into different categories with respect to the pattern of LL and LU.

**Limitations.** Some subjects might have very irregular vertical GRF curves that are not accurately quantifiable by this technique. However, a closer look at the data revealed that we had only a few subjects with very irregular LL and LU patterns and we did not include them in the analysis.

**Other potential measures.** No one has calculated the shape of the vertical GRF curves, but pattern of loading and unloading has been quantified using center of pressure path.[8, 45, 139] However, the center of pressure path provides information about

the path traversed by the point of action of force rather than the actual pattern of LL and LU.

Loading rate has also been used to quantify the slope of the curve[140-142]; however, it gives a single number that quantifies the LL per unit time irrespective of the shape of the curve. Quantifying the LL and LU using the polynomial fit provides us more details about the variability and alterations of LL and LU pattern. For example, LL and LU in two individuals, one with a concave slope and the other with a convex slope have similar net loading rate. This could be because they presented with similar changes in the force magnitude per unit time. On the other hand, the polynomial fit method would group them both into different groups based on the difference in the shape of loading and would inform that the person with concave loading had initial reluctance towards loading, while the person with a convex loading initially, rapidly loaded their limb, but slowed later on.

**Kinematic parameters**

**Medial-lateral leg angle.** The average medial-lateral leg angle is defined as the angle between the vertical and the projection of the line connecting the center of mass of pelvis and the center of mass of foot in the frontal plane. The leg angle reduces in the first double limb support (LL) phase (e.g., the pelvis is moving toward the foot for the entire phase, with a change of angle of approximately 7 degrees during this phase for control subjects walking at 0.3m/s) and it increases in the second double limb support (LU) phase (e.g., the pelvis is moving away from the foot for the entire phase, with a change of angle of approximately 7.5 degrees during this phase for slow walking control subjects). Thus, this average will characterize how close the pelvis got to the loading leg for the full the loading period, as opposed to only characterizing the value achieved at

the end of the loading phase. Since we aim to individually investigate the correlation between LL and LU and medial-lateral leg angle, we suggest average leg angle value could capture these changes in respective phases of the gait cycle and help us investigate the relation with the LL and LU.

**Average knee-flexion**. The average knee flexion-extension angle was calculated during each of the two double limb support phases of the gait cycle.

**Benefits.** We selected to investigate the relation between the medial-lateral leg angle and LL and LU post-stroke, because evidence in the literature demonstrates that lateral foot placement asymmetry (the wider paretic foot placement relative to the pelvis than the non-paretic foot), strongly correlates with the weight support.[143] We aimed to verify this in the context of our study. Furthermore, knee joint angle goes through a sequence of flexion and extension during the gait cycle. We believe that average is relatively repeatable and can be consistently interpreted, while other variables, like the peak or minimum knee angle or joint angle at a particular percentage of gait cycle have higher variability and are not as repeatable.

**Limitations**. Although knee angle and leg angle are significantly related to LL and LU (as discussed in study 1), they might be affected by other factors, like muscle strength etc. However, since we will be using a correlation analysis, a significant correlation would suggest role of knee and leg angle in LL and LU, in addition to the others factors which might be affecting them.

**Study 2**

Muscle activity represents the sole mechanism that allows control over movement of the body segments. The force that is exerted by the muscle needs to be scaled appropriately for each instant of movement sequence, resulting in continuous changes

in the magnitude of output as a function of time. Furthermore, muscles act at joints at every step of the movement, which can be characterized as moments. These joint moments are then used to quantify the work done at the joints.

The EMG activity and the work done at the joint (ankle, knee and hip) are the primary variables of the analysis. In contrast to the paretic leg, the non-paretic leg may not have obvious alterations in its performance. However, the small differences might also influence the performance of the non-paretic leg and the success in accomplishing the task. Therefore, it is important to carefully select measures to capture the nuances of the non-paretic leg performance.

**EMG timing variable**

Well timed muscle activity is important to achieve well patterned walking. For the purpose of this dissertation EMG timing was recorded over the gait cycle and different regions of the gait cycle (regions of the gait cycle as described in chapter 1, figure 1-1).

**K-means analysis.** K-means cluster analysis is a statistical technique that groups similar data points in a single cluster. In the context of EMG analysis it differentiates data as “on” and “off” by finding similarities between the data points of the rectified and filtered EMG signal.[125] The individual data points are assigned to k-cluster by MATLAB (Mathworks, Inc., Natick, MA) such that the distance from the centroid is minimized. Therefore, the most similar data points are grouped together. The cluster with the lowest mean value corresponds to inactivity and the other clusters correspond to periods of muscle activity.[125] (figure 2-3)

**Benefits of using K-means cluster analysis.** The k-means algorithm is based on minimizing the sum of squared distances from all the points in every cluster to their corresponding cluster center.[125] It is a simple algorithm that can be applied to large data

sets to separate them into different partitions, which help in identifying “on” and “off” of EMG. Since k-means individually identifies each data point, it takes care of even the short duration of muscle activity, by assigning them to the closest cluster.[125] Short bursts of muscle activation are a common occurrence in non-paretic leg.

**Limitations of k-means.** The adequateness of k-means depends to some extent on the cut-off frequency of the low pass filter and on the number of clusters set a priori.[125] For this dissertation we set the number of clusters to five, because Den Otter et al.[125] suggested that five clusters could capture the muscle activity in individuals walking after stroke. In case of a homogeneously distributed data set (a muscle displays no activity or continuous activity) this algorithm fails to provide any useful information.[125] However, close inspection of the raw data reveals that there were no such EMG signals in our data set. Finally, there is some possibility of error due to initializing of the cluster centers, but by selecting cluster centers far from each other this type of error can be minimized. Furthermore, we tested the reproducibility of the k-means function by performing it on a set of randomly selected walking trials. Visual inspection of the results revealed that they were consistent over several attempts.

**Percentage duration of activation over the gait cycle.** Percentage duration of activity of a muscle in a gait cycle is sum of all the "on" points in gait cycle divided by the total number of points in the same gait cycle. The muscle "on" and "off" times are determined using k-means clustering for each point in the gait cycle.

**Percentage duration of muscle activity in a region of the gait cycle.** Percentage duration of activity of a muscle in a region is calculated by dividing the

activity of muscle in a region of the gait cycle by the total number of points in that region and multiplying with 100.

**Demonstration**. To calculate the percentage activation of TA (Tibialis Anterior) over the gait cycle, assume the gait cycle has X total points, TA had X1 on points in region 1(which had total points Y1), X2 in region 2 (which had total points Y2), X3 in region 3 (which had total points Y3) and on. Therefore, the percentage duration of activity in the gait cycle would be

((X1+X2+X3+X4+X5+X6)/ X)*100

And the percentage activity of muscle in the first region of the gait cycle would be

(X1/Y1)*100

**Benefits of using percentage duration of activity of a muscle over the gait cycle.** Quantifying muscle activation over the gait cycle provides global information related to the temporal patterning of the non-paretic leg. Since k-means clustering includes even the short bursts of activation, it captures all the bursts of activation. Furthermore, dividing the “on” points in the gait cycle by the total points, the analysis also takes into consideration the variability in duration of the gait cycle.

**Limitations of percentage duration of activity of a muscle over the gait cycle.** In addition to the limitations of the k-means analysis discussed above, the percentage activity of a muscle over the gait cycle is a global measure, which could obscure the important differences in different phases of the gait cycle. In order to obtain detailed characteristics of temporal patterning of muscle activity in the non-paretic leg, we also investigated the percentage duration activation of muscle in different regions of the gait cycle.

**Benefits of percentage duration of activity in different regions of the gait cycle.** It provides information regarding duration of muscle activity in different regions of the gait cycle. Detailed information of temporal characteristics of muscle activity indicates the region of the gait cycle when the non-paretic leg muscle activity is different as compared to healthy control subjects. This could further help to categorize muscle activity being due to compensation or impairment, as discussed later.

**Limitations.** The percentage duration of activation of muscle in a region provides detailed information related to the temporal characteristics of muscle activity. It does not have any limitation except the ones discussed for k-means cluster analysis, because k-means cluster analysis forms the basis of all the temporal calculations of EMG in this dissertation.

**Other potential measures.** Timing parameters are often derived from the EMG profiles by applying a threshold to detect onset and offset of burst activity. However, there is no consensus in the literature about threshold selection. Moreover, several researchers have suggested that these methods introduce large systematic errors.[144-146] Another method for temporal characteristics used by Mulroy et al.[147] was using an analyzer software to identify the “on” and “off” time for each packet of muscle activity that had an intensity of at least 5% of the maximal contraction for minimum 5% of the gait cycle. Kawashima et al.[148], on the other hand included muscle activity in their analysis if the muscle was consistently three standard deviations above the recorded resting activity. However, these quantification techniques may not be adequate for pathological gait because it might not be possible for an individual to maintain a true resting state, or a real maximal contraction (due to the pathology). Furthermore,

sometimes the EMG may be well timed to maintain a steady walking state, but it may be less than 5% of the maximum or three standard deviations from the resting. In the cases like this the data might be lost because of the threshold of activity required to quantify for inclusion. However, k-means cluster analysis has the advantage of being able to include even the short bursts of activity.

**EMG magnitude**

For the purpose of the studies of this dissertation, the magnitude of EMG activity will be represented by i) the integrated EMG activity over the gait cycle ii) integrated EMG activity of a muscle in different regions of the gait.

**Integrated EMG activity.** The filtered and rectified EMG signal is numerically integrated using a trapezoidal rule, an extended closed formula integration rule with equally spaced abscissas, with respect to time in each region and each gait cycle. Integration calculates total area under a curve, and numerical integration divides a curve into a bunch of component areas and sums these component areas together to get the total integral (figure 2-2).

**Benefits.** Integrated EMG is a robust measure of muscle activity and has been most often reported in the literature.[149] It has proven to be a useful method for evaluation of muscle activity and offers an instrument for documenting muscle rehabilitation. It is robust against movement artifact.[150]

We hypothesized that there would be significant differences in the magnitude of the muscle activity in the non-paretic leg and healthy control subjects. Therefore, the next step will be to investigate closely different regions of the gait cycle. In order to quantify the magnitude of muscle activity in different regions of the gait cycle we calculated the integrated EMG activity in each region of the gait cycle.

**Limitations.** The amplitude of the surface EMG is related to the net motor unit activity, i.e. recruitment and discharge rates of motor units.[151] However the amplitude of EMG detected using surface EMG is sensitive to many intrinsic (thickness of subcutaneous fat, distribution of motor units, conduction velocities and detection systems used), and extrinsic factors, i.e. the factors which can be influenced by the experimenter (orientation, location and area and shape of the electrode and distance between them[152]), which renders it highly variable. Furthermore, the reliability of the magnitude of the EMG in the dynamic contraction is also influenced by length and force change due to stationary shift in relative position of the electrode and changes in tissue conductivity.[153] Furthermore, change in the joint angles cause the recording electrodes to shift relative to active muscle fiber and the change in the muscle fiber direction to alter tissue conductivity. Additionally, the length of muscle fiber and the shortening velocity is also inversely related.[153] The factors discussed above might increase the variability and reduce the reproducibility of the EMG magnitude; however, we took careful measures to minimize the error due to extrinsic factors.

**Other potential measures.** To investigate the amplitude of muscle activity over the gait cycle, peak Root Mean Square and mean Root Mean Square have been often reported in the literature. Peak root mean square uses a single value[150], the maximum of the root mean square smoothed signal to represent muscle activation.[150] However, it is not robust against movement artifact and error inherent within the EMG signal.[150] On the other hand, the mean root mean square is a robust measure that limits the effects of movement artifact, however is less sensitive to changes in the EMG signal and may mask the differences in muscle activation and intensity between the experimental

conditions.[150] Therefore, integrated EMG activity is our best choice for the global EMG activation. Similarly, to obtain detailed information regarding the magnitude of activity in different regions of the gait cycle, integrated EMG was calculated. Each of these measures, if used adequately would provide important information about the muscle activation and the information obtained from each of them is similar. However, as discussed above, there are some inherent problems associated with each of them.

**Work Done**

Work done at a joint is calculated by numerically integrating the joint power curve using a trapezoidal rule, an extended closed formula integration rule with equally spaced abscissas with respect to time in each region and each gait cycle. Integration is finding the total area under a curve and numerical integration is dividing a curve with several component areas and adding these component areas together to get the total integral. A2, H1, H3 represent the most important and significant phases of positive work during the gait cycle and have been shown to relate to the walking performance.[12, 13, 59, 60] In addition, other source of power generation is, K2, i.e. positive power by concentric activity of the knee extensors while, A1, K1, K3 and K4 represent phases of negative work. H4 (hip abductor power in frontal plane) is an important source of positive power generation reported in individuals walking post-stroke[12], however it is not found in healthy subjects. Although Kim et al.[12] demonstrated that the magnitude of the joint powers in the non-saggital planes (esp. at the hip) are significantly related to the individuals walking performance (as measured by walking speed), stronger correlations were found with the hip and ankle joint in the saggital plane. Therefore, for the purpose of this dissertation we selected to study positive work done at the hip and ankle joint.

**Benefits.** Work done is an important measure of motor performance.[149] It closely represents musculo-tendon work[154] and has been used to examine locomotor deficiencies[155, 156], determine how the muscle functions[157]and identify impairments associated with neurological deficits.[59, 60]

**Limitations.** The primary limitation is the inability of work to account for individual muscle contributions, primarily due to co-contraction causing the net moment to be less than the absolute sum of the individual muscle flexor and extensor moments and muscle tendon energy storage and release that allows negative work in one phase to be recovered as positive work in a subsequent phase.[154] We aim to investigate the correlation between the work done at the joint and the muscles which might have an important contribution to work production.

**Other potential parameters.** Joint power could have been another important variable to investigate the contribution of different muscles. However, power is generally presented as an instantaneous property (as peak or at a particular time in a phase of gait cycle, or gait cycle) while work done is the time integral of the joint power and since we aimed to investigate the magnitude and timing of EMG activity of the muscle over a period of time we chose a measure of motor performance that represents the whole region of the gait cycle, like the work done at a joint.

## Definitions of Important Terms

### Compensation

Compensation is a strategy employed to perform an old task in a new manner following an injury.[158] For the purpose of this study, in the context of the non-paretic leg, compensation implies the change in the motor control of the non-paretic leg, presumably either an increase in output during a normally active period or activity in a

normally inactive period, to perform a function usually performed by a healthy contralateral leg that is not being performed by the paretic leg due to weakness and/or discoordination. For example, the increase in the activity of the ankle plantarflexor (Medial Gastrocnemius (MG), Soleus (SO)) during the pre-swing phase of walking may generate greater propulsion to compensate for the impaired ability of the paretic leg.

**Impairment**

Impairment, on the other hand, is the deviation from the generally accepted standards in the biomedical status of the body and its functions.[159] For the purpose of this dissertation, impairment in the non-paretic leg is suggested when there is an excess or deficit in the output of any parameter as compared to healthy control subjects, which would be expected to deteriorate the performance of the non-paretic leg itself. We recognize that this is a difficult question and that it is unlikely that we will be able to answer it with certainty. However, we will use a consistent and rigorous analysis approach in order to determine how strongly the data support the identification of impairment.

**Analysis Plan**

All the variables of EMG magnitude and timing will be calculated for healthy control subjects walking at three pre-selected speeds (0.3, 0.6, 0.9 m/s, to approximately match the self-selected speed of individuals post-stroke). Once the detailed information regarding all the variables of muscle activity from the healthy control subjects is obtained, it will be then used to evaluate the performance of the non-paretic leg.

The post-stroke individuals will be divided into three groups (slow < 0.4m/s, moderate 0.4- 0.8m/s, fast > 0.8m/s) based on their self-selected walking speed on the

treadmill. Classification will be based on the treadmill speed because we aim to compare the biomechanical characteristics of these subjects that were recorded while they were walking on the treadmill. Therefore, classifying them based on their self-selected over ground speed and comparing their performance on the treadmill would result in some ambiguity regarding the results and the conclusions drawn may not be generalizable.

The next step of analysis will be to perform independent samples t-test to compare 1) integrated EMG activity over the gait cycle for all the muscles (8 variables), 2) percentage duration of muscle activity over the gait cycle (8 variables), between the non-paretic and healthy control subjects walking at matched speeds. Thus, timing and magnitude of 8 muscles (16 total variables) in the non-paretic leg will be compared to timing and magnitude of healthy control subjects for each speed group.

Furthermore, we will compare the integrated EMG activity between the non-paretic leg and healthy control subjects (for specific muscle duration, magnitude or both) in the various regions of the gait cycle. If a significant difference is revealed in the region of predominant activity of a muscle in healthy control subjects, that would suggest compensation. This is because the non-paretic leg has to generate greater magnitude of activity or fire for a longer duration in order to maintain a steady walking pattern. However, if the prolonged duration of activation or increased magnitude persists beyond the region of predominant activity (as revealed by control subjects) it might indicate impairment, owing to the inability of the non-paretic leg to adequately modulate the muscle activity during walking post-stroke.

**Hypotheses**

The hypotheses are based on the preliminary analysis of the control data and the evidence from the literature.

TA is reported to have two major bursts of activity during the gait cycle: in region one when it prevents the foot slap and in region five when it contracts to help foot clear the ground. The results of descriptive analysis for healthy control subjects revealed that irrespective of the walking speed, TA had maximum activity in the fifth region of the gait cycle. Therefore, significantly greater EMG activity in the fifth region of the non-paretic leg gait cycle would indicate compensation. Nevertheless, if the maximum activity of TA is in another region of the gait cycle, we will investigate its role in impairment/ compensation.

Furthermore, significantly increased activity of TA in the fourth region of the gait cycle will interfere with the performance of the plantarflexors.[160] To test this we will evaluate the correlation between the positive work done at the ankle in the fourth region of the gait cycle and TA activity in the same region. A significant negative correlation would indicate that an increase in the TA activity in the fourth region of the gait cycle will reduce the positive work done at the ankle and thus, indicating impairment.

During the second and third region of the gait cycle, TA contracts in conjunction with plantar flexors to maintain the limb in extension to facilitate weight support.[125, 161]To evaluate the role of TA in postural balance during second and third region of the gait cycle, we will calculate correlation between TA activity and A1 (work done by eccentric contraction of plantar flexor). A significant negative correlation would suggest that an increase in TA contraction reduces the A1, i.e. work done by ankle plantarflexors which might influence the stability of the leg during these regions.

Medial Gastrocnemius (MG) and Soleus (SO) are the two primary plantarflexor muscles and are responsible for generating maximum positive propulsive work during the pre-swing phase to accelerate the leg and trunk. Healthy subjects revealed maximum activity of the MG and SO during the third region of the gait cycle (late single limb support) at all speeds. Furthermore, literature indicates that the activity of MG and SO is of paramount importance in the fourth region, i.e. pre swing phase, of the gait cycle, for generating the positive ankle plantarflexor power.[19, 32, 162] We will investigate the region of predominant activity in the non-paretic leg. Significantly greater MG and SO activity (time and magnitude) in third or fourth region of non-paretic leg will suggest compensation. The increased SO and MG activity will compensate by generating greater positive work at the ankle to propel the leg and increase its kinetic energy. The increased kinetic energy will help the leg to swing through rapidly to reduce the non-paretic swing phase, and thus single limb support time of the paretic leg.[28] To further confirm the compensation, we hypothesize that the work done at the ankle in the fourth region of the gait cycle will have a significant positive correlation with the MG and SO activity in the same region of the gait cycle. This will suggest that greater activity of the MG and SO is associated with greater work production at the ankle in non-paretic leg, which has been reported to be important contributor to the walking speed of the subject, thus indicating compensation. Nevertheless, if the maximum activity is found in some other region of the gait cycle, we will investigate its role in impairment or compensation.

Results of simulation analysis demonstrate that MG and SO contract during the single limb support, i.e. second and third region of the gait cycle to extend the leg and support the body weight to maintain balance.[161,162] Increased activity on the non-paretic

leg plantarflexors in these regions of the gait cycle would suggest that the muscles are working hard during the extended single limb support phase to support the body weight and maintain balance. A significant positive correlation between MG and SO activity and A1 in the second and third region of the gait cycle would indicate compensation by the ankle plantarflexors to maintain stability to support the weight. However, reduced plantarflexor activity during this region of the gait cycle (as revealed by the preliminary analysis) would suggest inability of the plantarflexors to maintain the leg in extension to support the weight of the body. This weakness of the plantarflexors is compensated for by increased Vastus Medialis (VM) activity.[161,163]

Excessive activity of the non-paretic leg plantar flexors in the first region of the gait cycle, in individuals with short non-paretic step length, would help in compensation by generating propulsive forces for the poorly coordinated paretic leg. Step length asymmetry has been quantified as step length ratio and is defined as the ratio of paretic step length to the total stride length.[164] Therefore we propose some individuals with high step length ratio (i.e. > 0.52; 0.5 being symmetric) will have a step-to-gait. To investigate this we will compare the non-paretic leg angle in individuals that generate A2 against those who do not generate A2. Significantly increased leg angle in individuals generating A2 would suggest compensation by the non-paretic plantar flexors.

In region five and six of the gait cycle MG acts in conjunction with primary knee flexors to assist in knee flexion.[165] Increased MG activity in these regions would suggest compensation to facilitate ground clearance (counteracting the effects of SO and Rectus Femoris (RF) over activity) or to increase the energy of the non-paretic leg to shorten the swing phase.

Furthermore, increase in the SO activity in region five and six of the gait cycle would likely suggest impairment, because there is no biomechanical advantage of the SO activity in the fifth and sixth region of the gait cycle, because they might just interfere with ground clearance.

VM is primarily a knee extensor, which helps to extend the knee and support body weight during stance phase of gait. Descriptive statistics of healthy control subjects revealed maximum activity of VM during the first region of the gait cycle or during the weight acceptance phase. Therefore, if the results of analysis show increase in activity of VM during the first region it would suggest compensation by the non-paretic leg to prevent knee collapse and facilitate weight acceptance.

Simulation analysis demonstrated that VM has an important role in the stance phase to support the body upright.[163] VM works in association with the plantarflexors (mainly SO) to maintain the leg in extension. These studies further stated that the plantarflexors are the first line of action. However, in case of impaired plantarflexor function, VM contracts to assist in maintaining extension of the leg.[162, 163] On similar lines if during the region two and three of the gait cycle the muscle activity is significantly reduced in the plantarflexors (as suggested by the preliminary analysis) and there is a simultaneous increase in the VM muscle activity in the second and third region of the gait cycle, this would suggest compensation by the VM for the weakness of the plantarflexors.

Furthermore, results of a recent simulation analysis study indicated that the increased activity of VM in mid-stance facilitated propulsion of the paretic leg.

Therefore, increased VM activity compensates for the reduced propulsion of the paretic leg.

In addition, the activity of the VM in the sixth region of the gait cycle contributes to deceleration of the rapidly flexing knee, in preparation for landing. Increased activity of VM in this region would suggest greater force requirement to control the rapidly flexing knee, which could be a result of increased kinetic energy and increased knee flexion of the non-paretic leg.[28] However, during the swing phase, the knee needs to be flexed to facilitate ground clearance, therefore, excessive activity of the VM during the fifth region would indicate impairment, and reduced ability of the non-paretic leg to modulate the muscle activity.

RF is another muscle that belongs to the quadriceps group. There is a controversy in the literature regarding the EMG activity of the RF. Annaswamy et al.[166] and others[167] have reported biphasic activity, while Nene et al.[168] and Perry et al.[19] have reported a single burst of activity during the gait cycle. The preliminary analyses of our control data reveal two prominent phases of activity of the RF muscle: one during the first region of the gait cycle and the second during the region four (pre-swing phase) of the gait cycle where the RF acts as a hip flexor in propelling the limb forward into swing. Descriptive statistics of control subjects' data revealed that the loading response phase activity is greater than the pre-swing phase, as reported in literature.[19,168] However, simulation studies do not support this function of the RF, and suggest that the RF generates an extension moment at the hip and the knee during the pre-swing phase. Furthermore, Hernández et al.[169], in a study of electrical stimulation of the RF during walking reveal that the RF activity before toe-off, i.e. region four of the gait cycle diminishes hip and

knee flexion. In addition, several studies have reported that increased activity of the RF during the early swing phase, i.e. region five of the gait cycle, is associated with the stiff knee gait.[170, 171] The stiff knee might thus interferes with ground clearance and results in altered walking pattern. Stiff knee gait due to the over activity of the RF muscle in the pre-swing and the swing phase is characterized by reduced peak knee flexion.[171, 172] Furthermore, the results of simulation analysis showed that elimination of RF activity during the pre-swing and swing phase increased the peak knee flexion angle during swing.[172] To confirm the influence of the RF activity on the peak knee flexion angle achieved during the swing phase of the gait cycle we will correlate the peak knee flexion angle during the fifth region of the gait cycle with the RF activity during the same region. A significantly negative correlation would indicate that the increased RF activity is interfering with the knee flexion. Therefore, we hypothesize that the increased burst of RF activity of the non-paretic would indicate impairment.

In first through third and sixth regions, RF is involved in decelerating the leg and shock absorption and weight support during the single limb support, when the whole limb is maintained in extension.

Lateral Hamstrings (LH) and Medial Hamstrings (MH) are some of the major contributors to energy generation over the gait cycle.[173] They are active in late stance and beginning of the stance restoring energy to the body near double limb support, a most effective time to utilize the passive locomotion properties of the body.[173] Maximum activity of hamstrings muscles was present in region one (0.3m/s) and region six (0.6m/s and 0.9 m/s) of the gait cycle in the healthy subjects. If the maximum activity is present in the first/ sixth region of the gait cycle of the non-paretic leg, then we will

compare the integrated EMG activity in these regions. Significantly greater non-paretic leg activity would suggest compensation by the non-paretic leg. Nevertheless, if the maximum activity is found in some other region of the gait cycle, we will investigate its role in impairment or compensation.

We hypothesize that increased activity of the hamstrings (LH and MH) in the first and sixth region of the gait cycle (depending on the walking speed) would indicate compensation by the non-paretic leg to produce greater work to support the non-paretic leg to facilitate swinging of the paretic leg and maintain steady walking state. Furthermore, a significant correlation between the hamstrings activity and the H1 positive work in region would further strengthen it.

Buurke et al.[126] reported an extra burst of MH in the stance phase of the non-paretic leg and they suggested it assists in extension of the non-paretic hip, thereby facilitating the swing of the paretic leg and thus compensating for the diminished swing caused by the lack of push-off and reduced force of hip flexors on the paretic side. If an extra burst of the MH is revealed in our data of the non-paretic leg, we would evaluate the suggestion made by Buurke et al.[126], by evaluating the correlation between the MH activity in region three and four of the non-paretic leg and the positive hip extension work.

Gluteus Medius (GM) contraction during the single limb support phase provides stability to the pelvis in the frontal plane. GM provides body support and forward propulsion in early stance.[161, 174] Excessive activity of the GM during the stance phase of the non-paretic leg would indicate compensation for increased loading of the same.

**Approach to Interpretations**

First we will investigate differences in muscle activity over the gait cycle to broadly identify differences between the motor control of the non-paretic leg and healthy control subjects. The next step will be to identify all the regions of the gait cycle that present altered muscle activity in the non-paretic leg. We will inspect the kinematics (e.g. leg angle) for any difference in the leg configuration that might be related to the difference in the muscle activity. Following this we will evaluate related measures of motor performance, i.e. the work done at the joint, to estimate the biomechanical effect of the same. The information gained from the steps discussed above can thus be used to discuss altered muscle activity in the non-paretic leg, relative to the biomechanical characteristics and our original hypotheses related to muscle activity.

The third study of the dissertation, will investigate the response of the non-paretic leg to LT. In this study, we will evaluate how the impairments and compensation mechanisms change after task specific LT. To accomplish this we will first group subjects into different speed based categories (as discussed above). We will compare the performance of their non-paretic leg to the healthy control subjects walking at matched speeds before LT, similar to chapter 4. Next we will group the subjects based on their self-selected walking speed on the treadmill after the LT. The performance of the non-paretic leg will be compared to healthy control subjects walking at the matched speeds. It would be interesting to evaluate the changes in the non-paretic leg, i.e. does it continue to be impaired and compensating, or is there some optimization in the regions of the gait cycle which revealed altered behavior before the training. There might be reduction in impairment, or increased compensation or optimization of the muscle output.

**Limitations of the analysis**

Several other variables were considered before selecting the variables for the analysis of these studies; however, there are some limitations.

EMG variables form the core of the analyses for chapter 4 and 5. The information obtained from the EMG recordings may not reflect the true state of the muscles as they may be confounded by external (orientation and location of the electrode, machinery, power lines) and internal factors (muscle fiber type, depth and the amount of tissue between the muscle and electrode)[151], which cannot be ruled out. Therefore, we performed correlation analysis with the measures of motor performance, i.e. work done to confirm the results of the EMG analysis.

Work done is an important quantity in gait analysis and has been often used to analyze energy generation, absorption and/ or transfer within the body segments[175, 176], examine locomotor efficiencies[155, 156], determine how muscles function[157] and identify impairments associated with neurological deficits[59, 60]. A correlation analyses like this provides information regarding the role of the non-paretic leg muscle in the function of walking.

Furthermore, in a bilateral task like walking, it may be difficult to differentiate between the role of the non-paretic leg in compensation and impairment post-stroke. However, in this dissertation, we tried to explicitly define compensation and impairment for our studies and then set strict criteria to demark the non-paretic leg's role in either of the two. Furthermore, we also performed a detailed analysis of the muscle activity and motor performance measures to ensure correct inference of the non-paretic performance.

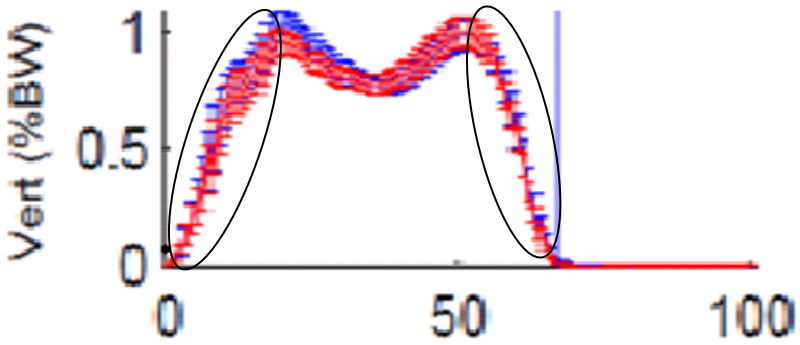


Figure 2-1. Represents the vertical GRF in a healthy control subject. The blue and red plots represent right and left leg, and the part of the curve in black circles represent the loading and unloading phases.

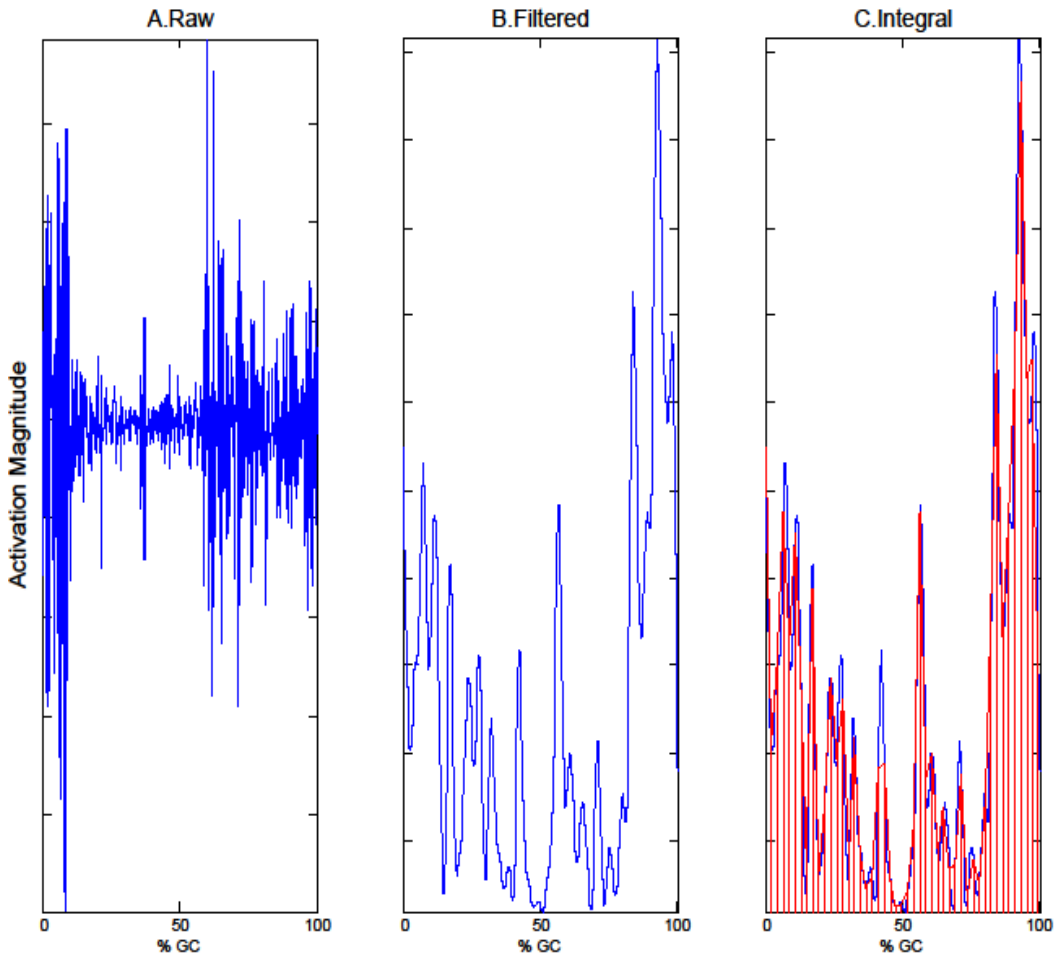


Figure 2-2 . Represents the calculation of integrated EMG activity. First figure represents the raw EMG signal, second panel represents the rectified and filtered EMG signal and the third figure represents integration rule with equally spaced abscissas (red lines), with respect to time in the gait cycle.

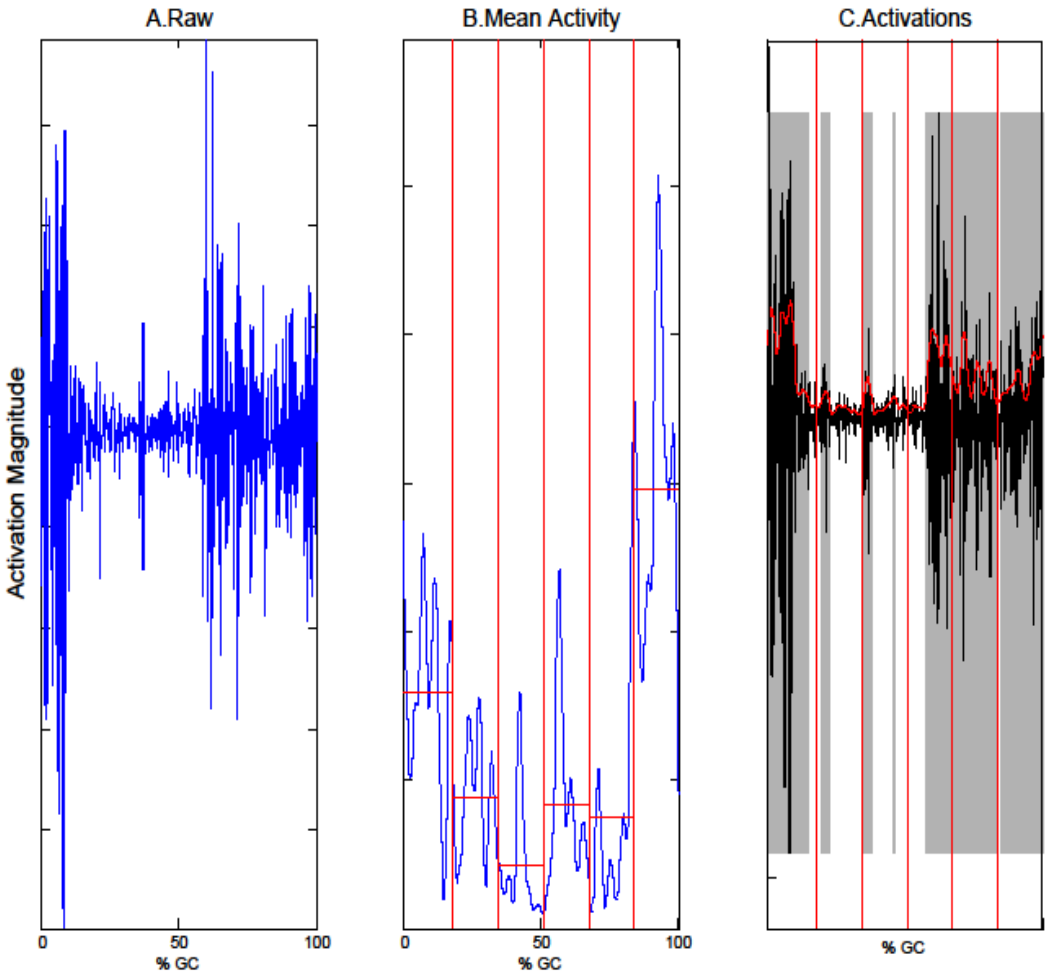


Figure 2-3. Represents the steps of k-means analysis. First figure represents the raw EMG magnitude, second figure shows the filtered rectified EMG (linear envelope) and the third figure represents the original raw EMG signal with the detected periods of activity indicated in gray shaded areas.

# CHAPTER 3
# MAGNITUDE AND RATE OF LIMB LOADING AND UNLOADING DURING HEMIPARETIC GAIT IS ASYMMETRIC AND RELATED TO LEG KINEMATICS.

## Background

Hemiparesis following stroke results in poorly coordinated and asymmetric gait. In addition to well described kinematic asymmetries, there are also less well described kinetic asymmetries. Kinetic parameters (e.g. joint moments, powers and GRF) are particularly important due to their dependence on both kinematics and muscle activity and can provide insight into the underlying causes of changes in walking pattern. In particular, vertical GRF has been used to characterize post-stroke weight-bearing.[7, 46] The paretic leg typically exhibits lesser weight-bearing than the non-paretic leg (i.e., neither LL nor LU are symmetric).[36-41] LL and LU asymmetry are important contributing factors to postural instability, reduced dynamic balance, increased postural sway and thus falls.[36] Reduced paretic (as compared to non-paretic) LL has been reported in static and quasi-static tasks (e.g., standing in different stance positions)[7, 9, 10], stepping on stairs of different heights, and sit-to-stand from a chair[9]) and has been related to walking ability post-stroke.[35, 47, 48] However, studies typically report reduced paretic LL without quantifying the temporal characteristic of LL and LU, which may provide important information to understand leg loading. While it is often assumed clinically that reduced loading of the paretic leg is indicative of poor walking and recovery, LL and LU asymmetry during walking in individuals with hemiparesis has not been quantified and the relationship with walking performance or stroke severity is unknown.

Measuring LL and LU ability during static standing (standing on force plates and transferring weight from one leg to the other while the whole body center of mass stays between the two feet) does not provide much detail about walking ability, because

walking involves acceleration of all individual body segments[7] such that the whole body's center of mass is mostly positioned outside the base of support. In addition to forward and vertical acceleration of the whole body center of mass, walking also involves medial-lateral accelerations of the center of mass. Though the magnitude of movements and forces in medial-lateral direction are small, they are expected to be important to facilitate loading and unloading. This would be consistent with the observation that manual assistance provided at the pelvis by trainers during locomotor training to produce medial-lateral movement of body, presumably to facilitate appropriate weight shift, appears to improve walking pattern and increase stability.[108]

Multiple paretic and non-paretic LL and LU patterns can be employed by individuals with hemiparesis to maintain a steady walking state. It is important to quantify these patterns to understand the asymmetry (as well as the differences in asymmetry between subjects) in steady state walking. The presence of multiple loading patterns would be consistent with the observation that subjects with post-stroke hemiparesis exhibit multiple kinematic patterns to successfully maintain a steady walking state. The kinematic characteristics may provide more insight into the various patterns of LL and LU exhibited by different individuals. Therefore, we hypothesize that classifying post-stroke individuals based on their LL and LU and evaluating their relationship to kinematic and kinetic measures will provide deeper understanding of weight support and dynamic stability during walking.

The main purpose of this study was to determine the relationship between LL and LU asymmetry in individuals with hemiparesis and 1) functional walking status and 2) specific kinematic and kinetic variables during walking. We hypothesize that LL and LU

in the paretic and non-paretic leg will differ with functional walking status and that the asymmetry will be related to knee angle and medial-lateral leg angle (angle between vertical and the frontal plane projection of the line connecting the center of masses of the pelvis and foot). Since the magnitude of medial-lateral forces are directly related to the medial-lateral accelerations of the whole body center of mass, we hypothesize it will be intimately related to weight shift, thus we will also establish the relationship between medial-lateral forces and LL and LU. Detailed understanding of LL and LU asymmetry will provide deeper insight about the changes in locomotion in post-stroke individuals and potentially inspire new strategies to rehabilitate walking.

## Methods

### Participants

Data were collected from forty five individuals with chronic hemiparesis (age = 60.2 ± 12.3 years, 19 females, 19 left-side hemiparesis) and eighteen similarly-aged healthy individuals (age = 66.2 ± 10.0 years, 4 men) at the VA-UF Human Motor Performance Laboratory, in Gainesville, Florida. To be included in the study, individuals had to have: 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 regularly at home. Exclusion criteria for the participants from the study were: any orthopedic or neurologic conditions in addition to stroke that might limit hip and knee extension or ankle plantar flexion to neutral, or inability to provide informed consent. All participants in the study signed a written informed consent and the study was approved by Institutional Review Board at University of Florida. Three categories of subjects with different severity were identified based on functional walking status as defined by their self-selected overground walking speeds[15], individuals who walked <0.4

m/s (household ambulators), or 0.4-0.8 m/s (limited-community ambulators), or > 0.8 m/s (community ambulators).

**Experimental Protocol**

Retro-reflective markers were placed at specified bony prominences on the body. Clusters of reflective markers attached to rigid bodies, were also placed firmly on the pelvis, each thigh, shank and foot of the participants. All participants walked on an instrumented split-belt treadmill (Techmachine, Andrezieux Boutheon, France) without using any assistive device or ankle foot orthosis for three walking trials at their self-selected treadmill walking speed. Data were collected for 30s after the subject walked for ~ 10s to achieve steady state. 3-D GRF and kinematic parameters were obtained for each leg as the subjects walked on the treadmill.

A harness system mounted to the ceiling of the laboratory was used for safety in case participants lost their balance. The harness was worn across the shoulders and chest of the participant and no body weight was off-loaded by the system. A physical therapist stood near the subject, but did not provide assistance as they walked over the treadmill. Healthy control subjects walked at their self-selected speeds as well as at speeds of 0.3m/s, 0.6m/s, 0.9m/s to provide control data at speeds comparable to that of the hemiparetic individuals.

A twelve camera motion analysis system (Vicon Motion Systems, Los Angeles, CA) was used to collect the data as the subjects walked on the instrumented treadmill. Customized MATLAB (MathWorks Inc, Natick, MA) programs were used for data analysis. Kinematic data were low-pass filtered using a fourth-order zero-lag Butterworth filter with a 10 Hz cut-off frequency. GRFs were sampled at 2000 Hz and low-pass filtered using a fourth-order zero-lag Butterworth filter with a 20 Hz cut-off

frequency. GRF data were normalized to subject's body weight and the gait cycle was expressed as percent of paretic leg gait cycle i.e. paretic foot contact to paretic foot contact. LL and LU occur during the double limb support phases of the gait cycle; therefore, all the variables in this study were calculated in these two phases.

**Study Variables**

**Magnitude of loading/unloading**

Average LL and LU were quantified by the average vertical GRF in the first and second double support phases, respectively (figure 3-1a, 3-2a). The legs in first and second double support phase were defined as loading and unloading, respectively (i.e., when one leg is being loaded the other is being unloaded in the same double limb support phase).

**Timing of loading/unloading**

The timing of LL was calculated for each leg as the cross-over point of the two vertical GRF curves during the double limb support (i.e. the percent gait cycle from the beginning of the double support until the majority of weight shifts from the leg in second double support to the leading leg in first double support (figure 3-4a)). Thus, the timing of LL of one leg is equal to that of LU in the other leg and expressed as a percentage of the double support phase (i.e., 50% = symmetric).

**Pattern of limb loading/unloading**

The pattern of LL/LU was calculated from the shape of the vertical GRF. We identified three patterns (figure 3-1d): concave curvature (i.e., initially slow then rapid loading), convex curvature (i.e., initially rapid then slow loading) and linear. The best fit curve for a polynomial was calculated (JMP statistical software, SAS, Cary NC) for the vertical GRF values for the loading phase and unloading phase (end of the single limb

support to toe off) for each subject. Significance was set to $p<0.001$ as a correction for the large number of calculated curves. A significant first order term meant a linear slope, whereas a second order coefficient term meant the LL/LU profile had a curvature (positive coefficient was concave and negative coefficient was convex).

**Kinematics**

The average leg angle in the medial-lateral direction was calculated during each double limb support phase as the angle between the vertical and the projection of the line connecting the center of mass of pelvis and the center of mass of foot in the frontal plane (figure 3-1c, figure 3-2c). The average knee flexion-extension angle was calculated during each of the two double limb support phases of the gait cycle (figure 3-1b, figure 3-2b).

**Statistical Analysis**

One-way ANOVAs were performed to investigate the differences in the LL and LU between the paretic leg, non-paretic leg and the similarly aged healthy individuals walking at matched speeds and their self-selected speeds. We then correlated LL (LU) with the kinematic measures during each double limb support phase of the gait cycle. Pearson's correlation was used to examine the relation between LL and LU and various parameters selected (i.e. knee angle, leg angle, medial-lateral GRF). The value of significance for all analysis was set at $\alpha = 0.05$ and alpha values were reported. SPSS v. 17 (SPSS, Inc.) was used for statistical analysis.

## Results

The control subjects were symmetric (e.g., no statistically significant differences in the LL (LU) and kinematic variables between the two legs). Therefore, the average value of all parameters for both legs was used at all speeds.

**Magnitude of Limb Loading**

Average magnitude (normalized by body weight) of LL was less in the paretic leg as compared to the non-paretic leg (0.509±0.102 N/kg vs. 0.651±0.146 N/kg, p<0.0001) and to similarly aged healthy control subjects walking at the self-selected speeds (0.509±0.102 N/kg vs. 0.596±0.0296 N/kg, p= 0.021).There was no significant difference between LL of the paretic leg and control subjects walking at matched speeds (control (0.6m/s/ 0.3m/s) = 0.554 ±0.117 N/kg, 0.585 ± 0.075 N/kg, p= 0.807 and 0.061).

The reduced paretic LL was related to the functional walking status of individuals with hemiparesis. LL of the paretic leg in the household (0.459±0.09N/kg) and limited-community (0.513±0.069N/kg) ambulatory individuals was less than in the non-paretic leg [(0.680±0.11N/kg; p=0.001 vs. household ambulators paretic), (0.646±0.08N/kg; p=0.001vs. limited-community ambulators paretic) and in healthy individuals walking at matched [(0.585±0.076N/kg; p=0.001 vs. household ambulators paretic), (0. 554±0.117N/kg, p=0.001 vs. limited-community ambulators)] and self-selected speeds [(0.596±0.373N/kg; p= 0.004 against household ambulators paretic, p= 0.001 against limited-community ambulators)]; whereas, the community ambulators did not show any significant difference as compared to the non-paretic leg (0.526±0.099N/kg vs. 0.565±0.105N/kg) and to the controls (0.596±0.373N/kg, p=0.98). (figure 3-1a (individual representative subject, figure 3-2a group average))

**Magnitude of Limb Unloading**

The LU was greater (for LU smaller value implies greater unloading) in the paretic leg as compared to the non-paretic leg (0.377±0.085N/kg vs.0.508±0.084N/kg, p=0.0001) and similarly-aged healthy control subjects walking at the self-selected

speeds (0.511±0.063N/kg, p= 0.001) and matched speeds (control (0.6m/s/ 0.3m/s)=0.513±0.034N/kg/ 0.504±0.040N/kg, p=0.001/0.001), as evident from the lower average magnitude of vertical GRF.

The increased paretic LU was related to the severity of the gait speed deficit. During the second double-limb support phase, the LU of the paretic leg in the household (0.310±0.06N/kg) and limited-community (0.390±0.056N/kg) ambulatory individuals was different (note that reduced GRF equals greater LU) from the non-paretic leg [(0.524±0.09N/kg; p=0.001 against household ambulators paretic), (0.646±0.08N/kg; p=0.001against limited-community ambulators) and healthy individuals walking at matched [(0.504±0.076N/kg; p=0.001 against household ambulators paretic),( 0.513±0.04N/kg, p=0.001 against limited-community ambulators) and self-selected speeds [(0.504±0.063N/kg; p=0.001 against household ambulators paretic, p=0.001 against limited-community ambulators)]; whereas, the community ambulators did not show a significant difference as compared to the non-paretic leg (0.438±0.08N/kg vs. 0.462±0.06N/kg) and the controls (0.504±0.063N/kg, P= 0.06). (figure 3-1a, figure 3-2a unloading part of the graph)

**Timing and Pattern of Limb Loading and Unloading**

In addition to the difference in the magnitude of LL and LU, the timing of LL and LU of the paretic and non-paretic legs and healthy controls was significantly different. One-way ANOVA revealed that the difference in timing of LL of the paretic leg (LU of the non-paretic leg) and LL of the non-paretic leg (LU of paretic leg) and healthy controls was significant ($p<0.001$). The transition of majority of weight from the paretic to the non-paretic leg (timing of paretic LU) occurred at 32±8.2% of the duration of the double limb support phase, which was earlier than that from the non-paretic leg to the

paretic leg (timing of non-paretic LU) (51.6±9.9%) or control legs (44.5±4.6%) (figure 3-4b). The above conclusions are drawn from the information gained by the duration of the second double limb support phase of the paretic (28.0±4.2% of the gait cycle or 0.427±0.07s), non-paretic (25.8±2.9% of the gait cycle or 0.38±0.09s).

This difference in timing of LL and LU was further investigated in each of the functional groups. One way ANOVA analysis revealed that the average LL time of the non-paretic leg (transition of the weight from the paretic to non-paretic leg) in the household ambulators (22.5±8.9%) was significantly less ($p=0.047$ vs. limited-community ambulators, and $p<0.001$ vs. community ambulators) than the limited-community ambulators (30.4±4.8%) and the community ambulators (39.8±7.7%). Additionally, the LU time of the paretic leg of the household ambulators (57.6±9.1%) was greater than limited-community ambulators (50.1±8.6%), and the community ambulators (47.1±6.9%) ($p=0.013$ vs. limited-community and $p=0.006$ vs. community ambulators).

In addition to the difference in timing, the LL and LU between the paretic leg could be classified into three groups based on their curvatures: for LL; concave curvature (12 subjects, 10 Household ambulators, 1 Limited-Community ambulator, 1 Community ambulator); convex curvature (20 subjects; 4 Household ambulators, 11 Limited Community ambulators, 5 Community ambulators); and linear (4 subjects; 3 Limited-Community ambulators,1 Community ambulator), 8 subjects (6 Limited-Community ambulators, 2 Community ambulators) could not be categorized because of very abnormal shape of the GRF curve (Table 2-1).

For LU: concave curvature (31 subjects; 11 Household ambulators, 16 Limited-Community Ambulators, 4 Community Ambulators), convex curvature (6 subjects; 3 Household Ambulators, 2 Limited Community Ambulators, 1 Community Ambulator), and Linear (2 subjects; community Ambulators) (Table 2-1). Six subjects could not be categorized because of abnormal shape of LU for their GRF curve (these subjects were different from the subjects mentioned in the pattern of LL analysis above). Household ambulators primarily had concave loading (slow initially then more rapidly – 8 out 14 and concave unloading (rapid initially then more slowly – 11 out of 14 pattern whereas most of the limited community ambulators had a convex loading (11 out of 21 and a concave unloading pattern (16 out of 21). In addition, the community ambulators did not show any consistent patterns.

**Relationship between Medial-Lateral Ground Reaction Forces and Limb Loading and Unloading**

Medial-lateral forces were not significantly correlated with vertical GRF during both double limb support phases of paretic leg (for first p=0.91 and second p= 0.342 double limb support)

**Relationship between Knee Angle and Limb Loading and Unloading**

There was no statistically significant correlation between average LL and LU and knee angle on the paretic leg, however, there was a trend towards significance for LU, suggesting that the individuals with greater unloading (lower average vertical GRF values) may have a greater knee flexion, (r=-0.150, p=0.162- for LL and knee angle; and r=-0.213, p=0.052 for LU and knee angle). The non-paretic leg LL was significantly related to knee angle (r=-0.459, p=0.001) showing that the individuals with greater leg

loading have lesser knee flexion, but the LU showed no significant correlation (r=0.196, P=0.261).

**Relationship between Medial-Lateral Leg Angle and Limb Loading and Unloading**

Subjects with reduced paretic LL placed the paretic leg further lateral relative to the center of mass of the pelvis. LL was negatively associated with the leg angle of the paretic side in the medial-lateral direction during the second double support phase of the gait cycle (r=-0.451, p=0.002). The paretic leg medial-lateral angle (9.4±2.6) was always significantly ($p<0.05$) greater than that of the non-paretic (7.3±2.0) and of healthy controls walking at matched speeds (figure 3-1c, figure 3-2c), demonstrating that the paretic leg was placed more lateral from the center of mass of the body as compared to the non-paretic leg and controls.

## Discussion

Our results demonstrate that the loading of the paretic leg is significantly less than either the non-paretic leg or the control subjects walking at self-selected or matched speeds. These results are consistent with the loading abilities of the individuals with hemiparesis during static and quasi-static tasks previously reported.[7, 8, 10] Furthermore, reduced LL of the paretic leg and the increased compensatory LL of the non-paretic leg varies with the severity of stroke (as defined by the self-selected walking speed). Our study found that the household walkers had significantly greater loading of their non-paretic leg (i.e., greater paretic unloading) during initial double limb support than the limited-community and community walkers. This compensation by non-paretic leg can be explained by the phenomenon of “impairment and compensation”[31],which suggests that greater the impairment of the paretic leg, the greater is the compensation offered by the non-paretic leg to maintain stability of the ongoing task. The asymmetry between the

paretic and the non-paretic leg in both LL and LU could be attributed to the weakness and decreased control of the paretic leg.[7, 177] Physiologically, reduced loading of the paretic leg might be associated with reduced muscle activation during walking, which might result in increased impairments during walking, for example, poor support or propulsion; the LU is increased on the paretic side, meaning that the individuals with hemiparesis unload their paretic leg rapidly.

In addition to the asymmetry in the magnitude of LL and LU the timing of LL and LU is also important because walking is a cyclic task produced as a result of synchronized phasic stimuli (hip extension and LL and LU). The individuals with hemiparesis, walking at their self-selected speed accept body weight much more slowly during the initial double limb support of the paretic leg (than the non-paretic leg), whereas during terminal double limb support the weight is rapidly transferred to the non-paretic leg. This demonstrates a reluctance to load their paretic leg, resulting in reduced single leg stance phase on the paretic leg and longer double limb support phase.

Although, the timing of LL and LU provides details about the LL and LU asymmetry, it does not capture the variability of biomechanical adjustments made by the individuals with hemiparesis as do the convex, concave or linear shape of the LL and LU curves. The individuals with concave LL pattern were primarily household ambulators, i.e. walking at less than 0.4 m/s and a majority of the individuals with convex LL pattern were limited-community ambulators and the linear pattern comprised both limited-community and community ambulating subjects. For 10 subjects who displayed both concave LL and LU, the loading of both paretic and non-paretic leg was slow, suggesting that they spent a large proportion of the gait cycle in the double limb

support phase and displayed slow loading of both the paretic and the non-paretic leg. Similar information about difference in pattern of LL and LU of the paretic and the non-paretic leg provides us more details about the changes and variability in walking and LL and LU abilities post-stroke. This information might assist in designing better rehabilitation techniques, for example, greater emphasis could be placed on the weight shifting abilities of the household ambulators to promote faster and more physiologic walking speed.

As mentioned before, individuals with hemiparesis make several biomechanical adjustments to minimize the loading of the paretic leg (i.e. by reducing LL and increasing LU and time to load, joint angle). Knee angle is an important component for supporting the body while accepting weight in order to prevent collapse and has been the most often reported kinematic variable in relation to walking ability post-stroke.[29, 30] Although knee angle was not significantly related to the LL and LU on the paretic leg, the trend towards significance suggests a possible importance of knee angle in understanding LL and LU. We found that the knee was more flexed in the non-paretic leg as compared to the paretic leg and healthy controls during both LL and LU. In addition, difference in terminal double limb support could be explained by reduced ability to plantarflex the ankle to create push-off power and the increased knee flexion could be credited to increased power generation requirements of the non-paretic leg (at hip, knee and ankle) to compensate for the decreased power output from the paretic leg.[28]

Additionally, LL and LU in subjects with hemiparesis is influenced by medial-lateral leg angle. The results of our study support the observation that the hemiparetic

individuals place their non-paretic leg close to their body while placing their paretic leg farther away[143], meaning that the medial-lateral leg angle is greater on the paretic leg and significantly less on the non-paretic leg. The lateral placement of the paretic leg may be a strategy to increase the base of support while walking or to minimize loading of the paretic leg. Minimizing loading of paretic leg may be attributed to weakness or impairment, spatial neglect, lack of confidence or fear of falling.

**Non-neurological concerns of increased loading of the non-paretic leg.** There is evidence in individuals post-amputation that suggests osteo-arthritis of the joints in the intact leg (e.g. knee[55] and hip[56]), because of the changes in the direction of the ground reaction forces and greater leg loading.[57] In addition, subjects with poliomyelitis typically have asymmetrical disease, with greater forces transmitted across the unaffected leg resulting in greater symptoms on the unaffected leg as compared to the affected leg.[58] Similarly, greater loading of the non-paretic leg might result in musculo-skeletal changes which might further interfere with the walking ability of the persons with hemiparesis. Therefore, assessment and rehabilitation should not neglect the non-paretic leg.

Table 2-1. List of loading and unloading details of hemiparetic individuals

| ID | Speed (m/s) | Pattern of LL | Pattern of LU | Average LL | Average LU |
|---|---|---|---|---|---|
| 1 | 0.35 | concave | concave | 0.534 | 0.296 |
| 2 | 0.34 | convex | concave | 0.415 | 0.294 |
| 3 | 0.21 | concave | concave | 0.463 | 0.321 |
| 4 | 0.21 | concave | concave | 0.463 | 0.340 |
| 5 | 0.39 | convex | concave | 0.418 | 0.280 |
| 6 | 0.19 | convex | concave | 0.400 | 0.265 |
| 7 | 0.18 | concave | concave | 0.536 | 0.317 |
| 8 | 0.26 | concave | convex | 0.481 | 0.444 |
| 9 | 0.35 | concave | concave | 0.380 | 0.277 |
| 10 | 0.36 | concave | convex | 0.557 | 0.354 |
| 11 | 0.15 | concave | concave | 0.414 | 0.248 |
| 12 | 0.35 | convex | concave | 0.270 | 0.289 |
| 13 | 0.22 | concave | convex | 0.631 | 0.240 |
| 14 | 0.24 | concave | concave | 0.862 | 0.388 |
| 15 | 0.4 | ** | concave | 0.696 | 0.432 |
| 16 | 0.7 | convex | concave | 0.457 | 0.508 |
| 17 | 0.42 | convex | concave | 0.479 | 0.436 |
| 18 | 0.45 | convex | concave | 0.466 | 0.374 |
| 19 | 0.42 | concave | convex | 0.537 | 0.382 |
| 20 | 0.44 | ** | concave | 0.576 | 0.378 |
| 21 | 0.46 | convex | ** | 0.422 | 0.382 |
| 22 | 0.63 | convex | concave | 0.601 | 0.262 |
| 23 | 0.4 | ** | concave | 0.471 | 0.379 |
| 24 | 0.73 | convex | concave | 0.523 | 0.465 |
| 25 | 0.75 | convex | concave | 0.541 | 0.433 |
| 26 | 0.51 | linear | concave | 0.419 | 0.308 |
| 27 | 0.57 | convex | ** | 0.458 | 0.319 |
| 28 | 0.63 | convex | concave | 0.552 | 0.429 |
| 29 | 0.48 | ** | concave | 0.520 | 0.356 |
| 30 | 0.65 | convex | convex | 0.491 | 0.478 |
| 31 | 0.8 | ** | concave | 0.573 | 0.549 |
| 32 | 0.49 | linear | ** | 0.514 | 0.350 |
| 33 | 0.42 | ** | concave | 0.424 | 0.370 |
| 34 | 0.62 | convex | concave | 0.471 | 0.359 |
| 35 | 0.43 | linear | concave | 0.584 | 0.295 |
| 36 | 0.99 | convex | ** | 0.050 | 0.510 |
| 37 | 0.99 | ** | concave | 0.506 | 0.456 |
| 38 | 1.05 | convex | concave | 0.353 | 0.516 |
| 39 | 0.84 | convex | concave | 0.545 | 0.332 |
| 40 | 0.8 | ** | convex | 0.602 | 0.518 |
| 41 | 0.9 | concave | linear | 0.595 | 0.430 |
| 42 | 1 | convex | convex | 0.522 | 0.551 |
| 43 | 0.86 | linear | linear | 0.582 | 0.363 |
| 44 | 0.87 | convex | concave | 0.619 | 0.438 |

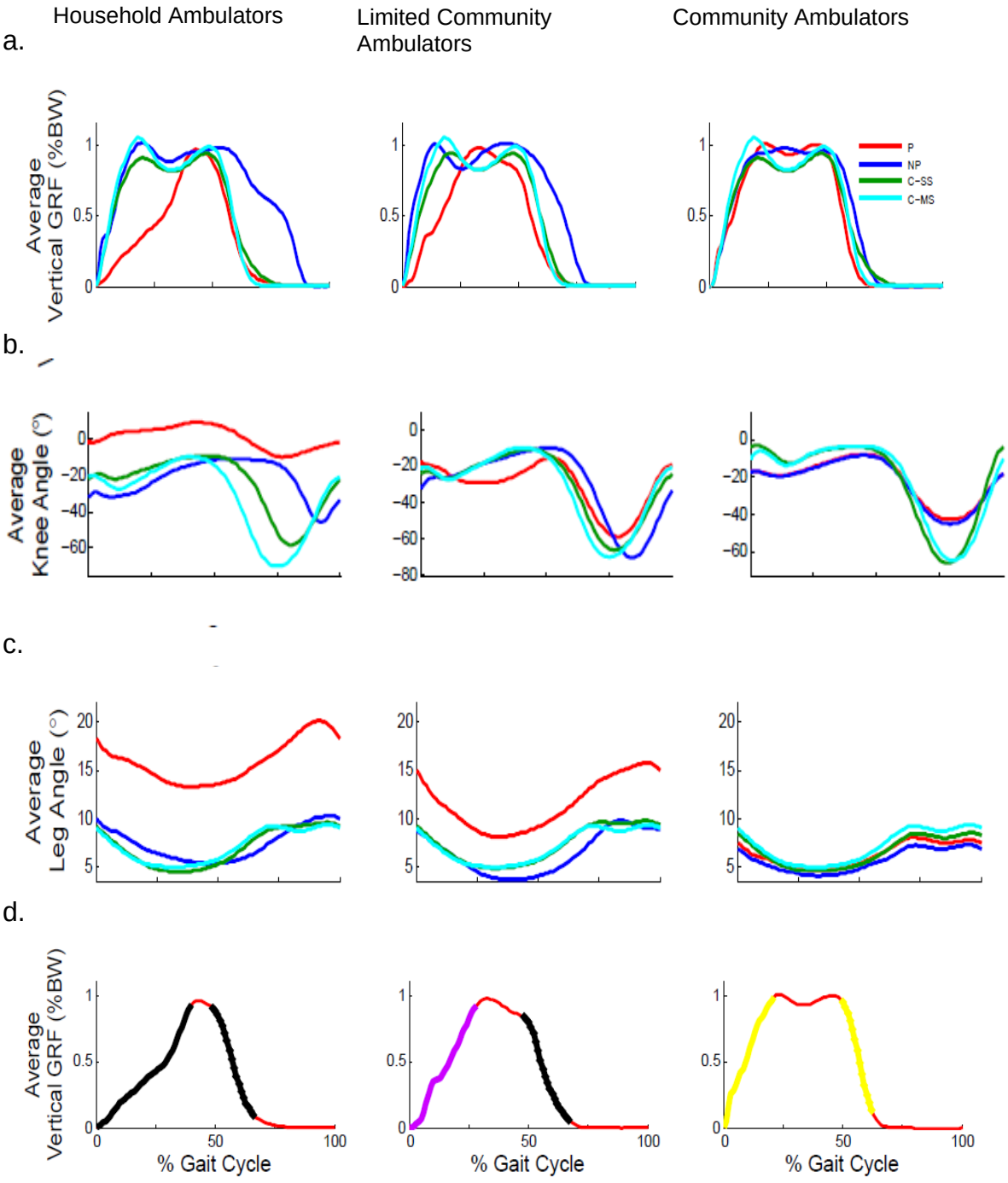


Figure 3-1. Represents characteristics of one subject in each speed group. a) the limb loading and unloading b) knee angle c) leg angle d) loading and unloading pattern in one representative subject of each speed-based functional group (column 1 = household ambulator; column 2 = limited community ambulatory; and column 3 = community ambulator. Red is paretic leg, blue is non-paretic leg, aqua blue is control at matched speeds, and green is control at self-selected speeds. In 1 d, the black highlight represents the concave pattern, purple shows the convex pattern and the yellow is linear pattern.

a.

b.

c.

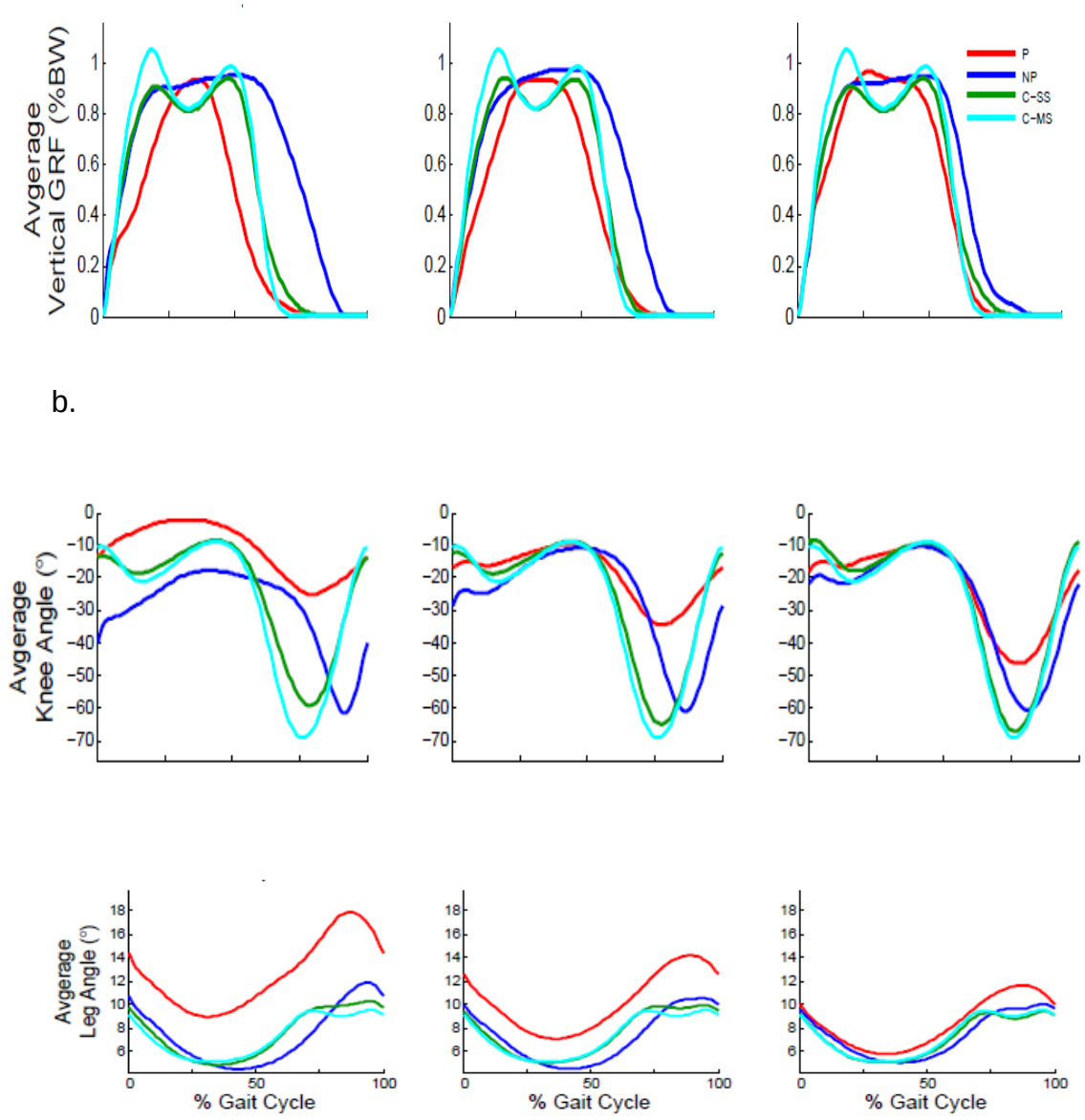


Figure 3-2. Represents average characteristics of all subjects in each speed group. a) Limb loading and unloading b) knee angle c) medial-lateral leg angle of all individuals in each functional group (based on speed). First column represents household ambulators; second column limited community ambulators while the third column community ambulators. Red represents paretic leg, blue: non-paretic leg, green is control at self-selected speed, aqua blue is control at matched speeds.

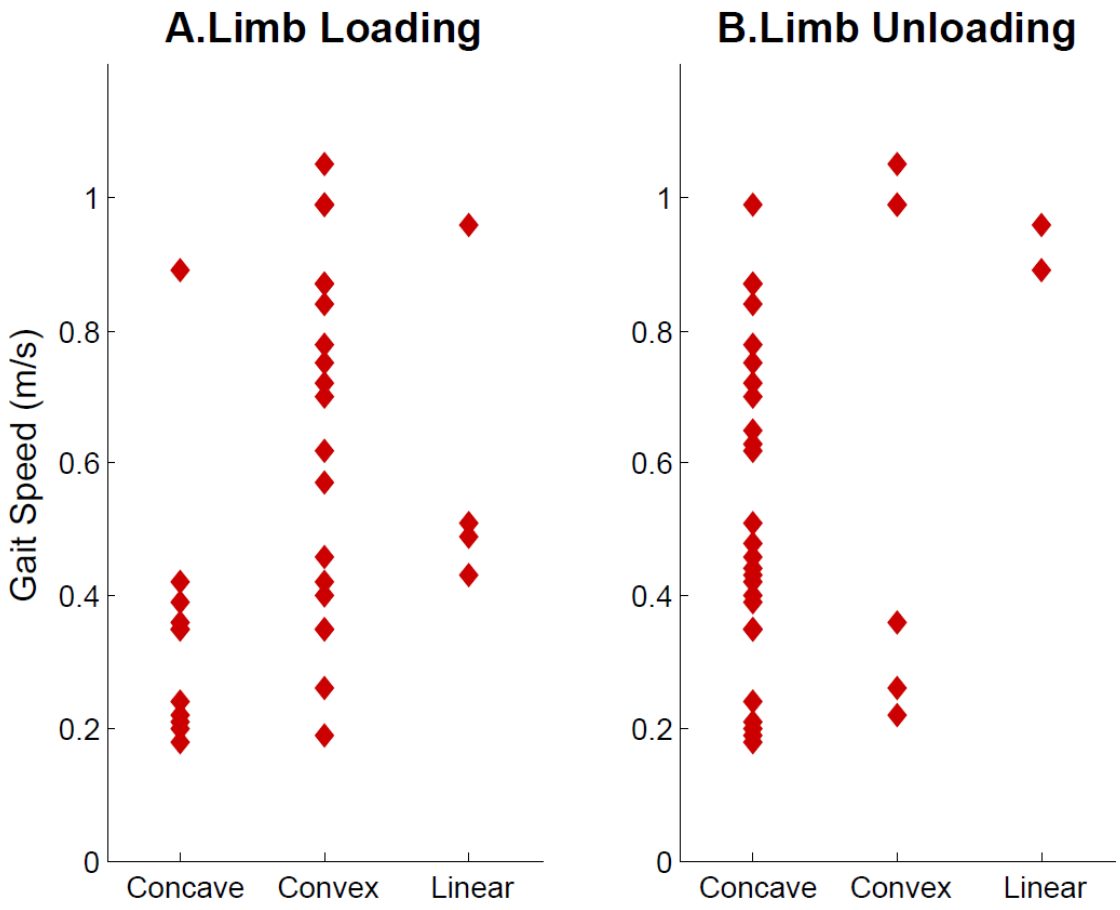


Figure 3-3. Represents individuals with different loading and unloading patterns against their self-selected walking speed. Self-selected walking speed is used in this study to categorize individuals into three groups (<0.4m/s= household Ambulators, 0.4- 0.8 m/s= Limited Community Ambulators, >0.8 m/s= Community Ambulators). Each point on the scatter plot represents a hemiparetic individual. X-axis represents the different loading and unloading patterns. NOTE: the number of points looks less than the total number of subjects because subjects with similar speeds are all represented with a single point.

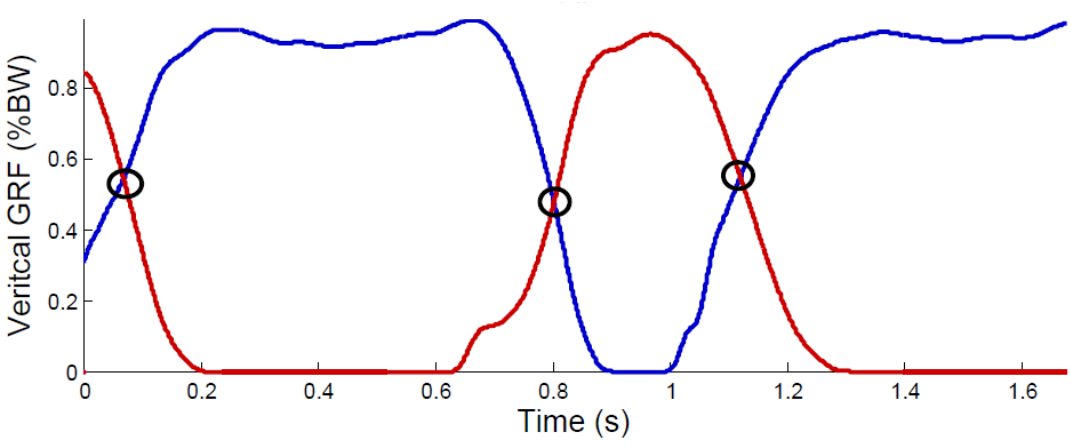


Figure 3-4. Represents the cross over point (circled in black) during paretic loading and unloading during walking.

# CHAPTER 4
# COMPENSATION AND IMPAIRMENT PATTERNS OF THE NON-PARETIC LEG IN HEMIPARETIC GAIT.

## Background

Hemiparesis post-stroke is usually considered a unilateral motor control problem of the paretic leg, while the non-paretic leg is assumed to have no deficits (sometimes even being used as a reference for comparison of the performance of the paretic side). Nevertheless, non-paretic leg performance differs from that of a healthy leg, with many differences presumed of value for compensation[12, 13, 59, 60] and some that might indicate impairment.[178] For example, Kim et al.[12] reported a high correlation between ankle plantarflexion angle in the saggital plane and ankle push-off and hip pull-off power bursts in the saggital plane of the non-paretic leg and gait speed; suggesting that the compensatory performance of the non-paretic leg itself may be an important contributing factor to the gait performance post-stroke. While non-paretic leg compensatory mechanisms may assist a person to attain a steady walking state even with a discoordinated paretic leg, it may also be the case that these compensations might also inhibit expression of a more appropriate biomechanical pattern of the paretic leg. This interference could be either a mechanical phenomenon (e.g. doing more than half of the task requirements does not allow expression of a normal pattern without requiring increased output) or it could be a neural phenomenon (e.g. the altered sensorimotor state of the non-paretic leg might interfere with normal bilateral pattern formation). For instance, pedaling studies in individuals with hemiparesis post-stroke have demonstrated a strong negative influence of the non-paretic leg participation in pedaling on the paretic leg performance, thereby further deteriorating the performance of the paretic leg pedaling performance.[11, 89] Thus, it is likely that the altered

sensorimotor state of a compensating non-paretic leg may interfere with expression of a normal motor pattern by the paretic leg. If this finding can be extrapolated to walking, the performance of the non-paretic leg should also be a clinical concern even when the focus is to improve performance of the paretic leg.

Although there is ample evidence to suggest that non-paretic leg output is a substantial contributor to the altered walking pattern post-stroke, to our knowledge no study has explicitly investigated and reported the performance of the non-paretic leg during walking after stroke. Through this study, we aim to investigate the performance of the non-paretic leg and quantify how it differs from a healthy control leg. We will test each motor pattern change we observe and present the evidence for and against the change representing compensation for the weakness and altered coordination of the paretic leg and/or a direct impairment. Compensation is a strategy employed to perform an old task in a new manner following an injury.[158] For the purpose of this study, in the context of the non-paretic leg, compensation implies the change in the motor control of the non-paretic leg, presumably either an increase in output during a normally active period or activity in a normally inactive period, to perform a function usually performed by a healthy contralateral leg that is not being performed by the paretic leg due to weakness and/or discoordination. Impairment, on the other hand, is the deviation from the generally accepted standards in the biomedical status of the body and its functions.[159] For the purpose of this dissertation, impairment in the non-paretic leg is suggested when there is an excess or deficit in the output of any parameter as compared to healthy control subjects, which would be expected to deteriorate the performance of the non-paretic leg itself.

In this study we will investigate changes in EMG activity in the non-paretic leg relative to a healthy control leg walking at the same speed by evaluating kinetic and kinematic measures during the regions of change. While observation of walking by itself will not allow us to definitely state that non-paretic leg impairment exists, we will be able to document changes in biomechanical variables that suggest compensation and demonstrate when the biomechanical consequences of an activation change may deteriorate performance. We will use kinesiological measures, such as EMG, kinetics and kinematics to evaluate the motor performance of the non-paretic leg. We will then associate these changes with the mechanical work done by a joint moment, which is an important measure of motor performance.[149] Interpretation of these measures will also be made within the context of the potentially different kinematics, which may allow muscle activity to have a different consequence than it would have with usual walking kinematics (e.g., if the non-paretic leg is placed behind the paretic leg in a step-to gait, then plantarflexor activity could produce propulsion very early in the non-paretic leg gait cycle as it will spend more of the cycle behind the center of mass of the body).

## Methods

### Participants

For the purpose of this study sixty individuals with chronic hemiparesis and twenty similarly aged healthy individuals were tested. The inclusion criteria for the study were: subjects should have hemiparesis secondary to a single onset unilateral stroke; should be able to ambulate independently with or without an assistive device over 10 m on a level surface; have the ability to walk on a regular basis at least at home; do not have any significant lower extremity joint pain and any major sensory deficits; do not have any significant lower limb contractures and any significant cardiovascular or respiratory

symptoms contraindicative to walking. Criteria for the exclusion of participants from the study were: any orthopedic or neurologic conditions in addition to stroke, or any significant musculoskeletal problems other than stroke that would limit hip and knee extension or ankle plantar flexion to neutral, or inability to provide informed consent. All participants in the study signed a written informed consent and the study was approved by Institutional Review Board of University of Florida.

Subjects were grouped into three groups based on their self-selected walking speed and compared to healthy control subjects walking at matched speeds. Three groups were defined as slow speed: walking at 0- <0.4m/s (compared to controls at 0.3m/s), moderate speed group: walking at 0.4-0.8m/s (compared to control subjects walking at 0.6m/s) and the fast speed group: walking at >0.8m/s (compared to control subjects walking at 0.9m/s).

**Procedures**

**Subject preparation**

Reflective marker spheres and rigid body clusters were positioned on specified bony prominences and limb segments respectively, to acquire 3D motion data. Marker positions were based on the Vicon PlugInGait marker set (modified Helen Hayes set). In addition, disposable bipolar Ag-AgCl surface electrodes (Vermed, Inc, bellows Falls, VT) were used to record muscle activity from eight lower extremity muscles bilaterally (TA: Tiabialis Anterior, SO: Soleus, MG: Medial Gastrocnemius, VM: Vastus Medialis, RF: Rectus Femoris, BF: Biceps Femoris LH: Lateral Hamstrings, MH: Medial Hamstrings and GM: Gluteus Medius). Adequate skin preparations were done (electrode placement site was shaved and wiped with alcohol) to facilitate maximum skin conduction. Reference electrode was placed on electrically neutral patella.

**Data Collection**

The subjects walked on the split-belt instrumented treadmill (Techmachine, Andrezieux Boutheon, France) for 30 seconds (3 trials) at their self-selected walking speed. The treadmill was started at a slower speed and the speed was gradually adjusted until the subjects' self-selected comfortable treadmill walking speed was attained. While walking on the treadmill subjects wore a safety harness (without any BWS) that was mounted to the ceiling, and would provide support in case of loss of balance. A physical therapist was also present for close supervision and providing assistance if required. Notes were made if any assistance was used during data collection and those data were not used in the final analysis. All subjects walked without ankle-foot orthosis or walking aid. Rest breaks were provided as needed by the subjects between the walking bouts.

Additionally, healthy control subjects walked at a self-selected speed and four additional walking trials, speeds introduced randomly from slow to normal: 0.3, 0.6, 0.9 and 1.2 m/s. These speeds were selected a priori to represent a likely range within which persons with stroke would elect to walk. Literature reports normal gait speed is 1.2 m/s and three incremental speeds slower than normal were chosen.[15]

**Data Recording and Processing**

Twelve camera VICON motion-capture system (Vicon Motion Systems, Los Angeles, CA) and instrumented treadmill and telemetric EMG system (Konigsberg Instruments, Pasadena, CA) were used to capture the kinematic, kinetic and EMG data as the subjects walked on the split-belt instrumented treadmill.

GRFs were acquired from each foot separately at a sampling rate of 2000 Hz, and low-pass filtered using a fourth-order, zero-lag Butterworth filter with a 20 Hz cut-off

frequency. A 13 segment musculoskeletal model was created using Visual 3D (V3D) (C-Motion, Inc., Germantown, MD). V3D models were used to conduct inverse dynamics analyses for calculation of intersegment joint kinetics. In addition, the EMG signals were collected as analog signals at 2000 Hz, which were then converted to digital signals and stored on the laboratory computer. The analog signals were amplified and pre-filtered before being digitized and stored. The EMGs were then filtered with a 40 Hz high pass filter, demeaned, rectified and then low pass filtered (25 Hz). Filtering was done with 4th order Butterworth filters in Matlab (MathWorks Inc, Natick, MA).

Data were processed with K-means cluster analysis (figure 4-3) to determine whether the muscle was active or not for each point in the gait cycle.[125] K-means cluster analysis is a statistical technique that groups similar data points in a single cluster. In the context of EMG analysis it differentiates data as “on” and “off” by finding similarities between the data points of the rectified and filtered EMG signal.[125] The individual data points are assigned to k-clusters by MATLAB such that the distance from the centroid is minimized. Therefore, the most similar data points are grouped together. The cluster with the lowest mean value corresponds to inactivity and the other clusters correspond to periods of muscle activity.[125]

**Calculations of Study Variables**

The same variables were recorded and calculated for healthy individuals and individuals with hemiparesis during walking. All the variables were calculated by averaging across all gait cycles of each trial. Since there was no statistically significant difference between the performance of the left and right side of the healthy control subjects, the mean value of the two sides was used for all the analyses.

## EMG Variables

**EMG magnitude.** Integrated EMG was used to quantify the magnitude of EMG activity over the gait cycle and for different regions of the gait cycle. It was calculated by numerically integrating absolute EMG signals using an extended open closed formula with equally spaced abscissas with respect to time (figure 4-2).

**EMG timing.** Percentage duration of activity of a muscle in each gait cycle/region is sum of all the "on" points divided by the total number of points in the gait cycle/region.

## Kinetic and kinematic variables

Total positive work done by the ankle plantarflexors, hip extensors and hip flexors was calculated in different regions of the gait cycle, as defined in figure 4-1.

Kinematics were calculated similarly for the same regions of the gait cycle.

## Statistical Analyses

Independent samples t-test were performed to compare the magnitude and timing of muscle activity in the non-paretic leg: 1) over the entire gait cycle, and 2) in different regions of the gait cycle, to healthy control subjects walking at matched speeds. The slow speed group (0-<0.4m/s) was compared to control subjects at 0.3m/s, the moderate speed group (0.4m/s - 0.8m/s) were compared to control subjects at 0.6m/s and the fast speed group (0.8m/s-1.0m/s) were compared to control subjects walking at 0.9m/s. Pearson’s correlations were conducted to evaluate the relationship between percentage average activation for the muscle in a specific region and the measures of motor performance. Significance for all tests was set at alpha < 0.05. All statistics were run using SPSS version 17.0 (SPSS, Inc.).

## Results

The results of the analyses reveal that the muscle activation in the non-paretic leg is significantly different than muscle activation of healthy control subjects during walking. In addition, we also performed a detailed analysis of the muscle activity and motor performance measures to ensure correct inference of the non-paretic leg output.

### Magnitude of EMG Activity

**Integrated EMG activity over the gait cycle.** The dominant feature of the magnitude of integrated EMG activity in the non-paretic leg gait cycle was an increase in the majority of muscles. Figure 4-4 presents the magnitude of integrated EMG activity in the non-paretic leg gait cycle as compared to the healthy control subjects walking at the matched speeds. Five of the eight recorded muscles in the slow and moderate speed groups (MG, VM, RF, LH, MH) had significantly increased integrated EMG activity in the non-paretic leg, while the fast speed group had significantly increased activity in MG, VM, RF, LH, MH, GM.

**Integrated EMG activity in different regions of the gait cycle in slow speed group.** The individuals in the slow speed group revealed significantly increased integrated EMG activity in quadriceps (RF and VM) muscles throughout stance phase in the non-paretic leg ($p< 0.05$), while all the muscles had significantly increased activity in the first region of the gait cycle (figure 4-5).

**Integrated EMG activity in different regions of the gait cycle in moderate speed group.** The upper leg muscles were mostly increased while the distal muscles were mostly unaffected. The quadriceps and hamstring muscles had significantly increased magnitude throughout the stance phase of gait cycle (except RF in region one). TA and SO did not reveal significant difference in any region of the gait cycle

(except SO in region six), while the magnitude of MG was increased in first, second and third region of the gait cycle (figure 4-6).

**Integrated EMG activity in different regions of the gait cycle in fast speed group.** The upper leg muscles had significantly increased magnitude in the stance phase of gait cycle. Furthermore, MG also had significantly increased activity in the stance phase (SO only region four) of the gait cycle. All muscles (except TA) had increased activity in the fourth region, while there was almost no difference in the magnitude of muscle activity in fifth and sixth region (swing phase) of the gait cycle (except VM in region 5 and GM in region 6) (figure 4-7).

**Timing of EMG Activity**

**Percentage duration of muscle activity for the entire gait cycle.** Only the subjects in the slow speed group showed significant changes (all muscles increased, TA activity reduced) in the percent duration of muscle activity for the entire gait cycle. Unlike the magnitude measures in which there were many changes, the individuals in moderate and fast speed groups had no significant difference in the percentage duration of activity as compared to healthy control subjects (except SO, $p=0.009$) (figure 4-8).

**Percentage duration of muscle activity in different regions of the gait cycle.** Individuals in slow group revealed most differences in the muscle activity, specifically most muscles revealed significant difference in the sixth and first region of the gait cycle. LH revealed significantly increased activity in all the regions of the gait cycle (except region five) (figure 4-9). On the other hand the duration of activity in individuals walking at moderate speed revealed significant difference in the duration of activity in SO and LH during the stance phase (figure 4-10). Participants walking at faster speeds

did not differ from the healthy control subjects walking at the matched speeds except for SO and LH in region four and MH in the second region of the gait cycle (figure 4-11).

**Correlation Analysis**

**H1 and LH & MH in region one of the gait cycle.** In the slow and fast speed groups the magnitude of LH activity was significantly positively ($r= 0.536$, $p=0.002$ and $r = 0.966$, $p= 0.008$) related to H1. However, individuals in the moderate speed group did not show any significant relation between any of the hamstrings muscles and positive work at hip in region one. However, the duration of activity of MH in the first region had a significant negative correlation with H1 ($r = -0.433$, $p = 0.01$).

**SO and MG muscle activity and A2 in region four of the gait cycle.** Although the duration of SO (also fast) and MG (also slow) in the fourth region of the gait cycle was significantly greater in the moderate speed groups, there was no significant correlation between the muscle activity and the positive work done at the ankle in that region of the gait cycle. Furthermore, the increased magnitude of SO in the fourth region of the moderate and fast speed group also did not reveal any significant correlation.

**TA muscle activity and A2 in region four of the gait cycle.** There was no significant correlation between the magnitude and duration of the TA activity in region four in all the three speed groups.

**RF muscle activity and peak knee flexion angle in region four and five of the gait cycle.** The percentage duration of the RF activity in the fourth region of the gait cycle was significantly negatively correlated with the peak knee flexion in individuals in the moderate speed group ($r = -0.464$, $p = 0.03$). However, no significant correlation was found in individuals in the slow and fast speed groups.

**MH and LH muscle activity and positive hip extensor work in region three and four.** A significant correlation was found between the magnitude of LH and the positive hip extensor work ($p=0.536$, $r= 0.002$) in individuals walking at the slow speed. However, no significant correlation was revealed in any other group.

**MG and SO muscle activity and leg angle in region one.** No significant correlation was present between the increased plantarflexor activity and the leg angle in the first region of the gait cycle.

## Discussion

This study demonstrates that the muscle activity of the non-paretic leg differs from healthy control leg of subjects walking at matched speeds and many of these changes appear to be compensatory in nature. This is because the activity is either increased in the muscles and regions known to be the primary power bursts (e.g., H1, A2 and H3) or in other specific regions of the gait cycle that revealed statistically significant correlation with the measures of motor performance. This implies that individuals walking post-stroke employ several compensatory mechanisms in order to attain a steady walking state. Some of these compensations are simply increased output of the usual dominant power bursts of a healthy leg, such as increased activity in the plantarflexors during late stance and the hip extensors during early stance. Others are novel adaptations in the non-paretic leg such as hamstrings activity in late stance to generate propulsion, early stance plantarflexor activity that generates propulsion because the non-paretic leg is not placed as far in front of the body, and mid to late stance activity in the knee extensors that facilitates paretic leg swing.

With respect to limitations of identifying impairment, it is important to emphasize that the performance of the non-paretic leg was evaluated during bilateral walking and

not in an experimental condition specifically designed to test the possibilities of non-paretic leg motor control. Nevertheless, we explicitly defined compensation and impairment for our study. Furthermore, because all of the muscles as well as the kinetic and kinematic measures influence walking and are not independent, we did not correct for multiple comparisons in our study. Since the results of the statistical analyses were mostly in agreement with our initial hypotheses that the muscle activity will be increased in the non-paretic leg based on some evidence in the literature, as opposed to many more random appearing changes in different directions, we feel that this choice was justified. Nonetheless, we believe that the reader can appropriately assess importance of the changes observed in the coordination of the non-paretic leg.

**Muscle Activity over the Gait Cycle**

Integrated EMG and percentage duration of activity over the gait cycle demonstrate that in general the muscles in the non-paretic leg are activated at a significantly greater magnitude and for a significantly greater duration than those of the healthy control subjects walking at matched speeds. This therefore suggests the non-paretic leg should not be considered unaffected or used as a “control” to gauge the performance of the paretic leg.

The pattern of difference in the global measures of EMG between the non-paretic leg of individuals in the three speed groups and the control subjects at matched speeds suggests that during walking the individuals with severe stroke had greater changes in the motor pattern of the non-paretic leg than the individuals with less severe stroke. For instance, the total integrated muscle activity was significantly greater in five, five and six muscles in the non-paretic leg of individuals in the slow, medium and fast speed groups respectively. Furthermore, only individuals post-stroke categorized in the slow speed

group revealed significantly greater percentage duration of activity in all eight recorded muscles (except TA, TA had reduced percentage duration of activity). No difference was present in moderate and fast speed walkers. The overall increased percentage duration of activity of different muscles over the gait cycle in the non-paretic leg suggests that these muscles are firing for greater percentage duration, which could either indicate compensation for the impaired coordination of the paretic leg after stroke, or impairment of the non-paretic leg to efficiently modulate muscle activity, i.e. turn 'on' and 'off' muscles efficiently, during walking. To discern the role of the non-paretic leg in compensation vs. impairment we investigated the muscle activity in the different regions of the gait cycle.

**Muscle Activity during Different Regions of the Gait Cycle**

Reduced propulsion and thus reduced walking speed is one of the primary concerns after stroke. The non-paretic leg typically generates greater propulsive force[179] to compensate for the weak and discoordinated paretic leg. Several mechanisms are thus involved.

One compensatory mechanism identified was increased hamstrings activity during early stance, presumably to increase propulsion by positive work done at the hip, i.e. H1. To this end, the results of our analysis reveal that the magnitude and duration (except in the fast speed group) of LH and MH are significantly increased in the first region of the gait cycle of the non-paretic leg (presumably contributors to the H1 power burst). The increased firing of the MH and LH muscles facilitate an increase in the magnitude of the usually dominant positive H1 power burst to increase propulsion. Simulation analysis reported an important role of the hamstrings in forward propulsion of the trunk [163] (instead of support and stability[163, 180]) during early stance. Thus increased

hamstring activity might compensate for reduced propulsion by promoting forward acceleration of the trunk at a time when the paretic leg is in pre-swing and is not contributing as much propulsion as a healthy leg normally would. This is further supported by a significant positive correlation between the positive work done by the hip extensors H1 and the hamstring (MH and LH) activity during this phase of the gait cycle.

Increased activity in the plantarflexors earlier in the stance phase was likely a novel compensation to produce increased propulsion that might possibly be facilitated by the altered asymmetric kinematics. The increased plantarflexors activity in this region of the gait cycle may facilitate propulsion if the non-paretic leg is not placed ahead of the body center of mass (as happens in a step–to-gait[160]). Studies have suggested that shorter non-paretic leg step results in reduced paretic propulsion (because the paretic plantarflexors are put in a biomechanical disadvantage position[160]). Therefore, the increased activity of the SO and MG in this region of the gait cycle may compensate for the reduced propulsion generation. Although as a group there was no significant correlation between the non-paretic leg angle and the plantarflexor activity in first region, there was a significantly greater positive work done by the plantarflexors in this region, particularly in individuals walking at slow speeds. Thus, those at slow speeds were in a kinematic position where they were able to plantarflex the ankle in this region, which is normally characterized by dorsiflexion.

Another potentially novel compensation by the non-paretic leg is increased VM and RF activity in the second and third region of the non-paretic gait cycle in the slow and moderate speed group individuals walking post-stroke. According to a recent simulation study the increased VM activity in the stance phase of the non-paretic leg

might facilitate the propulsion of the swinging paretic leg.[181] They also reported that the contribution of RF and VM was increased in the non-paretic leg of group of slow walkers (limited community ambulatory) as compared to group of fast walkers (community ambulatory)[181], consistent with the increased VM activity in slow and moderate speed group and not in the fast speed group of our analyses. The increased VM firing in the stance phase increases propulsion in the paretic leg to maintain a walking state. Furthermore, the increased VM and RF activity in the stance phase of the non-paretic leg also generates greater force to maintain the stability, while the total body weight is being supported. According to the simulation analysis, SO is the primary muscle to maintain the limb in extension to support body weight during the second and third region.[163] However, our analysis shows that the activity of SO was reduced in the slow and moderate speed group, therefore VM and RF contract to compensate.[161]

A third likely novel compensation in the non-paretic leg muscle activity is a much more pronounced hamstring burst in the late stance phase of the gait cycle that appears to be associated with increased propulsion. A similar pattern of activity has been reported by Shiavi et al.[69] and Buurke et al.[126] in individuals post-stroke and in some studies in individuals post-amputation.[182] Based on the information obtained from the amputation literature, Buurke et al.[126] suggested that the contraction of hip extensors (hamstrings) helps in generation of extension of the non-paretic limb, which might thereby facilitate the swing of the paretic leg, thus compensating for the poor plantarflexors and hip flexors. Generation of an extra extension force on the non-paretic leg, places the paretic leg in a mechanically advantageous position, which facilitates its swing without actually transferring energy to the swing leg. This, atypical power burst

(found on the non-paretic side of individuals post-stroke) increases the propulsion of the otherwise weak and discoordinated paretic leg. This phenomenon is further supported by a significant positive correlation between the positive hip extensor work and the MH and LH activity during the third region of the gait cycle in the slow speed group. However, no significant correlation was found in the moderate and fast speed group despite significantly increased magnitude of the LH and MH muscle. This could be due to high variability in the slow speed group whereby only some individuals extensively employ this mechanism in order to maintain a steady walking state.

An expected compensation that was observed was increased plantarflexor output during the usual A2 power burst. The magnitude and duration of the plantarflexors were increased in the fourth region of slow and moderate speed group individuals, in order to increase the magnitude of A2. A2 is the most important source of positive work generation in the gait cycle and thus helps to generate greater propulsive force to attain faster walking speed. This greater contraction of the plantarflexors on the non-paretic side generates greater force to produce increased propulsion to maintain steady walking state. In addition to increased plantarflexor activity there is an additional burst of activity in the TA during the fourth region of the gait cycle. In theory, the increased magnitude of activity of TA in this region of the gait cycle may interfere with generation of propulsion during this phase.[160]

An increased burst in the RF muscle during the pre-swing phase (both duration and timing) was exhibited in the non-paretic leg of individuals in the slow and moderate speed groups. Excessive activity of RF has been cited as a contributor to reduced knee flexion.[183, 184] Furthermore, RF tends to decrease the peak knee flexion velocity [170, 171]

and over activity during region four and five is often implicated in stiff knee gait.[170] Additionally, Hernandez et al.[185] in their electrical stimulation study reported that the stimulation of the RF before the toe-off largely reduced the peak knee flexion during swing as opposed to the stimulation during the swing phase. Interference with knee flexion is supported by a significant negative correlation between the peak knee flexion angle and the RF activity in the moderate speed group. However, lack of any significant correlation in the slow speed groups could be attributed to the increased contribution by other muscles (MG, iliopsoas, and hamstrings) to knee flexion.

**Implications of Analysis**

The conventional or novel compensatory mechanisms by the non-paretic leg are aimed at either providing support, stability or for providing greater propulsion. This may be accomplished by increase in normally existing sources of positive work. For instance, increased hamstring activity in the first region of the gait cycle provides greater forward propulsion of the trunk.[163] Additionally, increased activity of the plantarflexor muscles (MG and SO) also facilitate generation of greater propulsion of both the trunk (SO)[162] and leg (MG)[162] in the fourth region of the gait cycle. Additionally, novel power generation patterns of the non-paretic leg: increased activity of the VM and RF in the second and third region of the gait cycle provides increased support to fully loaded non-paretic leg.[161] In addition it has also been attributed to increase the propulsion of the swinging paretic leg.[181] Finally, the extra burst of the MH in the late stance or third and fourth region of the gait cycle generates extensor moment and provides biomechanical advantage to the paretic leg, thereby compensating for the weak plantarflexors and hip flexors.[126, 182]

On the other hand the non-paretic leg exhibits some increases in muscle activity which may not have a positive influence on walking and are might suggest impairments. For example, increased activity of TA in the pre-swing phase (region four) of the gait cycle, that likely interferes with generation of propulsion by the non-paretic leg[160] and the increased burst of RF during the fourth and fifth region of the gait cycle, possibly negatively influences knee angle and knee flexion velocity during the swing phase.[170, 171, 185]

**Clinical Relevance**

The clinical relevance of this study lies in providing evidence for the altered performance of the non-paretic leg post-stroke (which is usually considered unaffected). Therefore, the results of this study emphasize that stroke is not a unilateral motor control problem as often suggested, and there is a bilateral involvement. This study presents compensatory mechanisms of the non-paretic leg and understanding of these mechanisms is important to provide effective rehabilitation. For example, increased MH activity in late stance increases extension of the non-paretic leg, which in turn provides mechanical advantage to the paretic leg to swing the leg through without greater recruitment of muscle fibers. However, if the excessive extension of the non-paretic leg is prevented during therapy, it would facilitate greater use of the paretic leg plantarflexors and hip flexors to swing the leg in order to attain a functional walking state. In summary, to improve walking after stroke, it is important not only to investigate and understand the mechanics of paretic leg output, but understanding of the non-paretic leg performance provides additional insights.

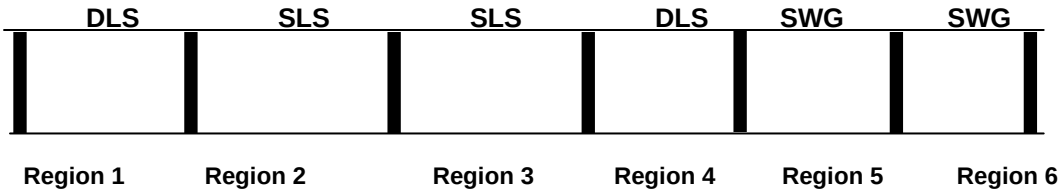


Figure 4-1. Represents the different regions of the gait cycle. At the top are the marked the different physiological phases of the gait cycle (DLS= double limb support, SLS= single leg support, SWG= swing phase) and at the bottom are the corresponding regions used in this study.

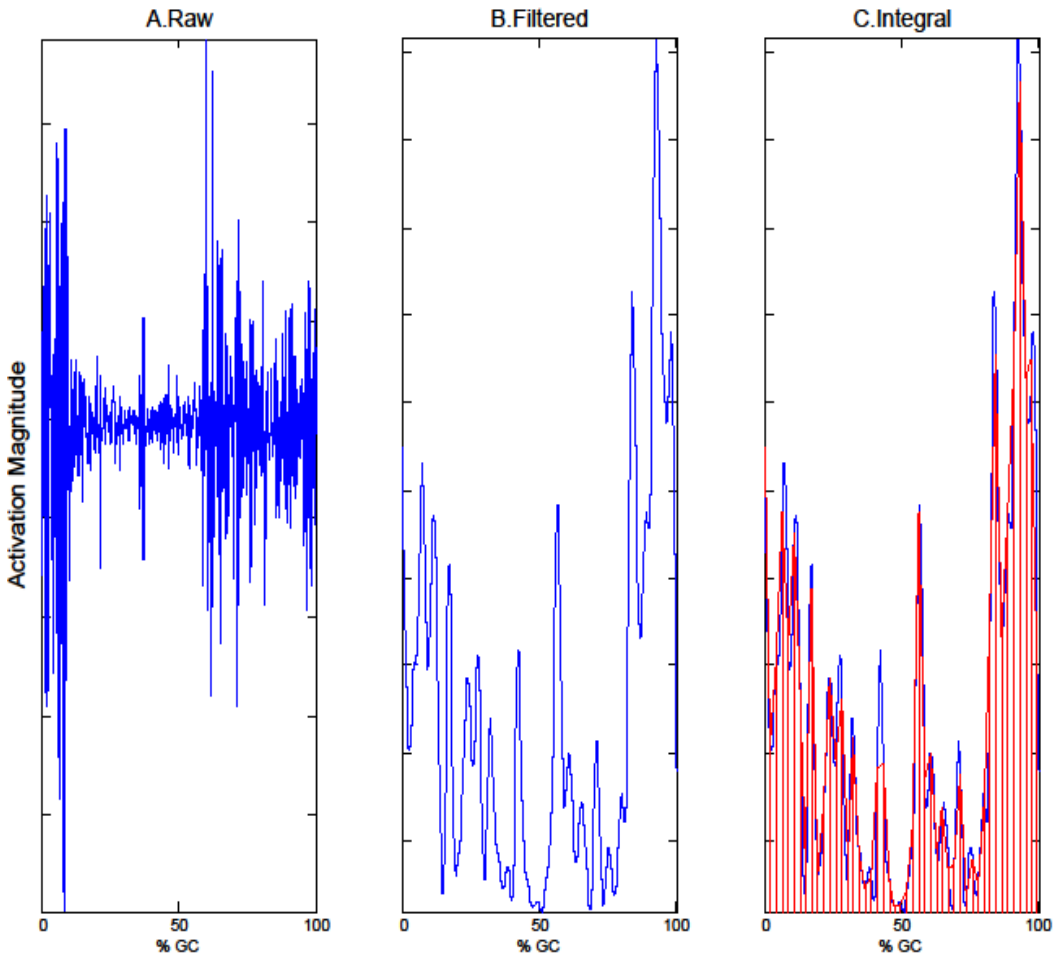


Figure 4-2. Represents the calculation of integrated EMG activity. First figure represents the raw EMG signal, second panel represents the rectified and filtered EMG signal and the third figure represents integration rule with equally spaced abscissas (red lines), with respect to time in the gait cycle.

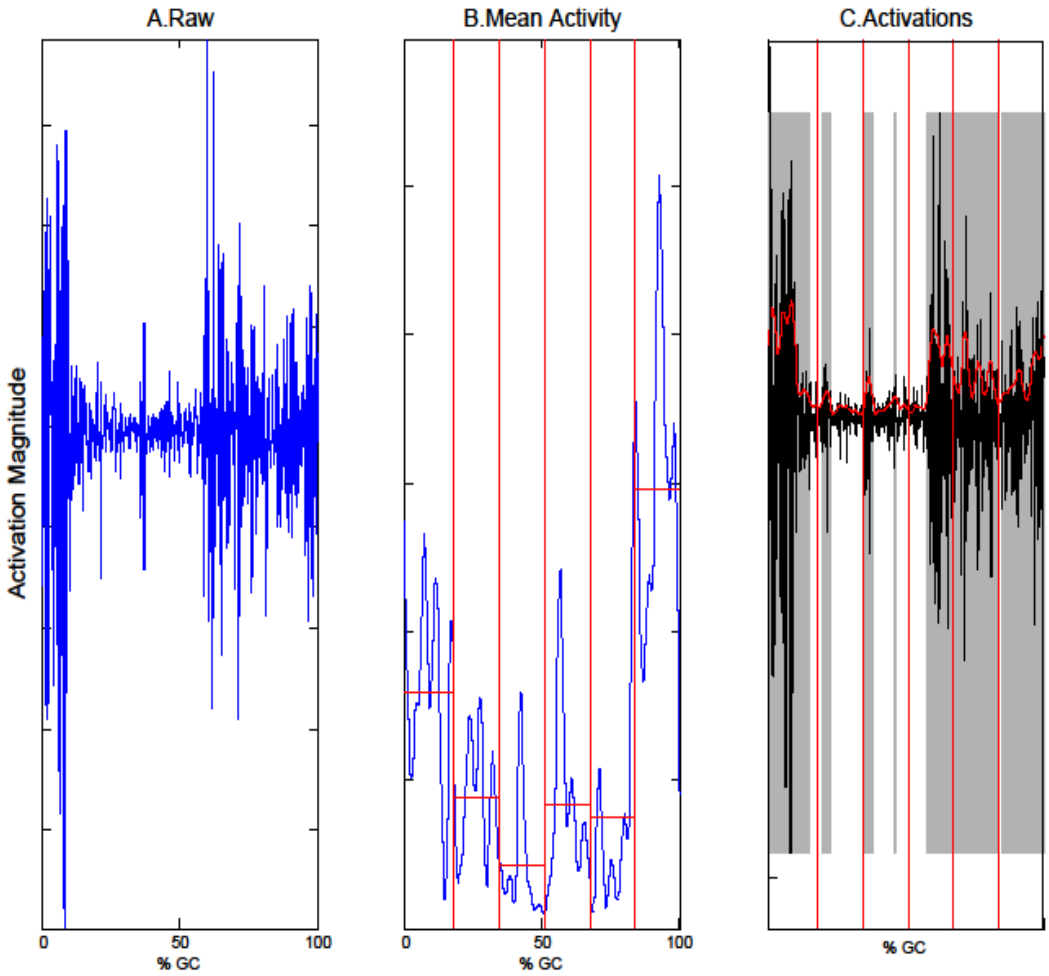


Figure 4-3. Represents the steps of k-means analysis. First figure represents the raw EMG magnitude, second figure shows the filtered rectified EMG (linear envelope) and the third figure represents the original raw EMG signal with the detected periods of activity indicated in gray shaded areas

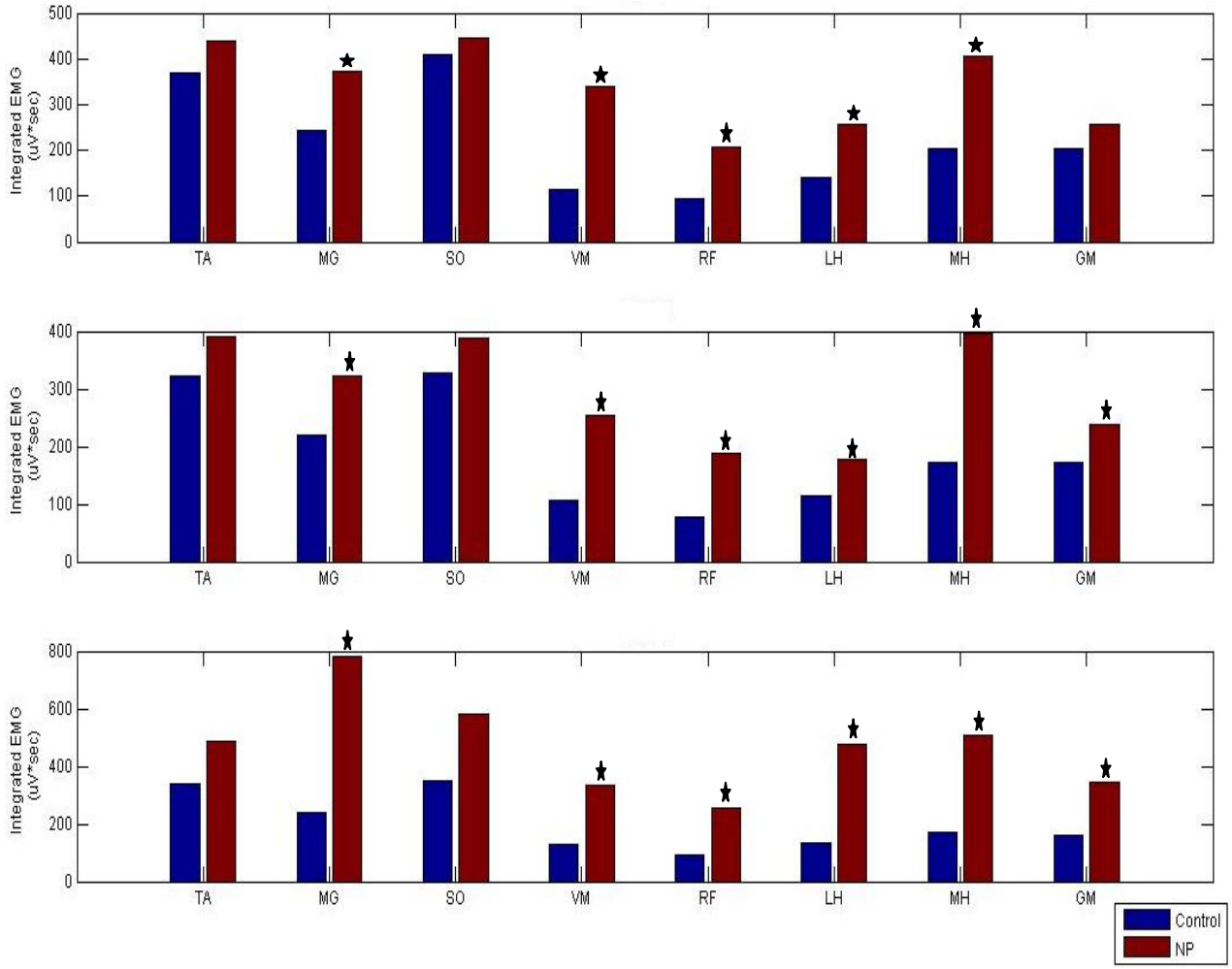


Figure 4-4. Integrated EMG activity for the entire gait cycle in the different groups is plotted to represent the magnitude of muscle activity. Group 1 is slow speed group, group 2 is moderate speed group and group 3 is fast speed group. Y-axis represents Integrated EMG activity over the gait cycle in µVs and x-axis represents different muscle (TA= Tibialis Anterior, MG= Medial Gastrocnemius, SO= Soleus, VM= Vastus Medialis, RF= Rectus Femoris, LH= Lateral Hamstrings, MH= Medial hamstrings, GM= Gluteus Medius). Red bars represent the activity of the non-paretic leg and Blue bars represent the control subjects walking at matched speeds.

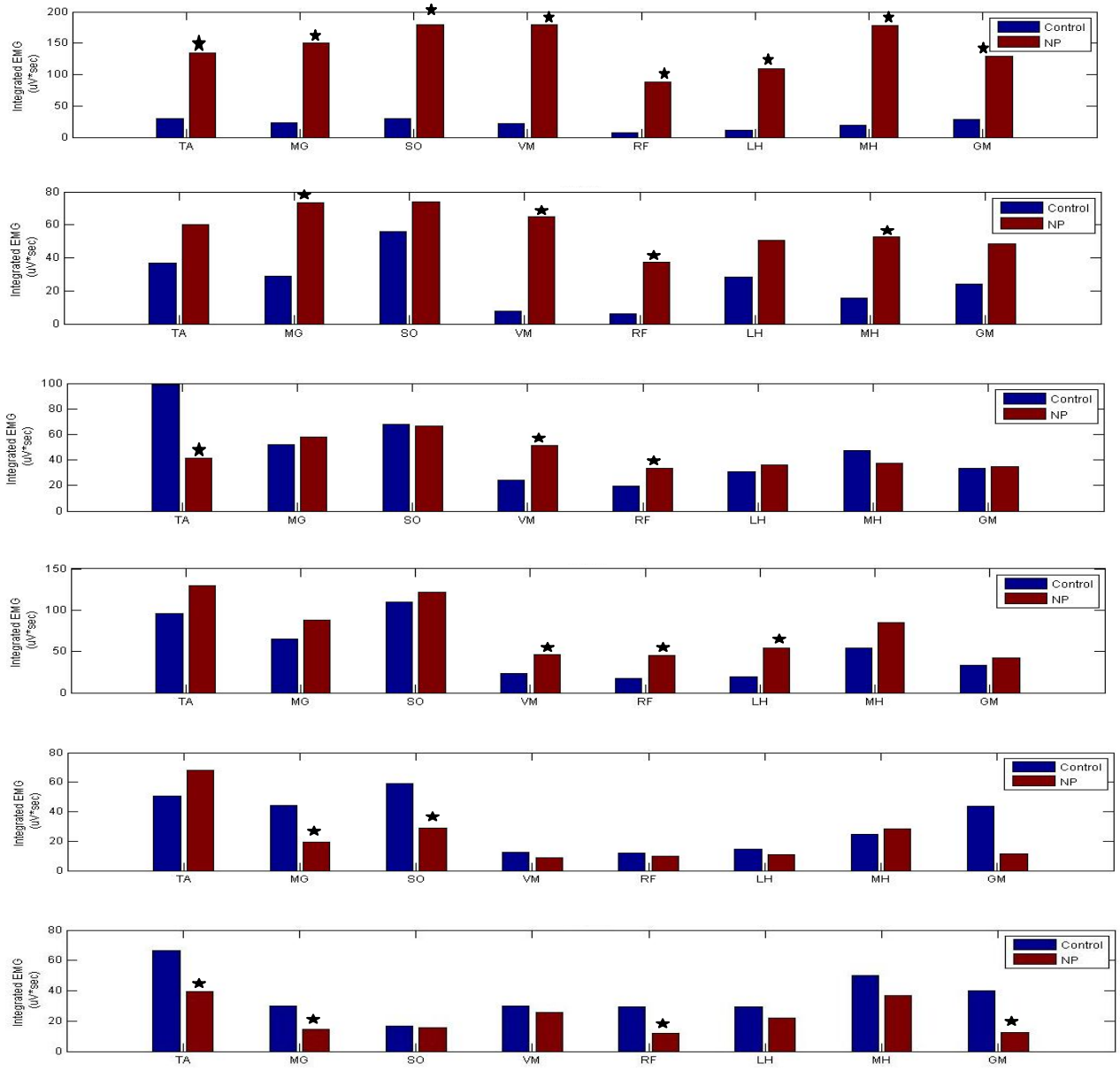


Figure 4-5. Represents intergrated EMG activity in different regions of the gait cycle in individuals in slow speed group. Each panel of plots represents different regions of gait cycle, ordered first to sixth. Y-axis represents integrated EMG (μVs), x-axis is different muscles (TA= Tibialis Anterior, MG= Medial Gastrocnemius, SO= Soleus, VM= Vastus Medialis, RF= Rectus Femoris, LH= Lateral Hamstrings, MH= Medial hamstrings, GM= Gluteus Medius). Red bars represent the activity of the non-paretic leg and Blue represent the control subjects walking at matched speeds.

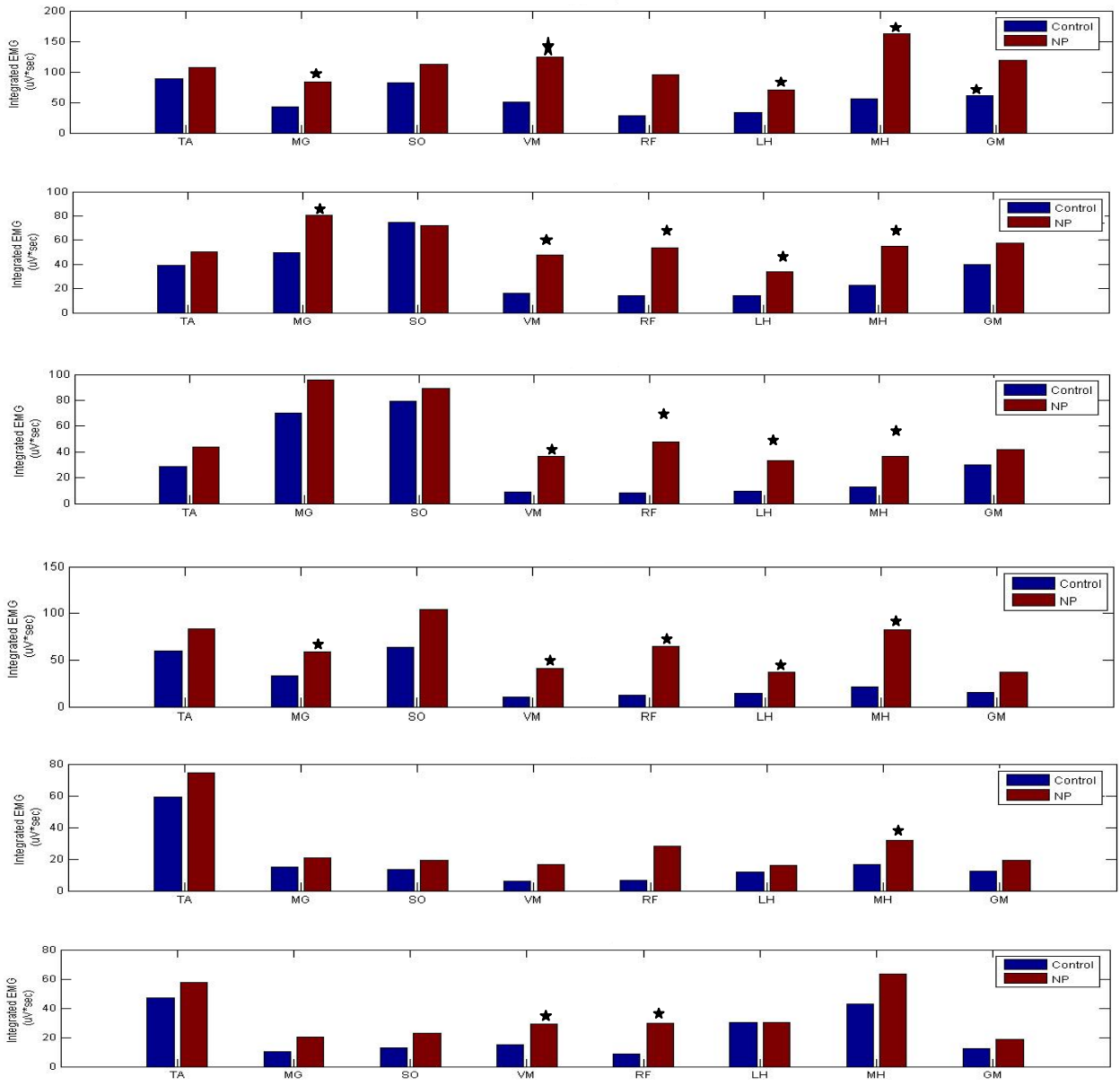


Figure 4-6. Represents intergrated EMG activity in different regions of the gait cycle in individuals in moderate speed group. Each panel of plots represents different regions of gait cycle, ordered first to sixth. Y-axis represents integrated EMG (µVs), x-axis is different muscles (TA= Tibialis Anterior, MG= Medial Gastrocnemius, SO= Soleus, VM= Vastus Medialis, RF= Rectus Femoris, LH= Lateral Hamstrings, MH= Medial hamstrings, GM= Gluteus Medius). Red bars represent the activity of the non-paretic leg and Blue represent the control subjects walking at matched speeds.

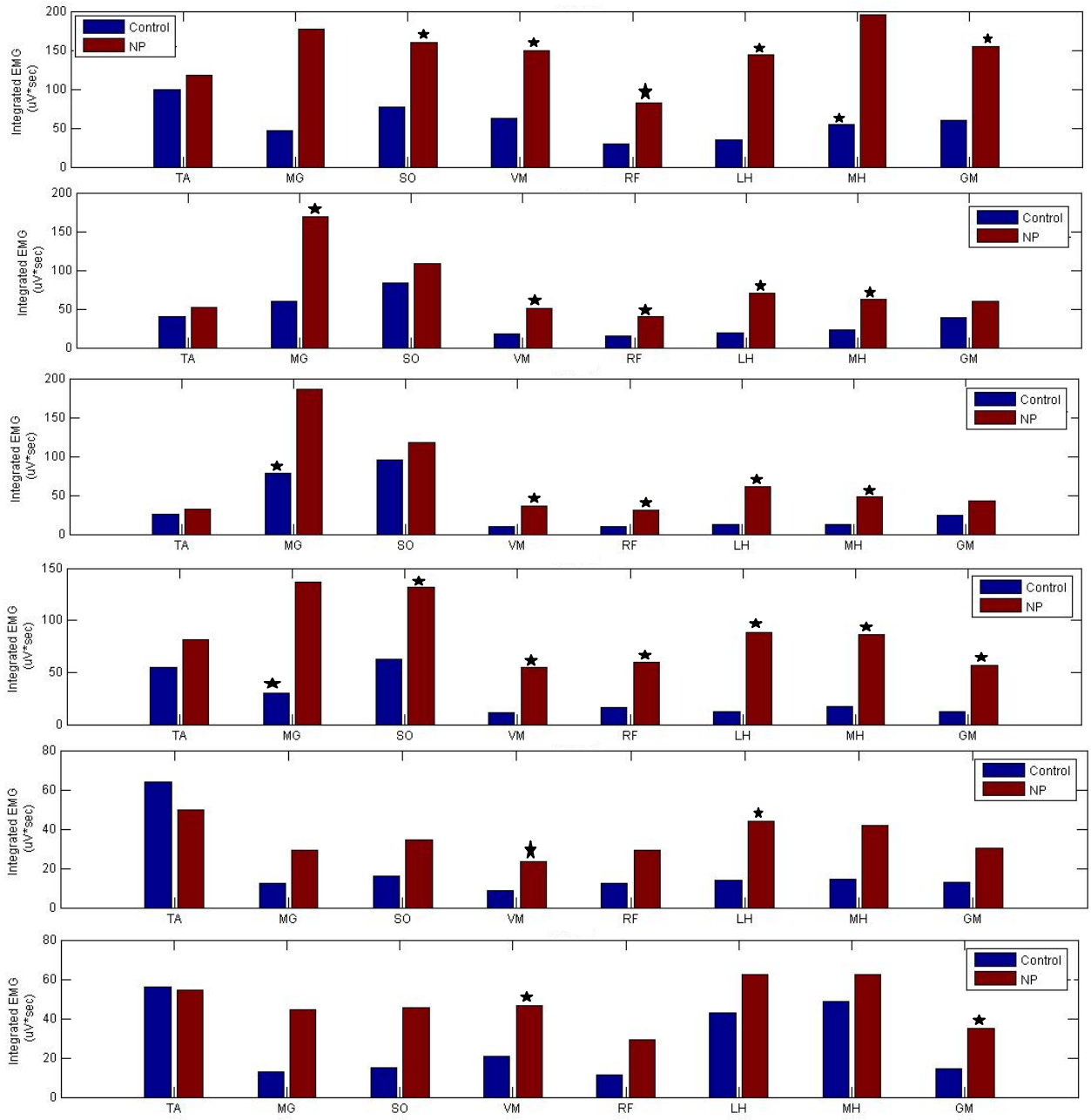


Figure 4-7. Represents intergrated EMG activity in different regions of the gait cycle in individuals in fast speed group. Each panel of plots represents different regions of gait cycle, ordered first to sixth. Y-axis represents integrated EMG (µVs), x-axis is different muscles (TA= Tibialis Anterior, MG= Medial Gastrocnemius, SO= Soleus, VM= Vastus Medialis, RF= Rectus Femoris, LH= Lateral Hamstrings, MH= Medial hamstrings, GM= Gluteus Medius). Red bars represent the activity of the non-paretic leg and Blue represent the control subjects walking at matched speeds.

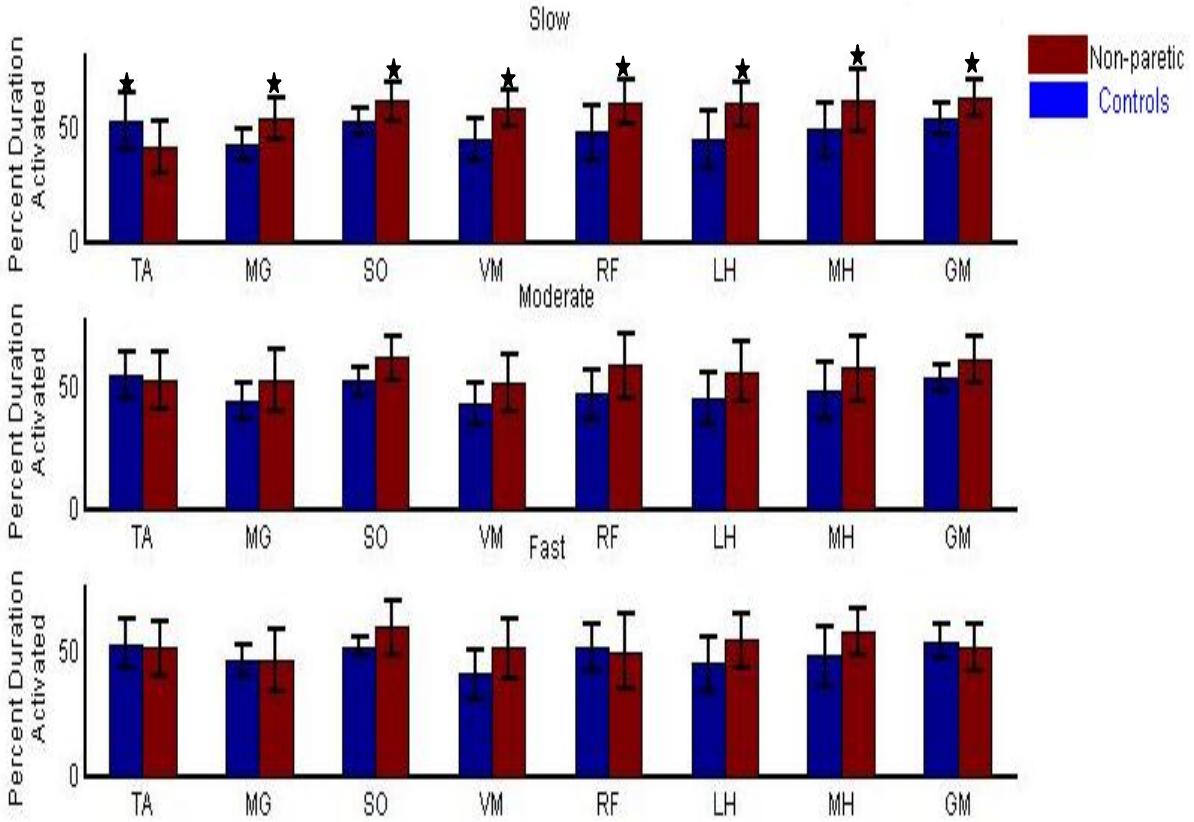


Figure 4-8. Represents percentage duration of EMG activity in different speed groups (slow, moderate and fast speed). Y-axis represents percentage duration activated over the gait cycle in µVs and x-axis represents different muscle (TA= Tibialis Anterior, MG= Medial Gastrocnemius, SO= Soleus, VM= Vastus Medialis, RF= Rectus Femoris, LH= Lateral Hamstrings, MH= Medial hamstrings, GM= Gluteus Medius). Red bars represent the activity of the non-paretic leg and Blue bars represent the control subjects walking at matched speeds.

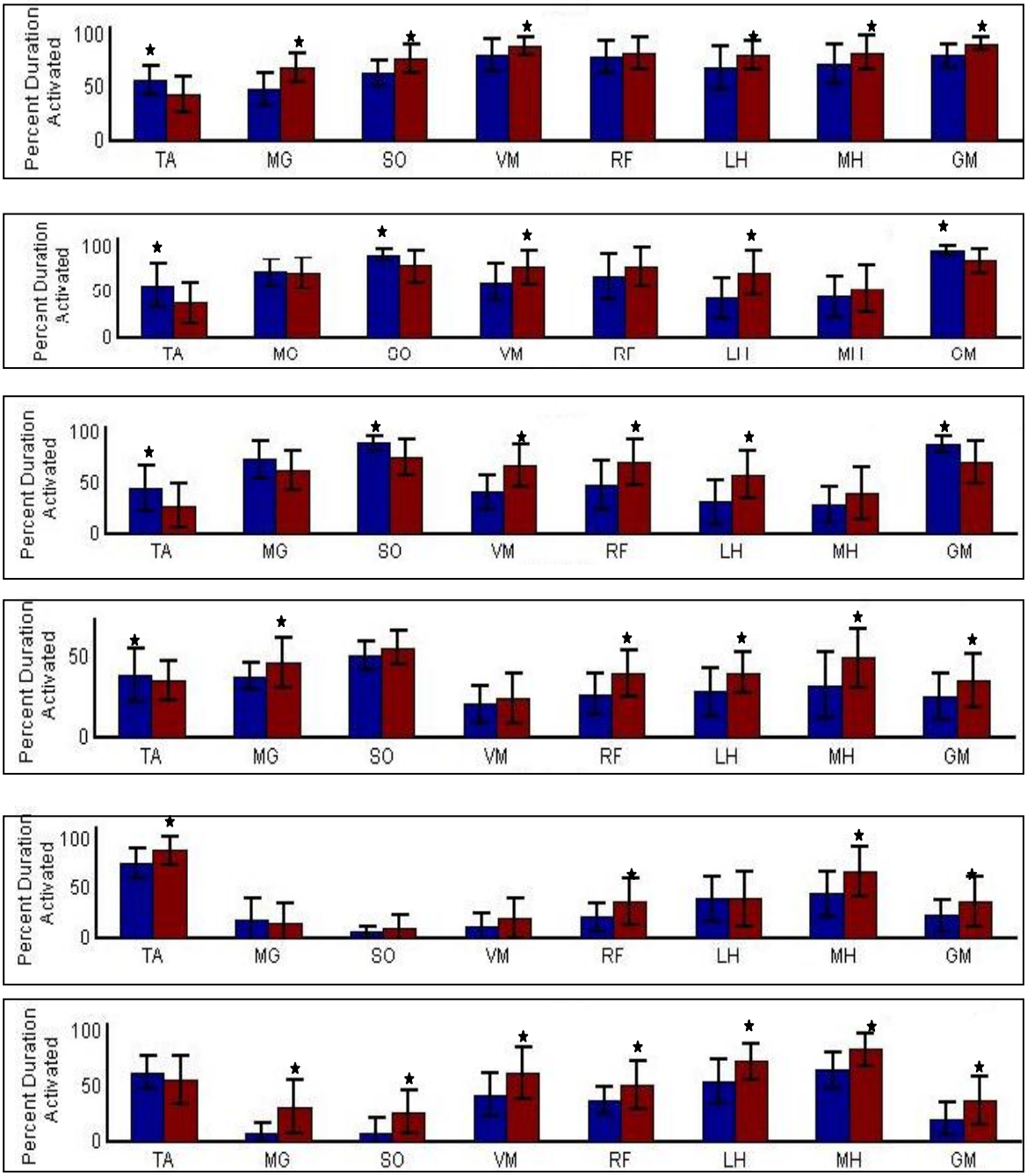


Figure 4-9. Represents percentage duration of EMG activity in different regions of the gait cycle in individuals in slow speed group. Each panel of Bar plots is different region of the gait cycle, from 1-6. Y-axis represents percentage duration activated in region of gait cycle and x-axis represents muscle (TA=Tibialis Anterior, MG=Medial Gastrocnemius, SO=Soleus, VM=Vastus Medialis, RF=Rectus Femoris, LH=Lateral Hamstrings, MH=Medial hamstrings, GM=Gluteus Medius). Red bars represent the non-paretic leg and Blue bars represent the control subjects walking at matched speed

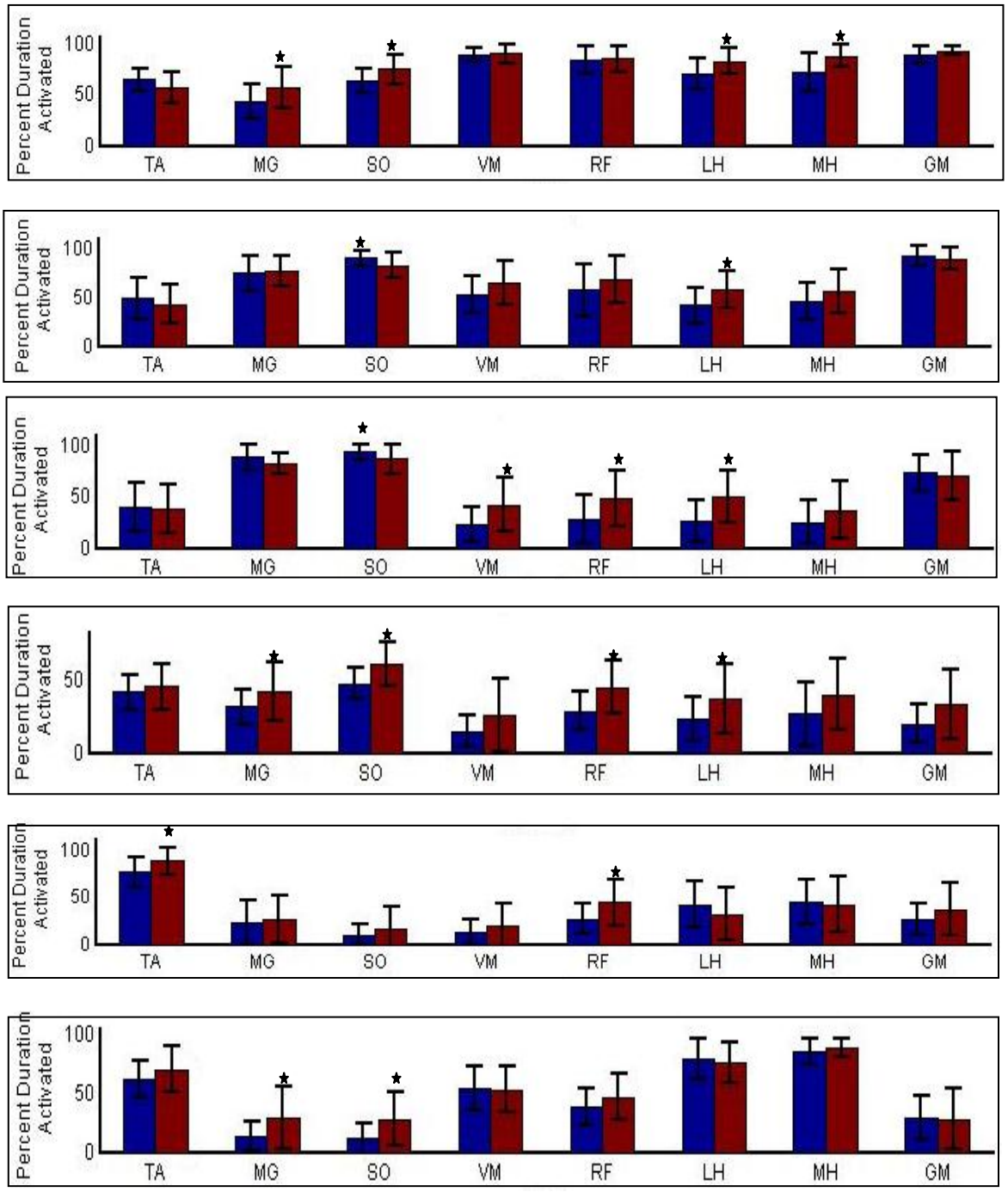


Figure 4-10. Represents percentage duration of EMG activity in different regions of the gait cycle in individuals in moderate speed group. Each panel of Bar plots is different region of the gait cycle, from 1-6. Y-axis represents percentage duration activated in region of gait cycle and x-axis represents muscle (TA=Tibialis Anterior, MG=Medial Gastrocnemius, SO=Soleus, VM=Vastus Medialis, RF=Rectus Femoris, LH=Lateral Hamstrings, MH=Medial hamstrings, GM=Gluteus Medius). Red bars represent the non-paretic leg and Blue bars represent the control subjects walking at matched speeds.

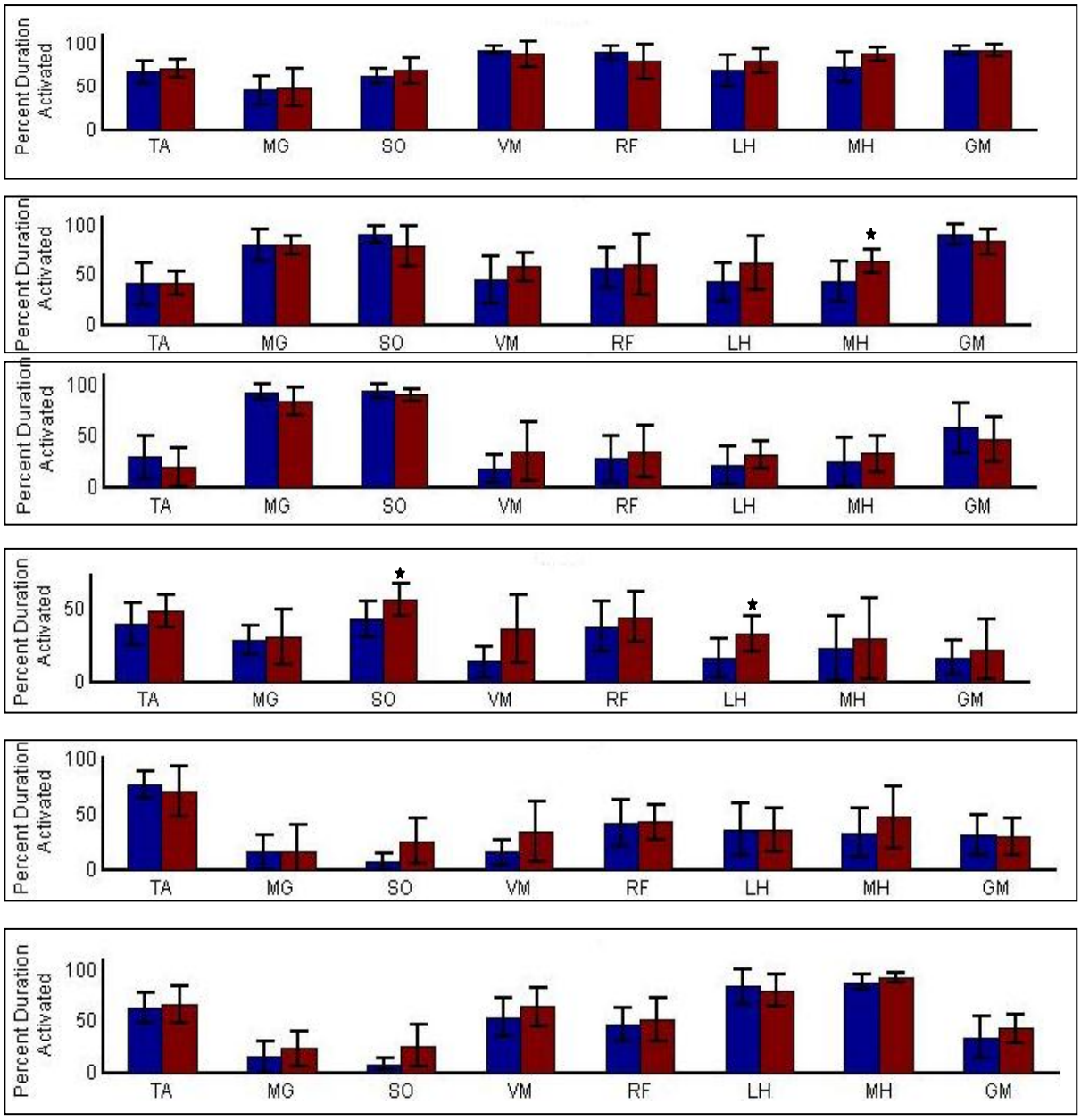


Figure 4-11. Represents percentage duration of EMG activity in different regions of the gait cycle in individuals in fast speed group. Each panel of Bar plots is different region of the gait cycle, from 1-6. Y-axis represents Percentage duration activated in region of gait cycle and x-axis represents muscle (TA=Tibialis Anterior, MG=Medial Gastrocnemius, SO=Soleus, VM=Vastus Medialis, RF=Rectus Femoris, LH=Lateral Hamstrings, MH=Medial hamstrings, GM=Gluteus Medius). Red bars represent the non-paretic leg and Blue bars represent the control subjects walking at matched speeds.

# CHAPTER 5
# RESPONSE OF NON-PARETIC LEG TO LOCOMOTOR TRAINING POST-STROKE

## Background

Stroke is one of the leading causes of death and long-term disability in the United States of America.[4] 75% of individuals affected by stroke have some level of long term disability[4] and less than 50% individuals can gain independent walking status.[15] Ability to walk independently is one of the most common stated goal of individuals post-stroke[20] and therefore, is the primary concern of therapists during rehabilitation.

Most rehabilitation techniques focus primarily on the performance of the paretic leg and the non-paretic leg is ignored, without much concern about the mechanisms used by the non-paretic leg to compensate for the weakness and discoordination of the paretic leg. In chapter 4 of this dissertation we investigated the performance of the non-paretic leg during walking post-stroke and revealed several expected and novel compensatory mechanisms employed by the non-paretic leg to accomplish walking. One conclusion from that study is that it is likely that the increased compensation by the non-paretic leg would inhibit development of biomechanically correct patterns in the paretic leg. Therefore, the altered performances of the non-paretic leg should receive clinical attention during rehabilitation that seeks to improve paretic leg motor output.

We believe that it is important to emphasize that allowing altered performance of the non-paretic leg while promoting proper paretic leg movement is likely not optimal for developing coordinated walking patterns. For example, increased activity by the non-paretic hip extensors in the stance phase (i.e., compensation by non-paretic leg) might result in reduced employment of the ankle plantarflexors and hip flexors of the paretic leg during the swing phase. If so, training that reduces the non-paretic hip extension in

the late stance phase might result in better expression of the paretic leg muscles. Furthermore, evidence in the pedaling literature[11, 89] suggests that the impaired motor pattern of the paretic leg during pedaling arises in part from generation of non-paretic leg motor activity and the interlimb afference associated with non-paretic leg loading and movement. Therefore, it is important to focus on both the paretic and the non-paretic leg in the rehabilitation regimen to modulate the activity of both and achieve optimal output.

Muscle activity or EMG is one of the most commonly used measures to evaluate motor coordination. There have been very few studies which have reported changes in muscle coordination during walking after rehabilitation following stroke. One of the early studies was reported by Shiavi et al.[69], whereby they reported abnormal muscle activation patterns in both the paretic and the non-paretic leg. Lately, studies have reported no change in the temporal characteristics of muscles in both the paretic and non-paretic leg following rehabilitation.[126] Another study reported an increase in the magnitude of semimembranosus muscle after training, in both low response and high response group and additionally, the high response group demonstrated an increase in the average intensity of the soleus muscle.[147] While these results may not be in conflict, note that there were differences in training methodology the use of task-specific locomotor training (LT) i.e. body weight supported treadmill training (BWSTT)) in the latter study and the use of Neuro-Developmental therapy (NDT) in the former study. However, to our knowledge, no study has reported changes in non-paretic leg muscle activity after a task-specific training, like the BWSTT.

BWSTT is a task specific rehabilitation technique based on the principles of activity dependent plasticity and focuses on the capacity to capitalize on inherent central nervous system plasticity by creating a permissive environment in which the focus of therapy shifts from compensatory mechanisms to recovery of function through production of normalized movement patterns.[108, 123, 186-189] BWSTT allows spatio-temporally and kinematically effective lower limb stepping practice with both legs, with adequate body weight support to facilitate proper trunk and limb alignment and weight shift during stepping.

The primary goal of the present study is to investigate if the motor pattern of the non-paretic leg (identified in the second study) can be changed following a task-specific locomotor training. We will evaluate if the pattern of the non-paretic leg normalizes following the locomotor training such that the compensatory mechanisms are reduced.

## Methods

### Subject Demographics

Individuals with chronic stroke were recruited for this study at the Department of Veterans Affairs Medical Center, Gainesville, FL. Thirty individuals with chronic hemiparesis (19 males; 60.2±13.2 (SD) years of age; affected side left-18) participated in the study. Data for healthy control subjects was not collected for this study; instead, we used the data previously reported from our laboratory (chapter 4). The healthy subjects did not undergo any training sessions, but only went through full biomechanical testing.

Inclusion criteria for persons post-stroke included; hemiparesis secondary to a single unilateral stroke and older than 18 years of age with a stroke within last five years (> 1 year). The subject should had to residual paresis in the lower extremity, Fugl Meyer

score < 34, but ability to sit unsupported for 30 s and be able to walk at least 10 feet with maximum one person assistance. In addition the subject needed to have a self-selected speed of <0.8 m/s and be able to follow three step commands and provide informed consent. Whereas, subjects who lived in nursing homes prior to the stroke or were unable to walk 150 feet prior to stroke had/ have any neurological or cardio-vascular or orthopedic injury other than stroke were excluded from the study.

**Subject Classification**

All subjects were classified into two groups before training based on their self-selected walking speed and compared to healthy control subjects walking at matched speeds. The groups before training; slow = 0-< 0.4 m/s (compared to control subjects walking at 0.3 m/s, n= 17 subjects) and moderate = 0.4 - 0.8 m/s (compared to control subjects walking at 0.6m/s, n=13 subjects). After LT all the subjects were grouped into three speed based groups and again compared to healthy control subjects. The groups after training were defined as: slow = 0< 0.4m/s (compared to control subjects walking at 0.3 m/s, n=6 subjects), moderate = 0.4 - 0.8 m/s (compared to control subjects walking at 0.6 m/s, n= 17 subjects and fast >0.8m/s (compared to control subjects walking at 0.9 m/s, n=7 subjects). Furthermore, individuals in the moderate group were grouped into two groups a) subjects who increased their speed post-LT (slow to moderate; n=13) and moved up one group, b) participants who increased their speed post-LT, but were still grouped in the moderate speed category (n=4).

**Intervention**

Subjects post-stroke underwent 36 sessions of LT on a BWSTT system. Three sessions each week for twelve weeks were provided. Missed sessions were compensated for at the end. Each session included 20 minutes of stepping on treadmill

(TM) and 20-30 minutes of training over-ground. Rest breaks were provided as required. The total duration of training session was 60-75 minutes. Vitals were measured before each session, after every five minutes of stepping during the session and at the end of each session to assess participant's response to training and ensure safety.

Subjects wore a harness which was hooked to the overhead motorized system which provided body weight support (BWS) to facilitate stepping (Robomedica Inc, Mission Viejo, CA). The level of BWS was adjusted to promote upright standing posture while ensuring maximum loading of both the legs. In addition trainers and physical therapists provided requisite assistance at the trunk and both paretic and non-paretic leg to mimic kinematics of the normal walking pattern.

In the beginning of the training, the non-paretic leg was provided greater guidance by the leg trainer, which was reduced gradually over time. The guidance was aimed at lateral and anterior-posterior placement of the foot. Individuals post-stroke tend to rapidly place their non-paretic leg close to the midline to facilitate greater loading of the non-paretic leg, while reducing the loading of the paretic leg (as indicated by chapter 3). Therefore, the non-paretic leg trainer provides guidance to place the foot laterally to reduce the loading of the non-paretic leg, while trying to increase the loading of the paretic leg. Furthermore, the anterior-posterior guidance was provided not only to achieve adequate step length, but also to increase the non-paretic leg swing time, which thus facilitates increased paretic single leg support phase. This promotes symmetry while also increasing paretic loading. In addition the anterior-posterior guidance by the non-paretic leg trainer also ensured adequate hip flexion during the swing phase. This

would reduce the hip extensor activity in the non-paretic stance which provides compensation to weak paretic hip flexors and plantarflexors during swing. This might result in forced use of the paretic leg hip flexors and plantarflexors. Furthermore, the non-paretic leg trainers also cued the knee to extension and flexion from stance to swing respectively. Some participants tended to maintain their non-paretic knee in flexion, which may be detrimental to stability during weight bearing (see chapter 3). In the initial few sessions, assistance to the non-paretic leg was typically provided throughout the training sessions. However, once the participants developed adequate control and pattern (usually after a few sessions), the amount of assistance to the non-paretic leg was subsequently reduced.

In addition to the efforts by the leg trainers, the trunk trainer also worked in collaboration to acquire normal walking pattern. Trunk trainers controlled the weight shift, by regulating the amount (decrease it) and time of loading (see chapter 3) to the non-paretic leg. They also prevented the non-paretic hip from hyperextending during stance. In doing so they ensured appropriate movement at the pelvis. As the subjects progressed through the training, focus was on an independent and normal walking pattern. BWS was reduced gradually, treadmill speeds were increased and the assistance provided by the trainers on both legs and the trunk was reduced.

**Biomechanical Data Collection**

All subjects underwent a full-scale biomechanical testing before the onset of training, at the end of training period. Training consisted of thirty-six sessions of manually assisted body weight supported treadmill training.

Biomechanical data collection was conducted within one week before and after thirty-six sessions of locomotor training. Participants walked on a split-belt instrumented

treadmill (Techmachine, Andrezieux Boutheon, France) at their self-selected comfortable walking speed. No ankle-foot orthosis or assistive device was used during data collection. However, all participants wore a harness, mounted to the ceiling of the lab, which eliminated the risk of falling and could hold the subjects in case they lost their balance. A physical therapist was also present at all times for vigilance and providing assistance if required. Notes were made, if assistance was used and those data were not used in the final analysis. All the participants walked at their self-selected speed on the split-belt instrumented treadmill for 30 seconds (3 trials). The treadmill was started at a slower speed and then speed was gradually adjusted until the subjects reached their self-selected comfortable walking speed.

**Data Recording and Processing**

Twelve camera VICON motion-capture system (Vicon Motion Systems, Los Angeles, CA) and instrumented treadmill and telemetric EMG system (Konigsberg Instruments, Pasadena, CA) were used to capture the kinematic, kinetic and EMG data as the subjects walked on the split-belt instrumented treadmill.

Ground reaction forces were acquired from each foot separately at a sampling rate of 2000 Hz, and low-pass filtered using a fourth-order, zero-lag Butterworth filter with a 20 Hz cut-off frequency. A 13 segment musculoskeletal model was created using Visual 3D (V3D) (c-motion, Germantown, MD) processing that fits the model to marker trajectories. V3D models were used to conduct inverse dynamics analyses for calculation of intersegmental joint kinetics. The EMG signals were collected as analog signals, and were then converted to digital signals and stored on the lab computer. The analog signals are amplified and pre-filtered before being digitized and stored. The EMGs are then filtered with a 40 Hz high pass filter, demeaned, rectified and then low

pass filtered (3 Hz). Filtering was done with 4$^{th}$ order Butterworth filters in Matlab (Mathworks, Inc., Natick, MA).

**Calculations of Study Variables**

The same variables were recorded and calculated for healthy individuals and subjects with hemiparesis during walking. All the variables were calculated by averaging across all gait cycles of each trial. Since there was no statistically significant difference between the performance of the left and right side of the healthy control subjects, mean value of the two sides was used for all the analysis.

**EMG variables**

**EMG magnitude.** Integrated EMG was used to quantify the magnitude of EMG activity over the gait cycle and for different regions of the gait cycle (figure5-1). It was calculated by numerically integrating absolute EMG signals using an extended open closed formula with equally spaced abscissas with respect to time (figure 5-2).

**EMG timing.** Percentage duration of activity of a muscle in each gait cycle/region is sum of all the "on" points divided by the total number of points in the gait cycle/region. The “on” and “off” points of EMG were calculated using the K-means analysis technique[125] (figure 5-3)

**Kinetic and kinematic variables.** Total positive work done by the ankle plantarflexor in region four, hip flexors in region four and hip extensors in region one, three and four was calculated.

Kinematic parameters of leg and knee were calculated during different regions of the gait cycle.

### Statistical Analyses

Independent samples t-test and Mann Whitney U-test were performed to compare the magnitude and timing of muscle activity in the non-paretic leg pre- and post-LT to healthy control subjects walking at speeds matched to pre- and post-LT walking speeds (respectively, subject classification described above). Comparisons were made: 1) over the gait cycle, 2) in different regions of the gait cycle (subject grouping described in methods section above). Thus, for each muscle we will determine if the pre-LT activity differed from speed matched control and the post-LT muscle activity differs from its speed matched control, instead of comparing pre-LT to post-LT directly. We have chosen this comparison because we believe that it will best account for changes in speed. Pearson's and Spearman's correlations were conducted to evaluate the relationship between percentage average activation for the muscle in a specific region and the measures of motor performance (work, angles) in that region. Significance for all tests was set at alpha $< 0.05$. All statistics were run using SPSS version 17.0 (SPSS, Inc.).

## Results

The results of the analyses reveal all subjects experienced increase in walking speed post-LT (except participants walking at slow speed before and after LT); in addition to changes in the non-paretic leg performance. However the amount of change was different for different groups. Specifically, individuals that increased their speed from moderate to fast revealed the maximum change (0.40m/s), followed by subjects walking at slower pre-speeds who proposed to moderate post-speeds (0.34m/s). The people walking at moderate speeds pre and post-LT had an increase of 0.17 m/s.

Participants in the slow group before and after training exhibited a non-significant increase of only 0.02 m/s.

**Magnitude of EMG Activity in the Entire Gait Cycle**

The results of our analysis suggest that the individuals walking at moderate speed post-LT had the most compensation by the non-paretic leg, while the individuals walking at slow speed had the least compensation. Specifically, individuals walking at moderate speed post-LT had increased magnitude of activity in most muscles (all except SO for subjects from slow-moderate or moderate-moderate), while those walking at slow speeds post-LT revealed increased activity in only VM (Appendix A, Figure A-1). The individuals increasing speed from the moderate pre to fast post-LT did not show any change in the magnitude of muscle activity as compared to speed matched control subjects (Appendix A, Figure A-2).

**Magnitude EMG Activity in Different Regions of the Gait Cycle**

**Slow speed group.** Individuals walking at slow speed pre-LT presented different changes in the muscle magnitude (compared to the appropriate speed-matched control group) depending on whether they still walked at slow speed after LT or they increased their speed to moderate level (figure 5-4, Appendix A: figure A-3-A-5). Continued compensation post-LT was provided by increased MG in region one and VM in region two (each are previously defined as novel compensations) and LH in region one (conventional compensation H1 burst) in subjects walking at slow speed after LT (6 subjects).

On the other hand, individuals who subsequently walked at moderate speeds (13 subjects, figure 5-5, Appendix A: figure A-4) after LT revealed significantly more activity in the thigh muscles during the entire stance phase relative to the speed matched

controls than they did relative to the speed matched controls before LT (i.e., in 20 of 20 possible muscle-region combinations after LT and 10 of 20 before LT). They also increased TA throughout the stance phase after LT when it had been reduced in most of the stance phase before LT. Note that while there was also much increased activity in swing phase, the absolute magnitude was much lower in most muscles and so it may not be clinically significant.

**Moderate speed group.** Individuals walking at moderate speed at both times showed fewer differences from the same speed matched control data after LT with respect to the thigh muscles (there were 8 of 20 muscle-region combinations increased after LT as opposed to 16 of 20 before LT) Appendix A: figure A-3 to A-7. As to the specific compensations we outlined in chapter 4: A2 in region 4 is present at both pre and post-LT, H1 in regions 6, 1 or 2 is present at both pre and post-LT; the novel MG in region 1 is only present at pre LT; the novel VM activity in regions 2 and 3 is present at both times; and the novel hamstrings activity in regions 3 and 4 is only present at pre LT (Appendix A, figure A-7).

Furthermore, individuals attaining faster speed post-LT (7 subjects) (figure 5-7, Appendix A, figure A-8) had significantly greater magnitude of activity in the upper leg muscles as compared to speed matched control subjects. The compensatory mechanisms involved were: hamstrings in region 1 to increase H1present both before and after-LT. Among the novel compensations MG was present in region one, VM in region two and three (figure 5-6) (mid-stance) and hamstrings in region three and four both before and after LT.

**Percentage Duration of Muscle Activity in the Entire Gait Cycle** (Appendix B, table B-1)

**Slow group.** The individuals walking at slow speed pre-LT exhibited normalization of percentage duration of activity post-training in most muscles. Individuals continuing to walk at slower speed had increased compensation from upper and lower leg muscles ( SO, VM, RF), while the group of participants that increased walking speed from slow to moderate speed had an increase in compensation only from the plantarflexor muscles (MG, SO).

**Moderate speed group.** Participants walking at moderate speed before-LT exhibited increased percentage duration of activity primarily in the power producing muscles. In individuals walking at moderate speed after LT increased compensation was provided primarily by the two plantarflexor muscles. On the other hand the individuals walking at faster speed post-LT revealed significantly increased percentage duration of activity in MG, SO, LH and MH.

**Percentage Duration of Muscle Activity in Different Regions of the Gait Cycle**

**Slow speed group.** The percentage duration of muscle activity during swing phase post-LT was similar to control subjects walking at matched speeds, while differences were present in muscle activity during the stance phase.(figure 5-4) For the specific compensations by the non-paretic leg in individuals walking at slow speed after LT: increased A2 by increased MG and SO was present both before and after LT (only MG). Among the novel compensatory mechanisms of the non-paretic leg: increased activity of VM in region three (pre-LT) or two (post-LT) (mid-stance) and increased plantarflexor activity in region one (only MG post-LT) was present both pre and post-LT,

while increased hamstrings activity in the late stance phase was present only before training (Appendix B, Table B-4).

On the other hand, the individuals walking at moderate speed after LT (slow pre) had significantly increased muscle activity in several muscles during stance phase (figure 5-5, Appendix B, Table B-5). For the specific compensations: A2 was present both pre (only SO) and post-LT, while increased H1 due to hamstrings activity was present in region one only post-LT. Among the novel compensatory mechanisms increased plantarflexor activity was present pre and post-LT (only MG post) and so was the increased VM activity in region two and three. However, increased hamstrings activity in region four was present only pre-training.

**Moderate speed group.** There was significant normalization of temporal patterning of other muscles in various regions of the gait cycle in individuals continuing to walk at moderate speed after-LT (figure 5-6, Appendix B- Table B-6). The trend of specific compensatory mechanisms in individuals walking at moderate speed post-LT was: increased A2 due to increased MG was present pre and post-LT and increased hamstrings in region 1 was also present both before and after LT (only LH after LT). However, among the novel compensatory mechanisms, all the three were present before LT, while none of them were revealed after LT.

On the other hand individuals walking at faster speed after LT had normalized temporal patterning of muscles in region three to six of the gait cycle (figure 5-7, Appendix B-Table B-7). The specific compensations as outlined in chapter 4: increased hamstrings activity in region one was [resent both before and after training, while increased activity of the plantarflexors in region four was present only pre-LT. with

respect to the novel compensatory mechanisms of the non-paretic leg, SO activity was significantly greater than speed matched control subjects both pre and post-LT and also increased activity of VM muscle in region two (region three only pre-LT) was present before and after training. Additionally, the increased activity of LH was present in region three only before LT.

**Correlational Analysis**

**H1 and LH & MH in region 1 of the gait cycle.** There was no significant correlation between the magnitude of hamstring activity and positive work done by the hip extensor in the first region of the gait cycle of the slow speed group both before and after LT. On the other hand, individuals in the moderate speed group had significant positive correlation between the MH and H1 following LT, ($r= 0.515$, $p= 0.04$) (not LH).

**SO and MG muscle activity and A2 in region 4 of the gait cycle.** Although the magnitude of SO and MG in the fourth region of the gait cycle was significantly greater in the slow speed groups before LT, there was no significant correlation between the muscle activity and the positive work done at the ankle in that region of the gait cycle both before and after LT.

**TA muscle activity and A2 in region 4 of the gait cycle.** There was no significant correlation between the magnitude and duration of the TA activity in slow speed group before and after LT.

**RF muscle activity and peak knee flexion angle in region 4 and 5 of the gait cycle.** The percentage duration of the RF activity in the fourth region of the gait cycle was significantly negatively correlated with the peak knee flexion in individuals in the fast speed group ($r=-0.792$, $p= 0.002$). No significant correlations were present after LT.

**MH muscle activity and positive hip extensor work.** In the slow speed group a significant positive correlation (r= 0.528, p= 0.026) was present before LT, however after LT there was a significant negative correlation between the two (r= -0.654, p=0.015).

**MG muscle activity and leg angle in first region.** There was no significant correlation between the magnitude and duration of the MG activity in slow and moderate speed group before and after LT.

## Discussion

The results of our analysis revealed mixed response to LT post-stroke. The muscle activity of non-paretic leg was significantly normalized in some individuals walking post-LT. Nevertheless, there were some significant differences in the muscle activity in the non-paretic leg post-LT when compared to healthy control subjects walking at matched speeds. Specifically, participants that walked at slower speeds before LT, but acquired moderate walking speed post-LT exhibited the most differences. This suggests that, in addition to the changes that were occurring in the paretic leg, the non-paretic leg also appears to have contributed to the increased walking speed through increases in one or more compensatory mechanisms. These compensatory mechanisms may be just increase in the normally occurring positive phases of work generation in the gait cycle (i.e. LH and MH for H1 in region one, SO and MG for A2 in region four). They could also be novel compensatory mechanisms developed by the non-paretic leg in an attempt to compensate for the poorly coordinated paretic leg (i.e. increased MG and SO in region one, or VM in region two and three or increased activity in hamstrings in region three and four of the gait cycle.

With respect to limitations, it is important to note, that because all of the muscles as well as the kinetic and kinematic measures influence walking and are not independent, we did not correct for multiple comparisons in our study. Furthermore, the amplitude of EMG detected using surface electrodes is sensitive to many intrinsic (thickness of subcutaneous fat, distribution of motor units, conduction velocities and detection systems used), and extrinsic factors, i.e. the factors which can be influenced by the experimenter (orientation, location and area and shape of the electrode and distance between them [152]), which renders it highly variable. The reliability of the magnitude of the EMG in the dynamic contraction is also influenced by length and force change due to stationary shift in relative position of the electrode and changes in tissue conductivities.[153] Furthermore, the changes in the joint angles can cause the recording electrodes to shift relative to active muscle fiber and the change in the muscle fiber direction to alter tissue conductivity.[153] Thus, all comparisons based on the magnitude measures must be cautiously evaluated. However, the amplitude of the surface EMG is related to the net motor unit activity, i.e. recruitment and discharge rates of motor units[151] and forms an important component of the EMG analysis. Care was taken while recording and analyzing EMG activity to minimize errors and caution is warranted with the interpretation from strictly amplitude measures.

**Muscle Activity over the Entire Gait Cycle**

Increased magnitude of muscle activity over the entire gait cycle was related to the severity of deficits and walking speed attained. Analysis of integrated EMG activity over the gait cycle in individuals with severe speed deficits was associated with significantly greater magnitude in more number of muscles (slow – moderate: seven muscles as opposed to five muscles pre-LT) as compared to individuals with moderate deficits

(moderate – fast: same four muscles both pre and post-LT). Although integrated EMG activity provides important information regarding the firing or recruitment of muscle fibers during the gait cycle, it is important to take under consideration the limitations associated with the processing of EMG amplitude (discussed above).

Furthermore, the temporal patterning in nearly all individuals (walking at all the different speeds) after LT revealed fewer differences from healthy control subjects walking at the matched speeds than were evident before LT. These results are contrary to the pre-existing evidence[126, 190] which suggests that the temporal patterning of the muscles is unaltered following rehabilitation. This difference could be attributed to the task specific nature of the gait training provided in our study (i.e. BWSTT), as oppose to pedaling aerobic training and Neuro-developmental therapy in the other studies mentioned. However, increased duration of activity was present in some muscles even after LT, suggesting some of the compensatory mechanisms used by the non-paretic leg continued to be used.

To obtain more details about the compensation mechanism of the non-paretic leg after a task-specific BWSTT and investigate the changes in its performance relative to healthy control subjects walking at similar speeds, we investigated the changes in muscle activity in different regions of the gait cycle.

**Muscle Activity in Different Regions of the Gait Cycle**

Ability to walk faster accompanied by normal kinematic and kinetic patterns is the important aims of the contemporary task specific therapies. Therefore it is important to reduce the compensation by the non-paretic leg, while targeting to increase the contribution of the paretic leg, as was done in this study. However, some of the compensatory mechanisms present before LT continued after LT, most notably in

people who exhibited increase in speed from slow to moderate. Compensation was provided by either increasing the normally existing phases of positive work generation over the gait cycle or increased contribution of the novel compensatory mechanisms developed in the non-paretic leg post-stroke.

The first compensatory mechanism was the increased positive work done by hip extensors (hamstrings) in stance phase of the gait cycle in both slow and moderate speed groups before and after LT (except in individuals who walked at slow speed before and after training). It may be a technique used to facilitate propulsion by promoting forward acceleration of the trunk in order to attain faster speeds. This is also supported by a significant positive correlation between the positive work done by the hip extensors H1 and magnitude of hamstring (MH) activity during this phase of the gait cycle in moderate speed group.

The second, compensatory mechanism was increases from the ankle plantarflexors in the fourth region of the gait cycle in individuals walking at slower speeds pre- and post- LT. This suggests that the individuals walking at slower speeds may attain faster speed by increased compensation from the ankle plantarflexors. This can be inferred from the increased magnitude (only MG) and duration (except MG in slow group) of ankle plantarflexors (MG and SO) present in the fourth region of the gait cycle before LT in individuals walking at slow and moderate speed as compared to healthy control subjects walking at matched speeds respectively.

Some novel mechanisms of compensation continued to exist even after LT. First the additional activity of the hamstrings in the late stance phase or region three and four of the gait cycle in participants that progressed from slow pre-LT- moderate post-LT)

was present both before and after LT. This extra burst of hamstrings (also reported by other researchers[126],[69]) increases extension of the non-paretic hip, which thus facilitates the swing of the paretic leg, thereby compensating for the poor activity of the paretic plantarflexors and hip flexor muscles.[126] Alternatively, the increased activity of the hamstring muscles in the late stance phase may hold head, arm and trunk and prevent postural lean[21] in subjects tending to walk faster. This phenomenon is further supported by a significant positive correlation between the positive hip extensor work and the MH activity during the third region of the gait cycle both before and after the training.

Secondly, the increased activity of VM in the mid-stance phase of the non-paretic leg has been indicated to facilitate propulsion of the swinging paretic leg[181] and was present in almost all groups before and after LT(specifically individuals progressing to next speed groups). The increased VM firing in the stance phase is a novel non-paretic leg adaptation aimed to facilitate increased propulsion in the paretic leg to achieve faster speed.

Third, all the individuals before and after (except moderate speed participants walking at moderate speed even after LT) LT presented an important compensatory mechanism by the ankle plantarflexors in the first region of the gait cycle. It is a novel compensation mechanism to produce increased propulsion that might possibly be facilitated by the altered asymmetric kinematics. The increased plantarflexors activity in this region of the gait cycle may facilitate propulsion if the non-paretic leg is not placed ahead of the body center of mass (as happens in a step –to-gait[160]).

**Implications of Analysis.** Individuals grouped in the slow speed group revealed significantly increased magnitude and duration of most recorded muscles while they

walked at moderate speed after LT. This suggests that the increase in walking speed could be due to increased contribution by the compensatory mechanisms of the non-paretic leg. On the other hand, individuals walking at moderate speed before LT, but fast speed post-LT revealed little difference in magnitude of muscle activity (although note limitations of magnitude discussed above), while the temporal pattern of muscle activity was significantly improved. And in the individuals who presented with a speed change without a change in speed group post-LT (i.e. slow- slow and moderate-moderate) exhibited significantly reduced contributions of the non-paretic leg during walking as compared to healthy control subjects. This suggests that, although changes in speed groups may not have occurred, compensation by the non-paretic leg may have reduced and the patterns of walking may have improved in these participants. This might be associated with increased and/or improved motor activity of the paretic leg.

The results of this study suggest that the LT using BWSTT is an effective technique of gait rehabilitation post-stroke. Despite the limitations of comparison of EMG amplitude between subjects and between time points in the same subjects in consideration, we believe the increased magnitude of most muscles in individuals walking from slow to moderate speed suggests additional involvement of compensatory mechanisms. We suggest that emphasizing reduction of the compensatory mechanisms of the non-paretic leg would force increased use of the paretic leg (in addition to the conventional emphasis of increasing the paretic leg use). This dual pronged approach to modulate the performance of individuals walking after stroke might produce more effective response to therapy.

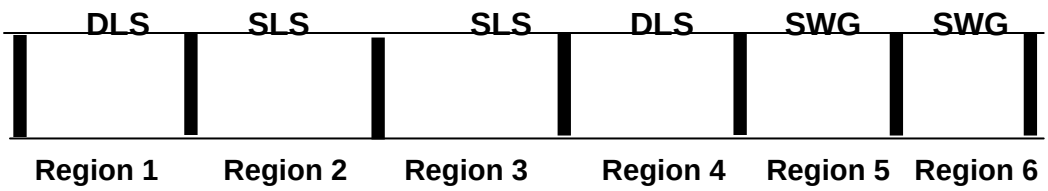


Figure 5-1. Represents the different regions of the gait cycle. At the top are the marked the different physiological phases of the gait cycle (DLS= double limb support, SLS= single leg support, SWG= swing phase) and at the bottom are the corresponding regions used in this study.

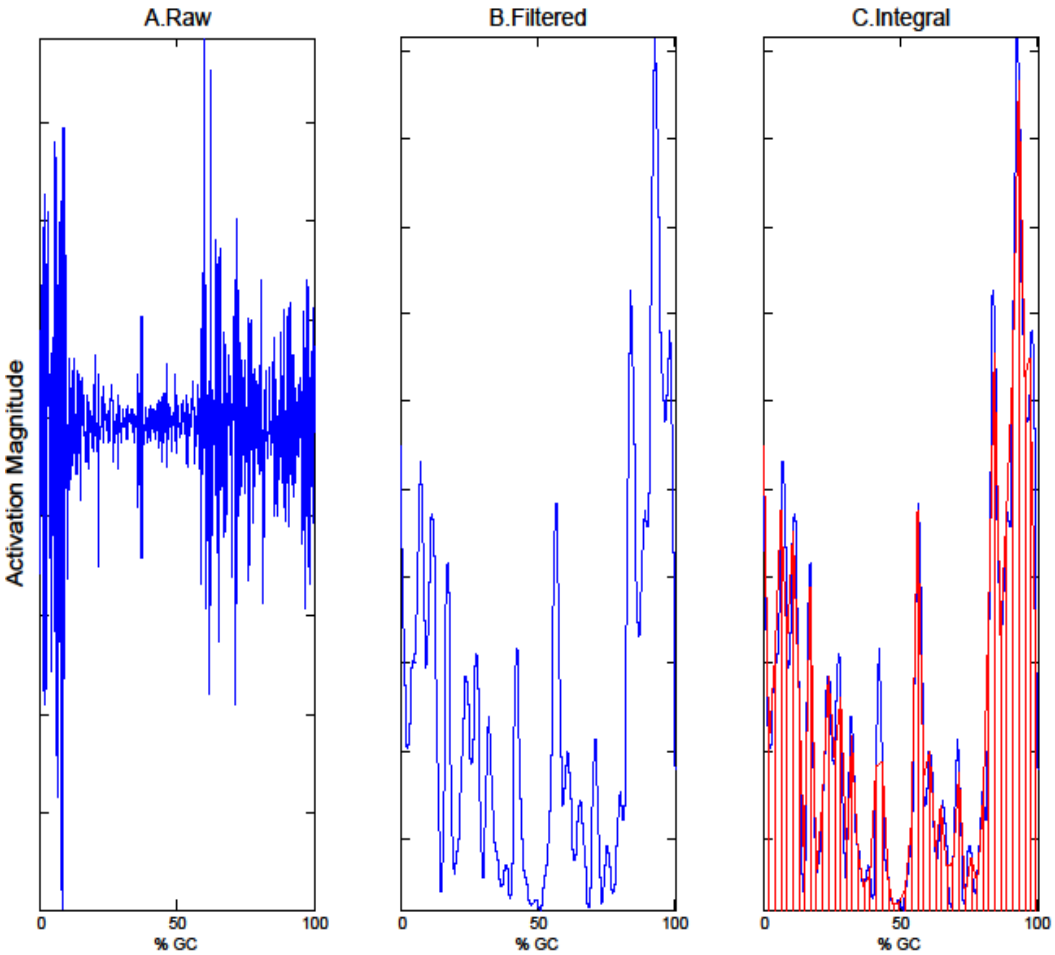


Figure 5-2. Represents the calculation of integrated EMG activity. First figure represents the raw EMG signal, second panel represents the rectified and filtered EMG signal and the third figure represents integration rule with equally spaced abscissas (red lines), with respect to time in the gait cycle.

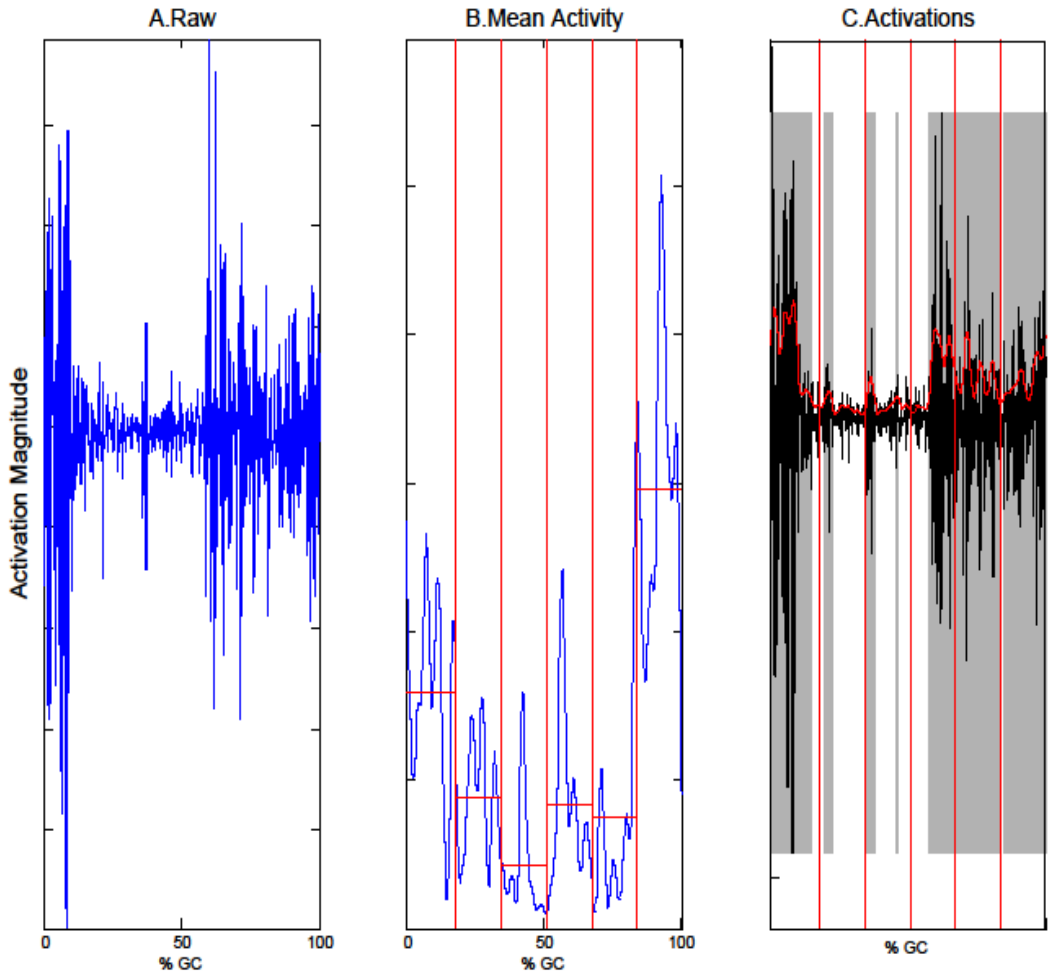


Figure 5-3. Represents the steps of k-means analysis. First figure represents the raw EMG magnitude, second figure shows the filtered rectified EMG (linear envelope) and the third figure represents the original raw EMG signal with the detected periods of activity indicated in gray shaded areas

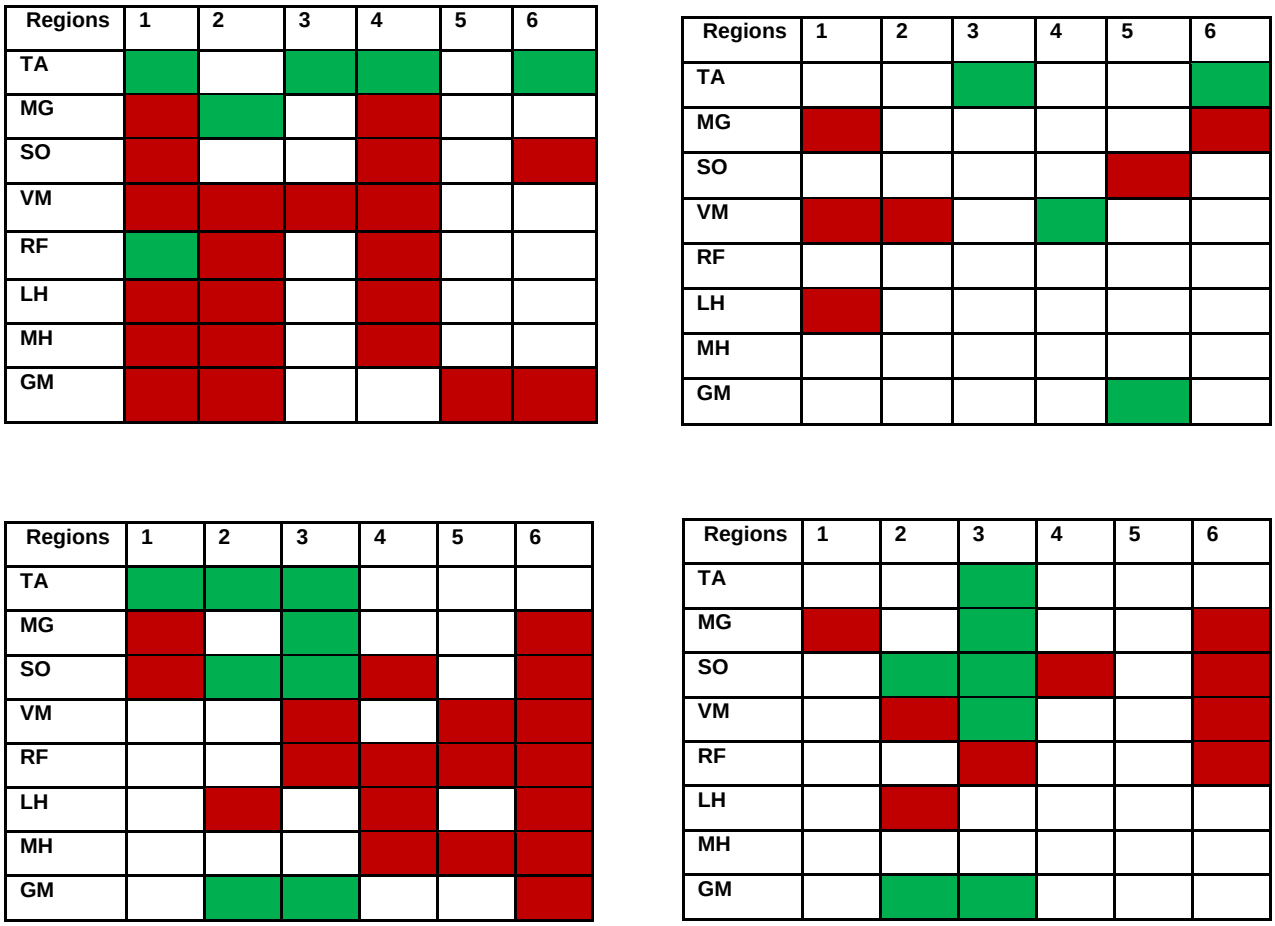


Figure 5-4. Represents difference in muscle activity in individuals walking at slow speed (0-<0.4m/s) before and after locomotor training as compared to healthy control subjects walking at matched speeds. Top panel shows the differences in the integrated EMG activity pre (left) and post training (right) when compared to the speed matched controls. Bottom panel shows the percentage duration of activity pre (left) and post-training (right). Red squares represent increased activity in the non-paretic leg and green represent reduced activity in the non-paretic leg as compared to controls. More details in appendix A and B

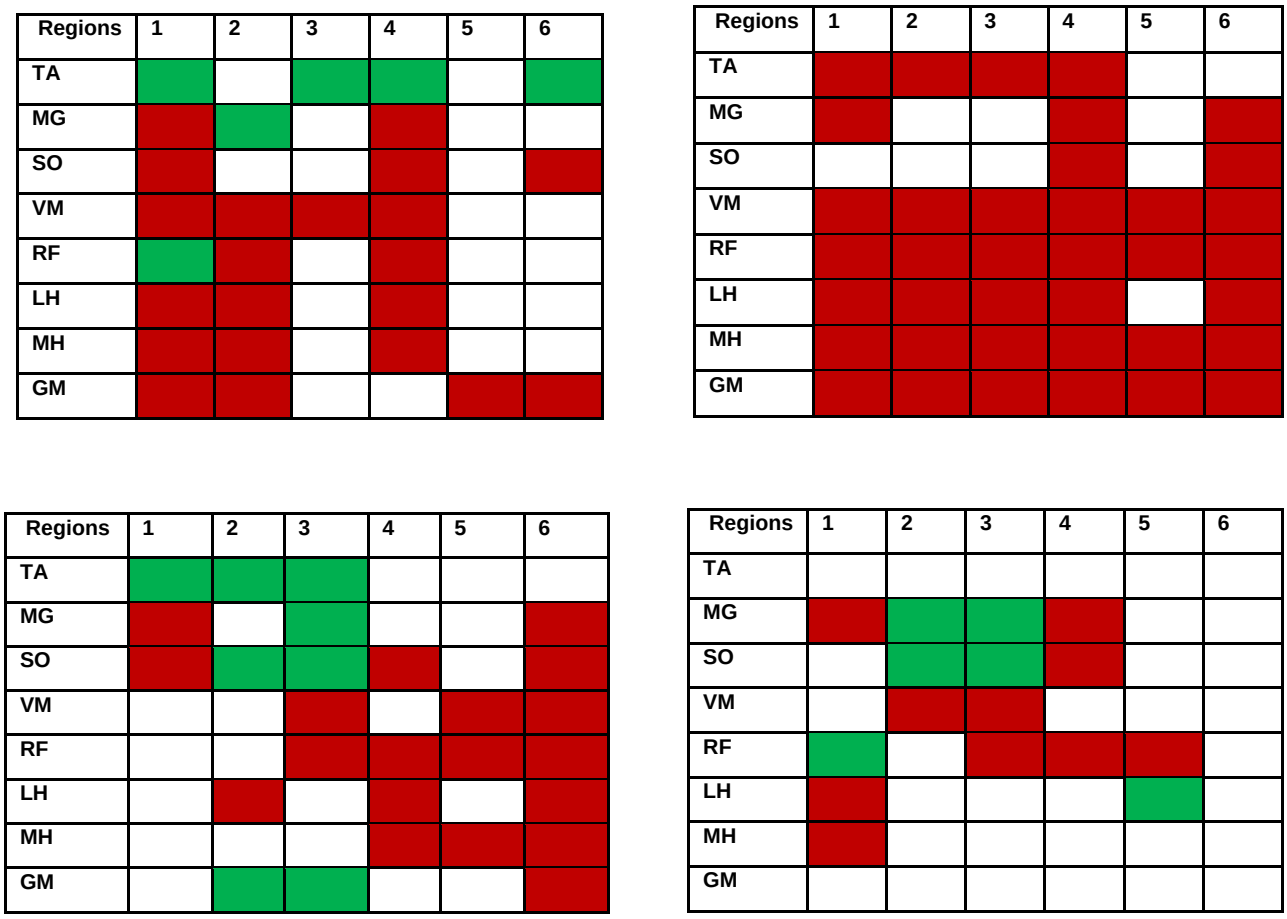


Figure 5-5. Represents difference in muscle activity in individuals walking at slow speed (0-<0.4m/s) before and moderate speed (0.4- 0.8m/s) after locomotor training as compared to healthy control subjects walking at matched speeds. Top panel shows the differences in the integrated EMG activity pre (left) and post training (right) when compared to the speed matched controls. Bottom panel shows the percentage duration of activity pre (left) and post-training (right). Red squares represent increased activity in the non-paretic leg and green represent reduced activity in the non-paretic leg as compared to controls. More details in appendix A and B.

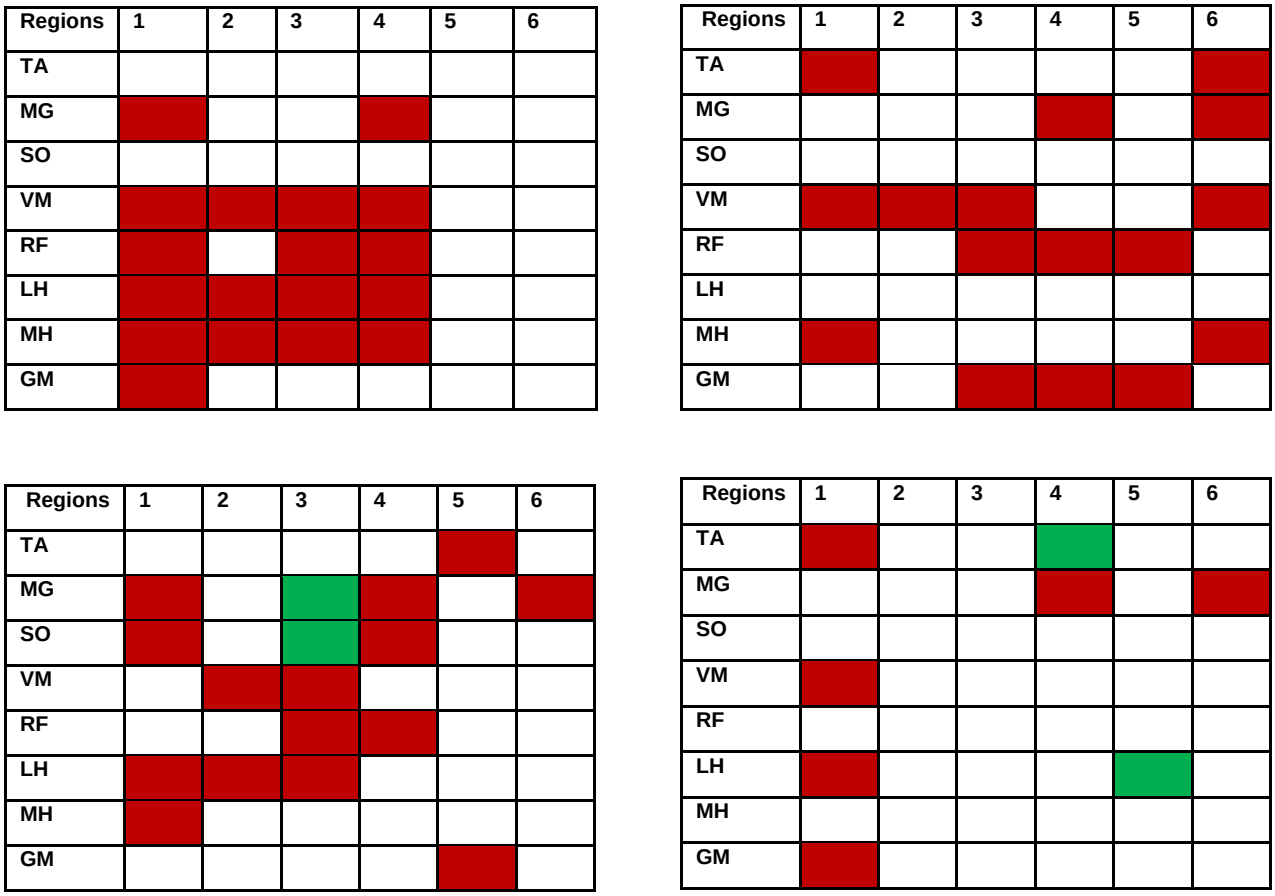


Figure 5-6. Represents difference in muscle activity in individuals walking at moderate speed (0.4- 0.8m/s) before and after locomotor training as compared to healthy control subjects walking at matched speeds. Top panel shows the differences in the integrated EMG activity pre (left) and post training (right) when compared to the speed matched controls. Bottom panel shows the percentage duration of activity pre (left) and post-training (right). Red squares represent increased activity in the non-paretic leg and green represent reduced activity in the non-paretic leg as compared to controls. More details in appendix A and B.

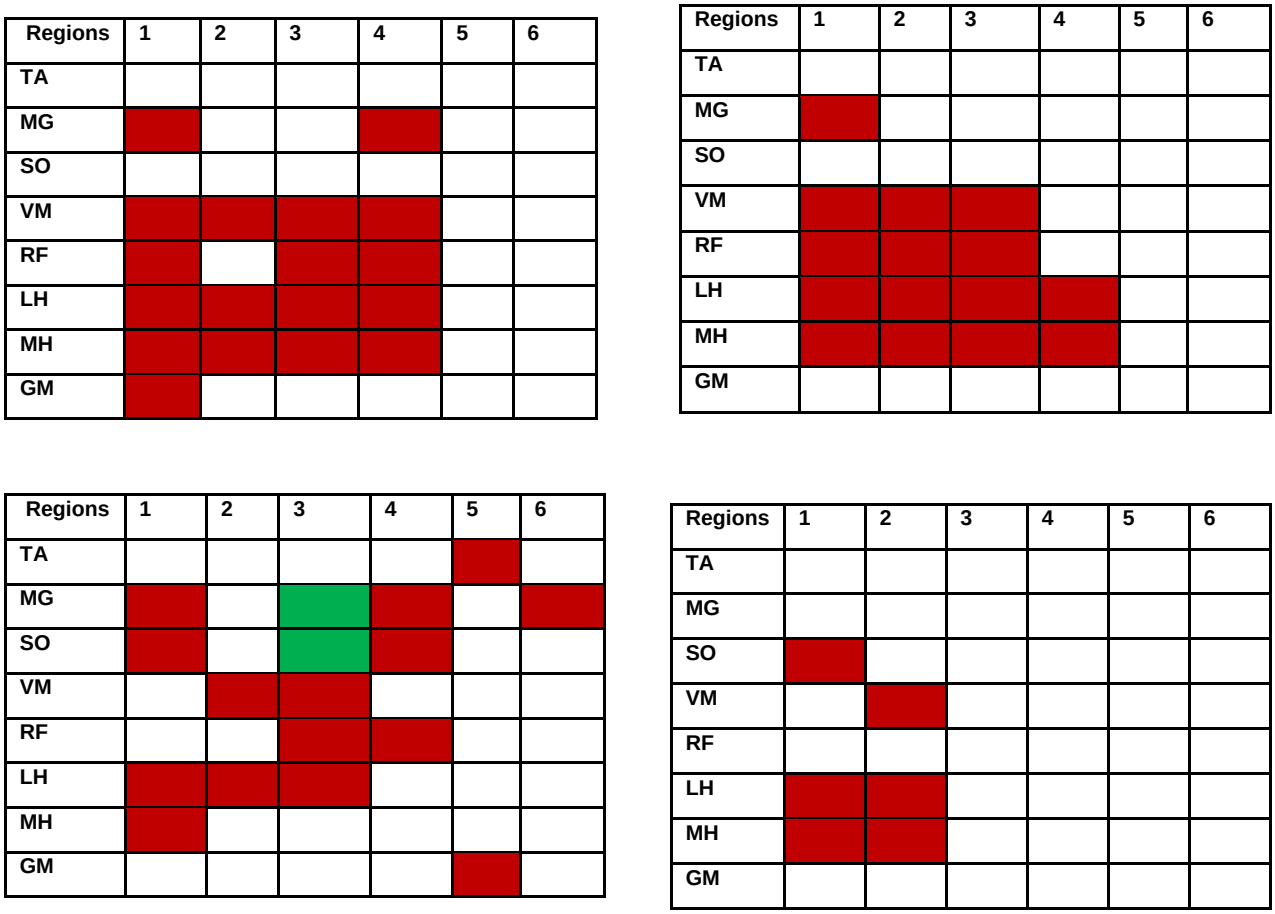


Figure 5-7. Represents difference in muscle activity in individuals walking at moderate speed (0.4- 0.8m/s) before and fast speed (> 0.8m/s) after LT as compared to healthy control subjects walking at matched speeds. Top panel shows the differences in the integrated EMG activity pre (left) and post training (right) when compared to the speed matched controls. Bottom panel shows the percentage duration of activity pre (left) and post-training (right). Red squares represent increased activity in the non-paretic leg and green represent reduced activity in the non-paretic leg as compared to controls. More details in appendix A and B.

# CHAPTER 6
# CONCLUSION

Hemiparetic gait is characterized by asymmetry in motor output of the paretic and the non-paretic leg. Usually, the non-paretic leg is perceived to compensate for the weakness and discoordination of the paretic leg. Therefore, the non-paretic leg has traditionally been considered unaffected, even to the point that it has sometimes been used as a standard to gauge the performance of the paretic leg. Some studies have documented significant contribution of the non-paretic leg output to walking performance of individuals post-stroke[12, 13, 59] while other studies have highlighted ( in pedaling literature[11, 89]) that the non-paretic leg exerts a strong negative influence on the paretic leg performance. We suggest that evaluating the non-paretic leg mechanisms should be important in order to identify its unique compensatory patterns. Furthermore, allowing normal performance of the non-paretic leg, while trying to promote proper paretic leg movement is not likely optimal for restoring the pre-stroke biomechanical pattern of walking.

While asymmetry in limb loading and unloading has been well documented in static and quasi-static tasks[9,7,8] there is limited information available related to limb loading and unloading during steady state walking. Therefore, in the first study of this dissertation, we quantified magnitude of limb loading and unloading during the two double limb support periods of the gait cycle. We also evaluated its timing, pattern and relation to medial-lateral leg angle and knee angle. Our results showed that the limb loading and unloading was asymmetric between the paretic and the non-paretic leg, with the non-paretic leg sustaining greater load and for longer duration (as revealed by cross-over time (the time to switch the predominance of weight from the leg in second

double support to the leg in first double support). Our analysis revealed that the loading and unloading asymmetry related significantly with the medial-lateral leg angle and knee angle indicating the biomechanical compensations employed to maintain the steady state walking despite asymmetry in loading and unloading. Our findings also showed the variability in the pattern of loading and unloading which provides greater insight into biomechanical adjustments made by individuals post-stroke. Asymmetry in limb loading and unloading results in structural [55, 56] and biomechanical changes[57, 58] in the non-paretic leg thus making it of importance to address clinically.

In the second study of this dissertation we exclusively focused on the biomechanical compensation and impairment mechanisms of the non-paretic leg. The non-paretic leg is often reported to be compensating for the poorly coordinated paretic leg, without any detail about the mechanisms of these compensatory patterns. Understanding of the compensatory mechanisms provides greater insight into motor control of hemiparetic gait (specifically the non-paretic leg) and therefore might guide in developing more effective rehabilitation techniques. The results of our study show significant difference in muscle activity between the non-paretic leg and the healthy control subjects walking at the matched speeds, associated with significant correlations with simultaneous measures of motor performance. Specifically, some of these compensations are simply increased output of the important sources of work done in the normal gait as indicated by significant positive correlations between the muscle activity and work done at a joint. For example, A2 was associated with SO and MG in the fourth region of the gait cycle or and H1 was associated with MH and LH were related in the first region of the gait cycle. Other compensations, however, were novel adaptations of

the non-paretic leg to compensate for the paretic leg and achieve steady walking state. For example, hamstrings activity in late stance to generate propulsion, early stance plantarflexor activity that generates propulsion because the non-paretic leg is not placed as far in front of the body, and mid to late stance activity in the knee extensors that facilitates paretic leg swing.

Information obtained from this study emphasizes that stroke is not a unilateral motor control problem as often suggested, and there is a bilateral involvement. This indicates that gait related rehabilitation post-stroke should focus not just on the paretic leg but also on the non-paretic leg.

Thus, in the third study of this dissertation we evaluated the response of the non-paretic leg to a task-specific locomotor training, i.e. body weight supported treadmill training. Body weight supported training provides a permissive environment in that it affords individuals a walking experience that more closely approximates the actual sensorimotor pattern of walking when compared with walking overground.[97] Furthermore, manual assistance is provided by trainers on both the non-paretic and the paretic leg and the trunk to maintain kinematically appropriate pattern of the non-paretic leg.

The results of our study reveal that body weight supported treadmill training facilitates increased walking speed with associated changes in the non-paretic leg muscle activity. For example individuals with severe gait deficits acquire greater speeds by increased compensation by the non-paretic leg (both conventional and novel adaptation mechanisms), while individuals who did not increase to moderate (from slow) or fast (from moderate) walking group reveal reduced compensation by the non-paretic

leg. This improved non-paretic leg muscle activity is also associated with better performance of the paretic leg muscles. Therefore we can state that improved walking performance in the individual post-stroke appears to have contribution from both the paretic and the non-paretic leg. Thus, in order to maximize the benefits of the body weight supported treadmill training, it is essential to train both the paretic and the non-paretic legs.

We suggest that focusing on both the non-paretic leg and the paretic leg provides a two pronged approach to the rehabilitation that is better than the conventional unilateral approach focused on the paretic leg. It can be supported firstly by greater increases in walking speed in our study participants as opposed to other studies[111, 119, 191] evaluating the benefits of the body weight supported treadmill training ( we had a trainer for both the paretic and the non-paretic leg, while most studies in literature only had a trainer only on the paretic leg). Secondly, including the non-paretic leg in rehabilitation results in a normalized sensorimotor state for the non-paretic leg, which possibly provides facilitatory input to the paretic leg (based on extrapolation of results from pedaling studies[11, 89]). Therefore, we suggest that it is important to emphasize that allowing altered performance of the non-paretic leg, while trying to promote proper paretic leg movement, is likely not optimal for developing coordinated walking patterns.

## Future Directions

The aim of this dissertation was to evaluate the non-paretic leg performance in hemiparetic walking in relation to loading asymmetries, compensatory mechanisms and responsiveness to locomotor training. Through the three studies performed as a part of this dissertation we were successful in providing detailed and interesting insights about the non-paretic leg performance, thus, filling in the previously existing gap in our

knowledge. This however leads us to some more valuable questions related to the motor control of hemiparetic gait, which still need to be answered.

The chapters 3 and 4 of this dissertation elucidate the altered performance of the non-paretic leg. Extrapolating the results from the pedaling studies in stroke[11, 89] and walking studies post-SCI[148] we believe that the performance of the non-paretic leg is likely to influence the output of the paretic leg. Therefore, future studies should be aimed at designing experiments to study the performance of each leg independently while biomechanically controlling the performance of the other leg, in a task of walking (or similar to walking). These studies should also focus to investigate the influence of performance of one leg on the performance of the other (i.e. paretic on non-paretic and non-paretic on paretic).

In the fifth chapter of this dissertation we established the benefits of including the non-paretic leg in the rehabilitation protocol (as compared to other studies which did not include the non-paretic leg). Future studies could evaluate the effect of locomotor training in two groups of individuals, where one group is provided guidance on both the legs, while the other groups receives guidance on only the paretic leg. Also it would be interesting to investigate the effect of locomotor training with augmentative therapy (e.g. functional electrical stimulation) on the paretic leg and only manual guidance on the non-paretic leg compared to only manual guidance on both the legs. In summary, it is important to train both the legs and maximize the output of the paretic leg, while trying to minimize the compensation by the non-paretic leg.

## Summary

The findings of this dissertation further illuminate the asymmetrical nature of the hemiparetic gait. Asymmetry implies altered output of both the paretic and the non-

paretic leg. Altered output of the non-paretic leg presents as either impairment or compensation. The compensation of the non-paretic leg might be increased during the conventional phases of the positive work in the gait cycle, or might be novel adaptive patterns. These compensatory patterns can to some degree be normalized following a task-specific rehabilitation technique.

APPENDIX A
MAGNITUDE OF INTEGRATED EMG ACTIVITY OVER THE ENTIRE GAIT CYCLE

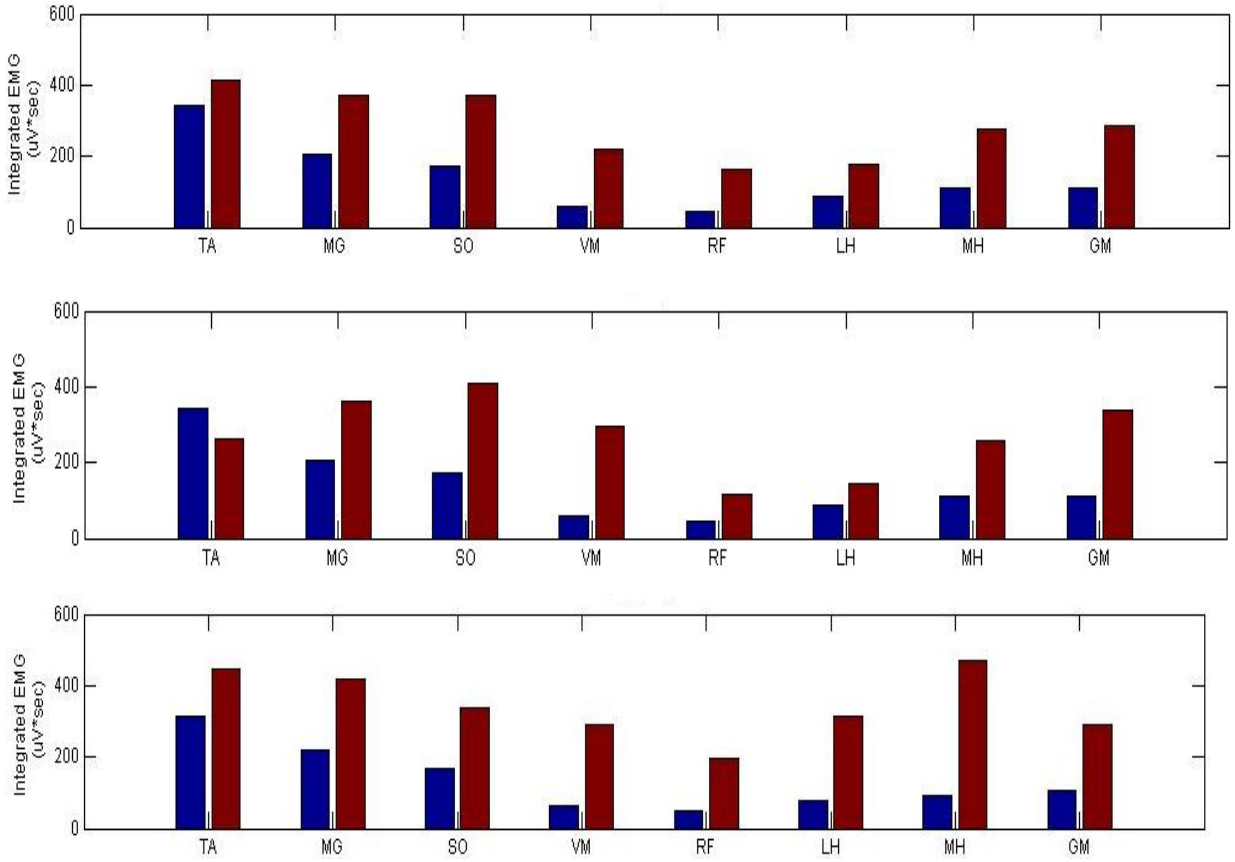


Figure A-1. Integrated EMG activity in individuals walking at slow speed pre-LT. Top plot represents individuals in slow-speed group pre-LT, middle bar plots represent individuals walking at slow speed post-LT and bottom represents individuals progressing to moderate speed post-LT. Y-axis represents integrated EMG in µVs and X-axis represents various muscles recorded. TA=tibialis anterior, MG= medial gastrocnemius, SO= soleus, VM= vastus medialis, LH= lateral hamstrings, MH= medial hamstrings and GM= gluteus medius.

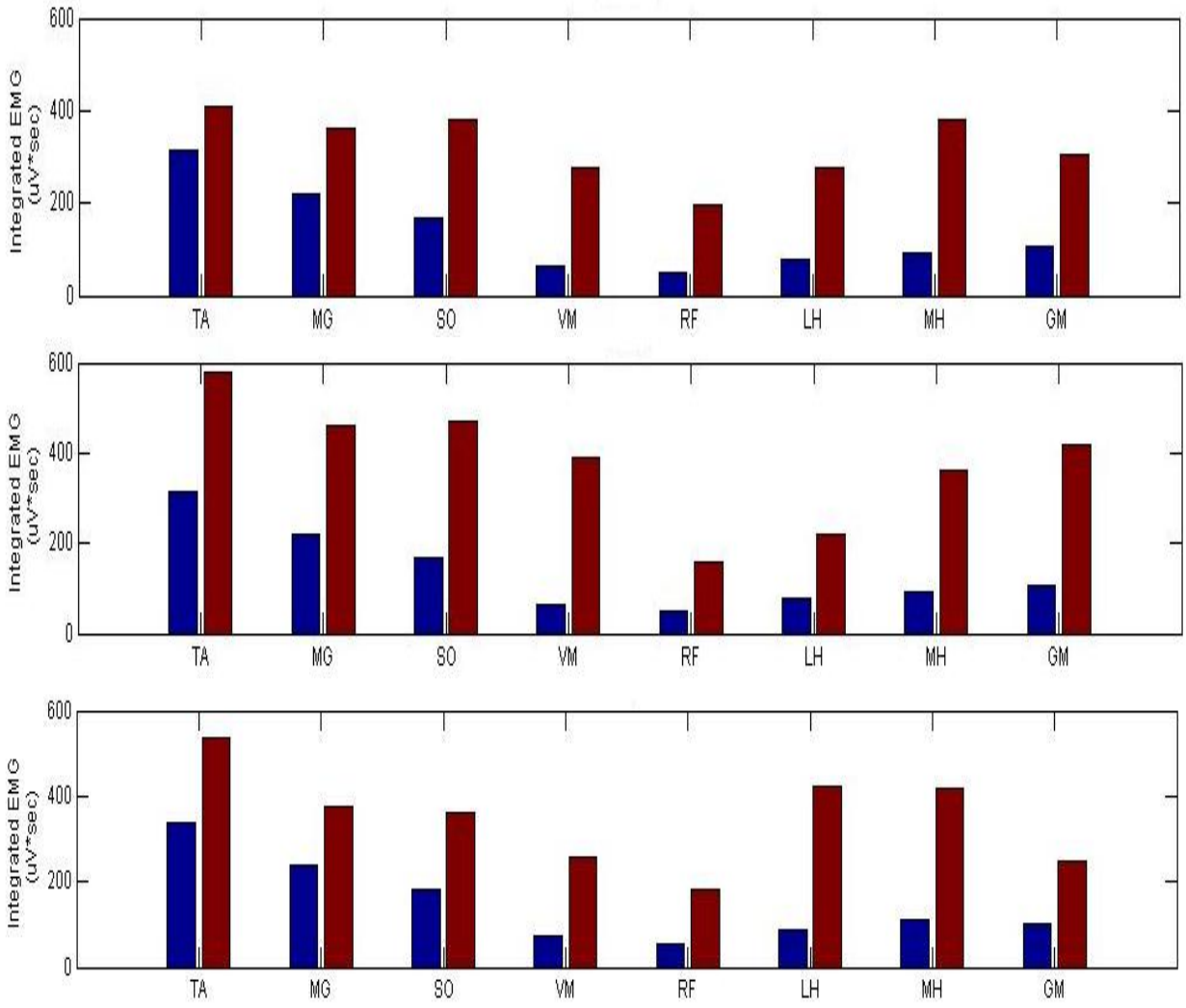


Figure A-2. Integrated EMG activity in individuals walking at moderate speed pre-LT. Top plot represents integrated EMG in individuals in moderate-speed group pre-LT, middle represents individuals walking at moderate speed post-LT and bottom represents individuals progressing to fast speed post-LT. Y-axis represents integrated EMG in µVs and X-axis represents various muscles recorded. TA=tibialis anterior, MG= medial gastrocnemius, SO= soleus, VM= vastus medialis, LH= lateral hamstrings, MH= medial hamstrings and GM= gluteus medius.

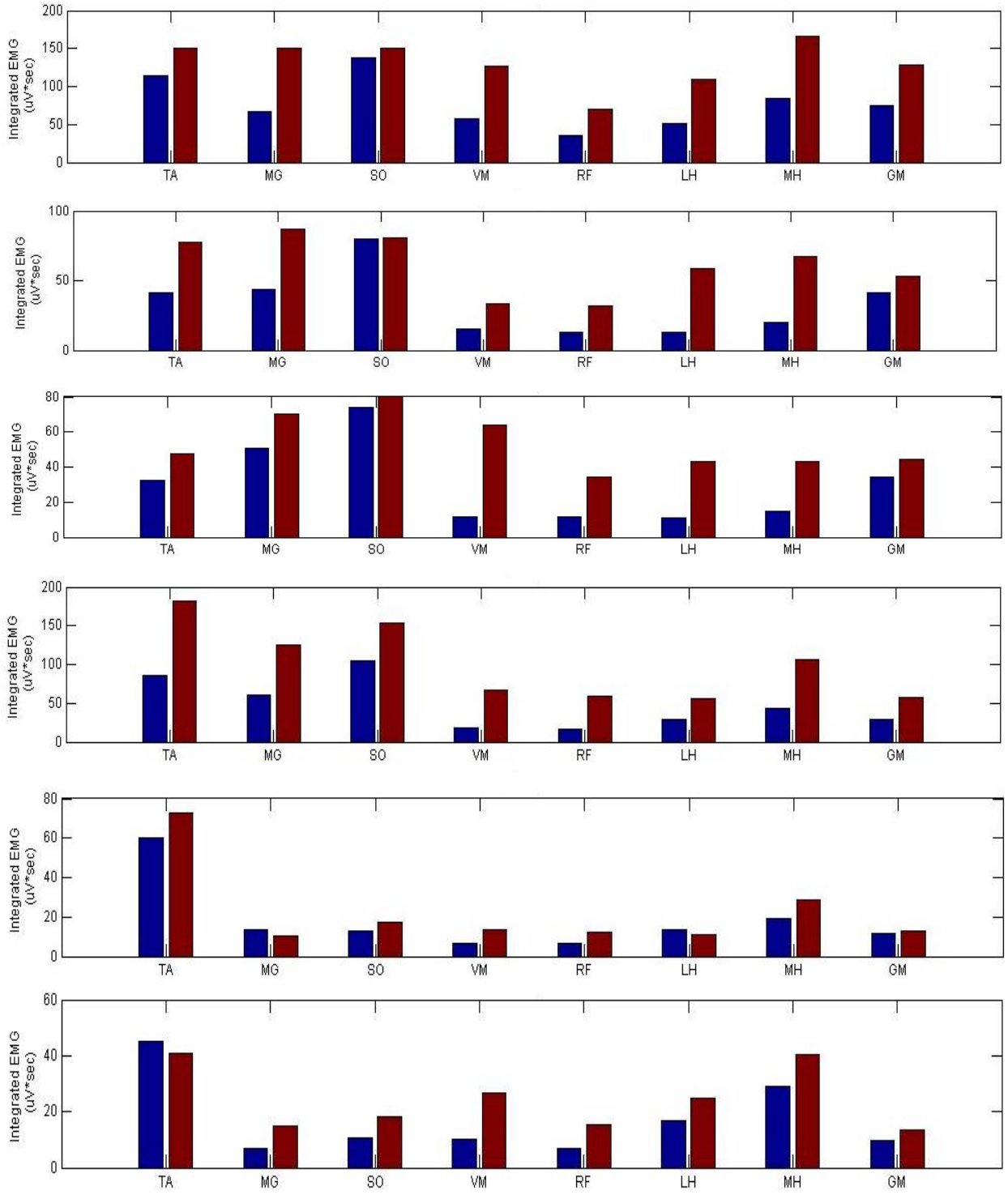


Figure A-3. Represents integrated EMG activity in different regions of the gait cycle in individuals walking at slow speed pre-LT. Y-axis represents integrated EMG in µVs and X-axis represents various muscles recorded. TA=tibialis anterior, MG= medial gastrocnemius, SO= soleus, VM= vastus medialis, LH= lateral hamstrings, MH= medial hamstrings and GM= gluteus medius.

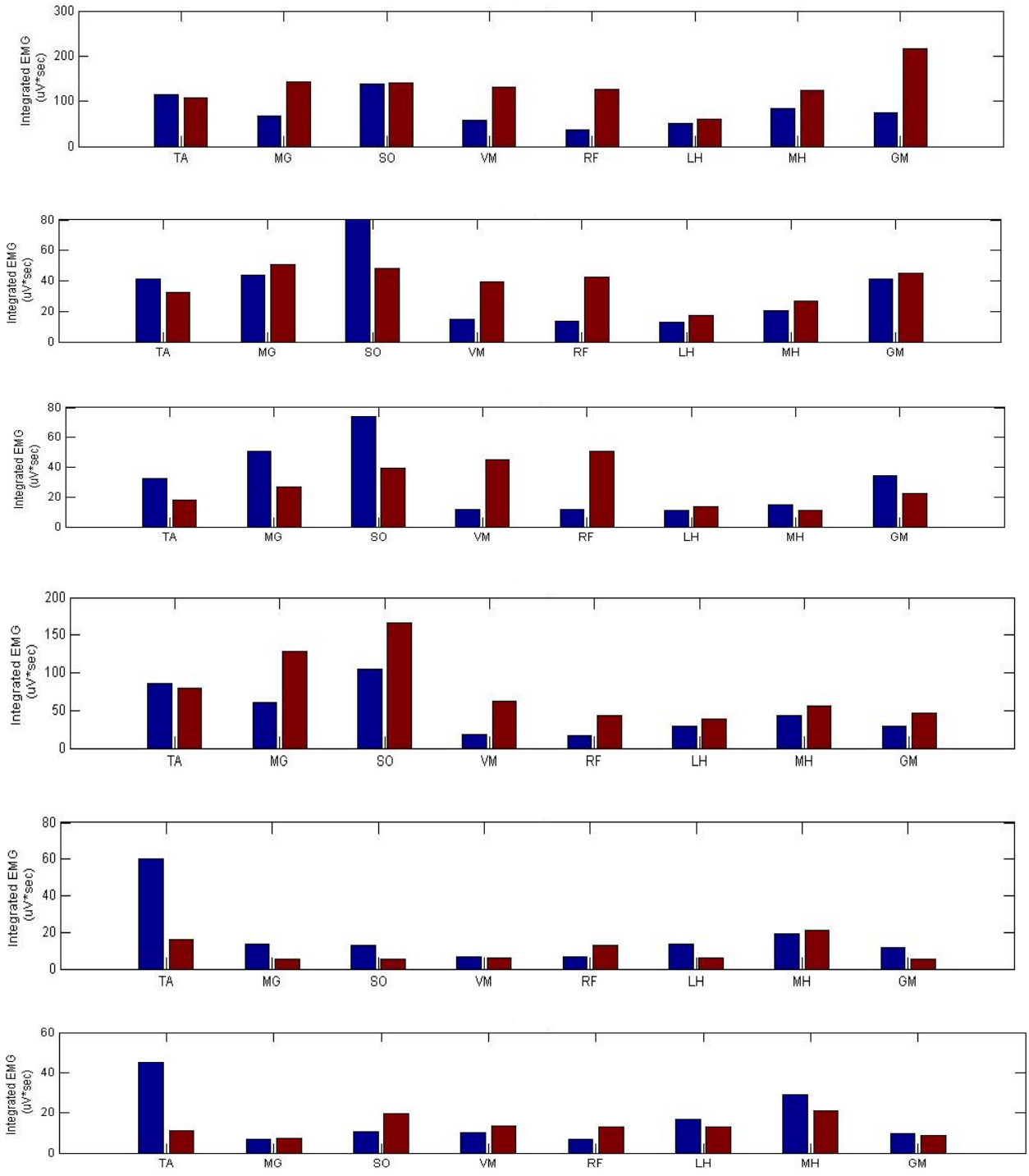


Figure A-4. Represents integrated EMG activity in different regions of the gait cycle in individuals walking at slow speed pre- and post-LT. Y-axis represents integrated EMG in µVs and X-axis represents various muscles recorded. TA=tibialis anterior, MG= medial gastrocnemius, SO= soleus, VM= vastus medialis, LH= lateral hamstrings, MH= medial hamstrings and GM= gluteus medius.

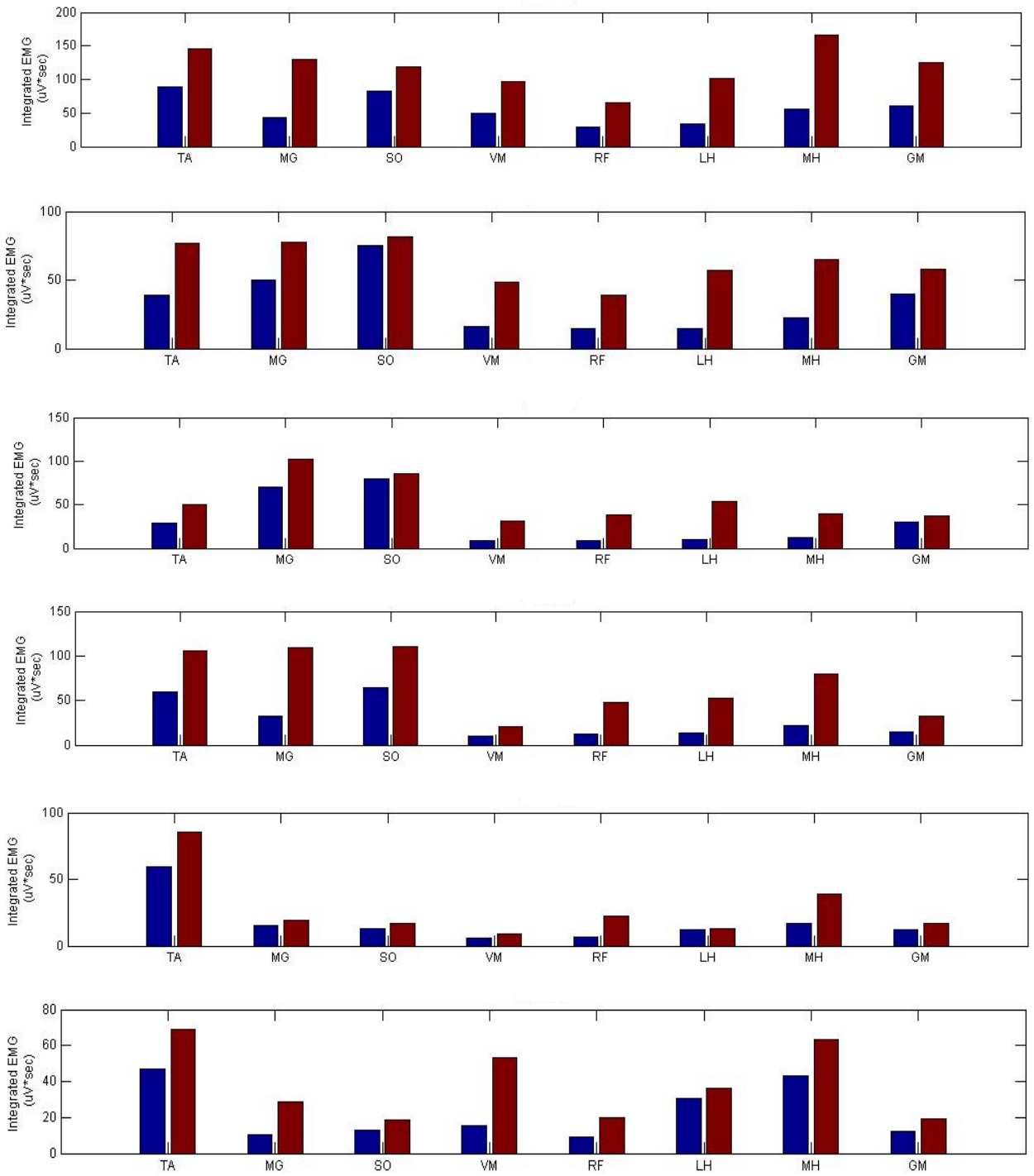


Figure A-5. Represents integrated EMG activity in different regions of the gait cycle in individuals walking at slow speed pre- and moderate speed post-LT. Y-axis represents integrated EMG in µVs and X-axis represents various muscles recorded. TA=tibialis anterior, MG= medial gastrocnemius, SO= soleus, VM= vastus medialis, LH= lateral hamstrings, MH= medial hamstrings and GM= gluteus medius.

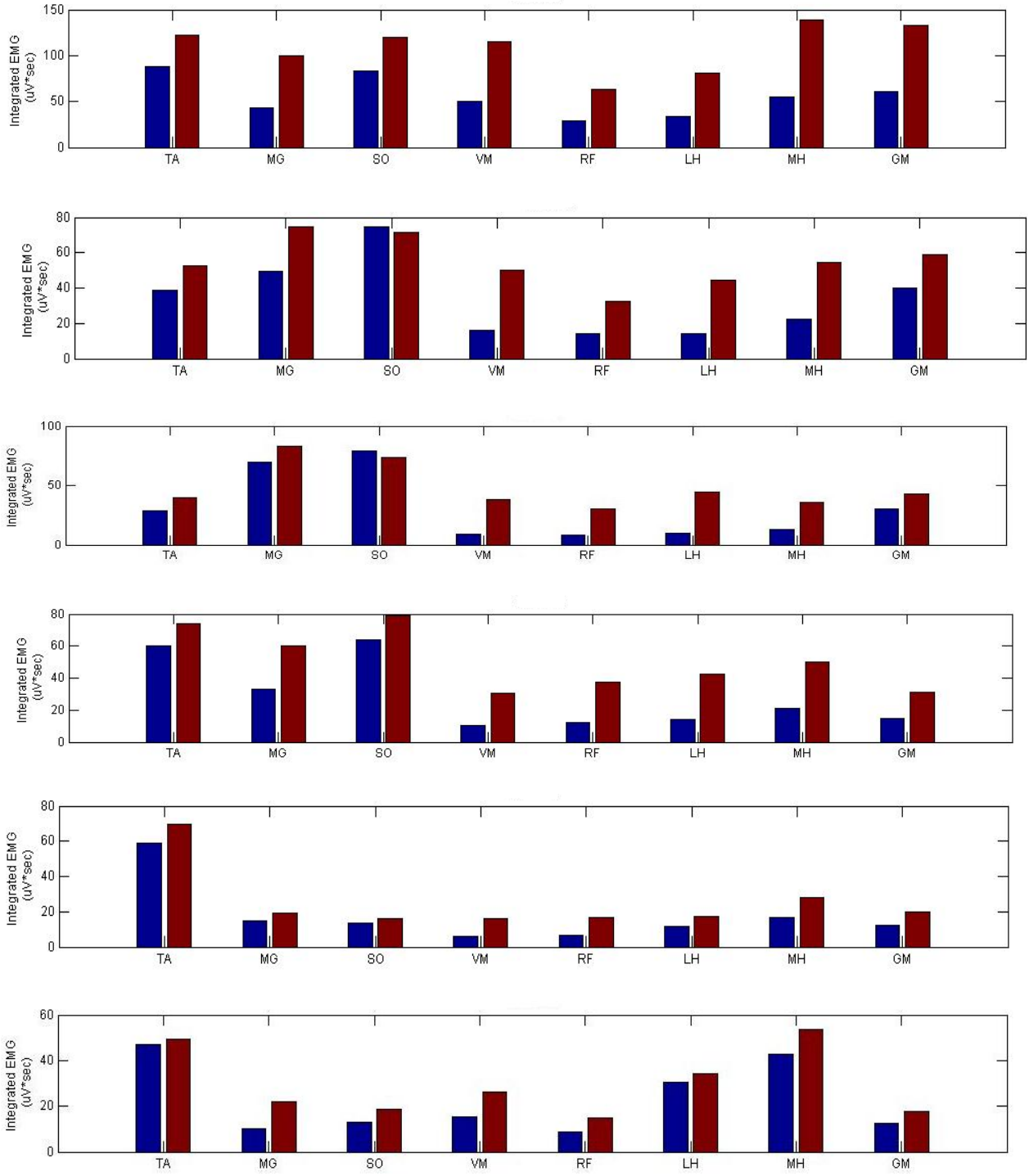


Figure A-6. Represents integrated EMG activity in different regions of the gait cycle in individuals walking at moderate speed pre- LT. Y-axis represents integrated EMG in µVs and X-axis represents various muscles recorded. TA=tibialis anterior, MG= medial gastrocnemius, SO= soleus, VM= vastus medialis, LH= lateral hamstrings, MH= medial hamstrings and GM= gluteus medius.

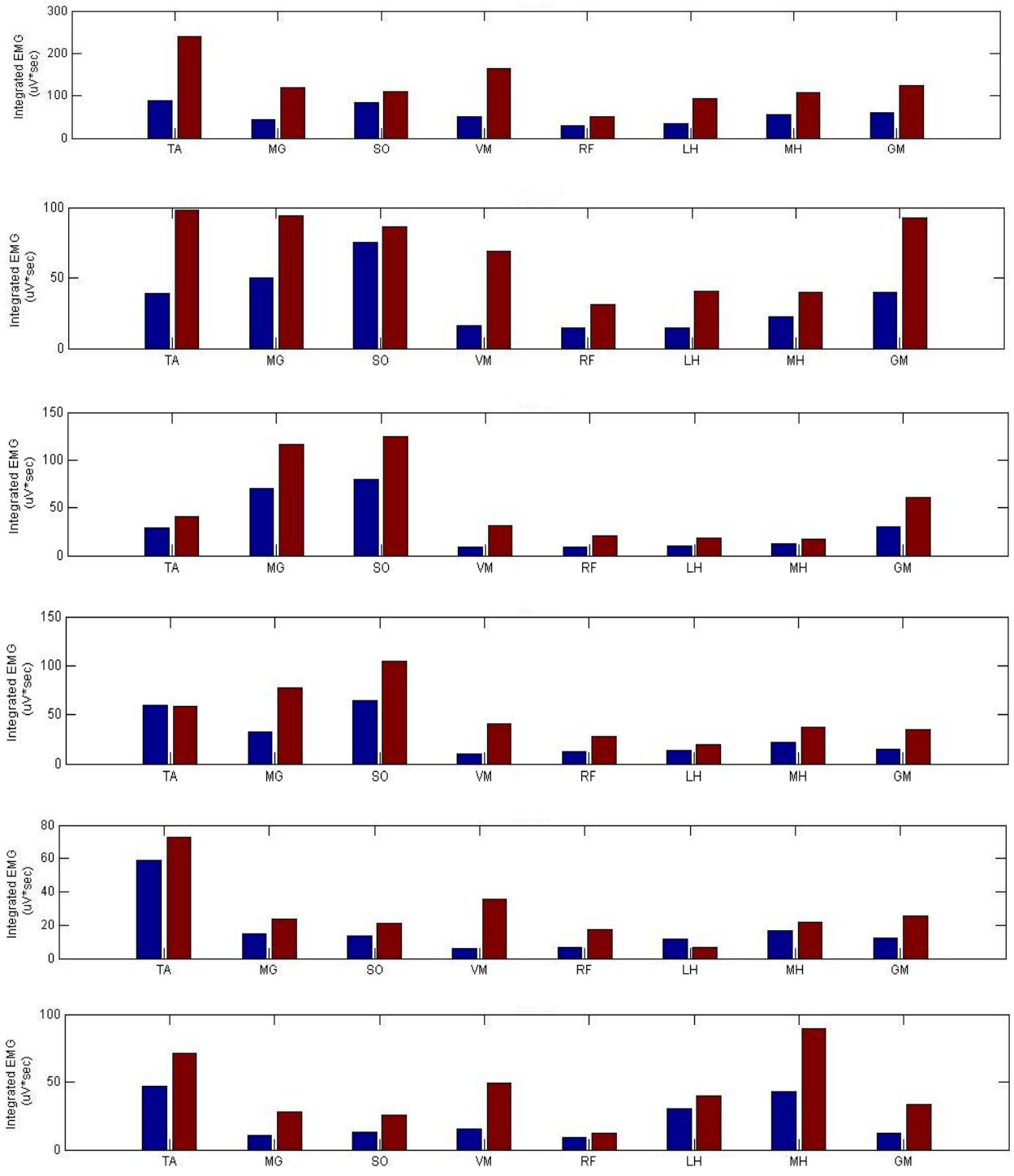


Figure A-7. Represents integrated EMG activity in different regions of the gait cycle in individuals walking at moderate speed pre- and post-LT. Y-axis represents integrated EMG in µVs and X-axis represents various muscles recorded. TA=tibialis anterior, MG= medial gastrocnemius, SO= soleus, VM= vastus medialis, LH= lateral hamstrings, MH= medial hamstrings and GM= gluteus medius.

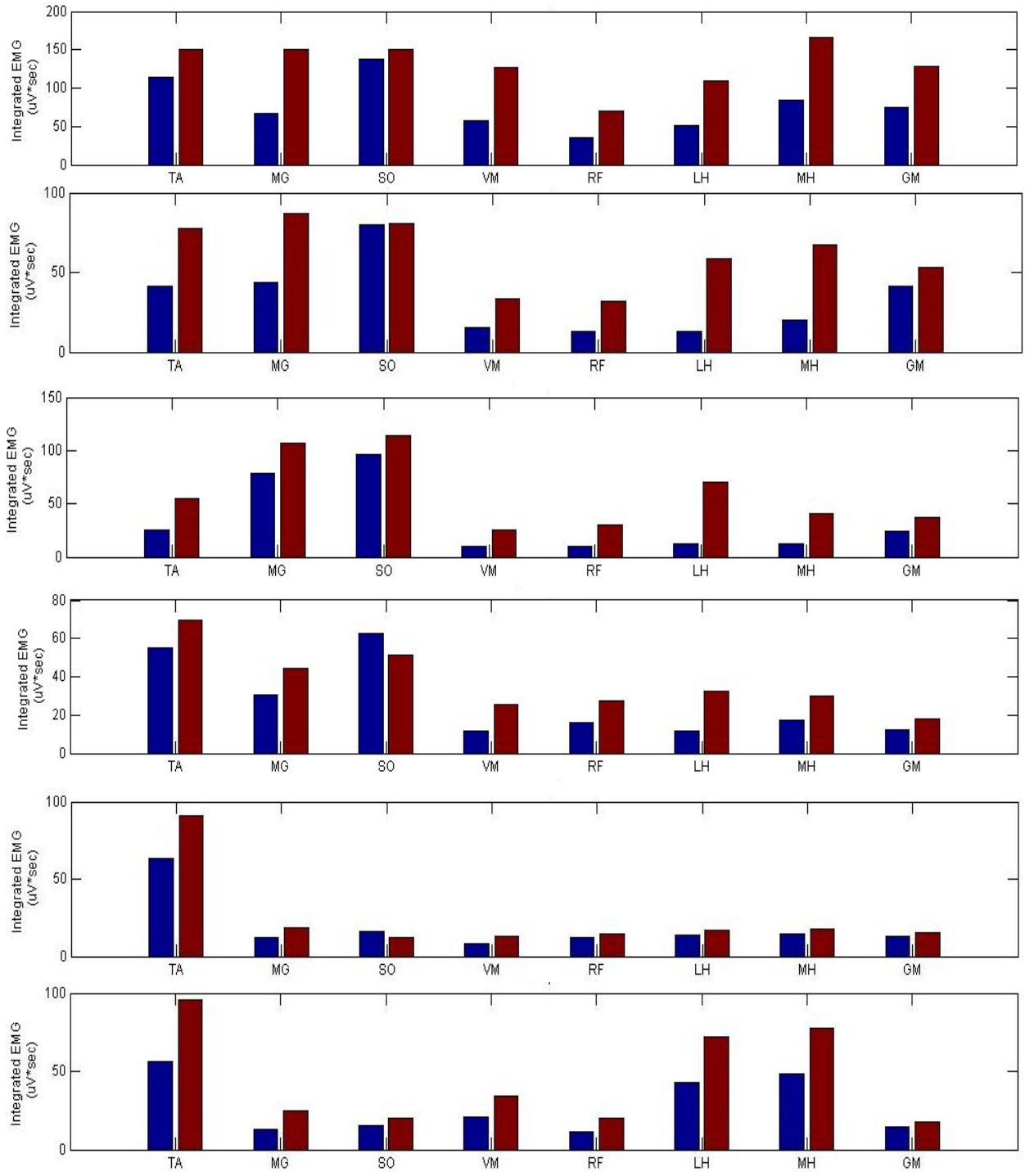


Figure A-8. Represents integrated EMG activity in different regions of the gait cycle in individuals walking at moderate speed pre- LT and at fast speed post-LT. Y-axis represents integrated EMG in µVs and X-axis represents various muscles recorded. TA=tibialis anterior, MG= medial gastrocnemius, SO= soleus, VM= vastus medialis, LH= lateral hamstrings, MH= medial hamstrings and GM= gluteus medius.

APPENDIX B
TABLES FOR PERCENTAGE DURATION OF ACTIVITY BEFORE AND AFTER LOCOMOTOR TRAINING

Table B-1. Represents the percentage duration of activity pre and post-LT over the entire gait cycle in individuals walking at different speeds. All values are represented as percentages.

| | Pre-LT slow | Post-LT slow | Control slow | slow-moderate post-LT | Control moderate | Pre-LT moderate | Post-LT moderate | moderate-fast post-LT | controls fast |
|---|---|---|---|---|---|---|---|---|---|
| TA | 39.88 ±12.3 | 35.34 ±3.3 | 53.27 ±12.6 | 50.26 ±12 | 55.70 ±9.76 | 50.47 ±8.6 | 52.05 ±4.9 | 51.21 ±8.4 | 53.07 ±10.1 |
| MG | 52.31 ±9.75 | 51.56 ±6.8 | 43.63 ±6.8 | 50.63 ±9.25 | 44.70 ±7.8 | 57.11 ±8.7 | 62.15 ±6.9 | 55.42 ±10.7 | 46.21 ±5.9 |
| SO | 64.23 ±11.6 | 65.26 ±2.65 | 53.85 ±5.6 | 56.89 ±7.1 | 53.20 ±5.8 | 64.27 ±7.3 | 64.965 ±3.4 | 58.33 ±4.08 | 51.85 ±3.7 |
| VM | 55.89 ±12.4 | 64.48 ±6.9 | 45.69 ±8.8 | 49.96 ±11.7 | 43.96 ±8.3 | 52.19 ±7.5 | 53.847 ±17.2 | 47.24 ±7.8 | 41.13 ±9.9 |
| RF | 58.44 ±11.6 | 64.38 ±3.7 | 48.60 ±11.7 | 55.45 ±11.8 | 47.71 ±10.4 | 54.86 ±15.6 | 54.195 ±13.2 | 57.51 ±8.2 | 51.69 ±9.2 |
| LH | 56.77 ±12.8 | 56.58 ±12.01 | 45.60 ±12.4 | 52.32 ±12.62 | 46.28 ±10.9 | 58.36 ±10.2 | 49.028 ±13.6 | 58.83 ±3.9 | 45.00 ±10.6 |
| MH | 62.07 ±14.2 | 53.55 ±9.9 | 49.78 ±11.9 | 58.21 ±9.4 | 49.51 ±11.7 | 62.98 ±9.7 | 47.36 ±10.4 | 60.82 ±6.6 | 48.18 ±11.9 |
| GM | 61.04 ±13.7 | 54.13 ±8.1 | 54.87 ±6.6 | 59.74 ±9 | 54.64 ±5.4 | 60.59 ±11.4 | 65.00 ±3 | 57.14 ±8.9 | 54.07 ±6.6 |

Table B-2. Represents percentage duration of activity of each muscle in different regions of the gait cycle in individuals walking at slow speed (0- <0.4m/s) pre-LT. All values are in percentages. Column 1 represents all the muscles.

| Column1 | Region 1 | Region 2 | Region 3 | Region 4 | Region 5 | Region 6 |
|---|---|---|---|---|---|---|
| TA | 41.51±16.7 | 38.91±20.5 | 25.24±20.3 | 35.99±13.5 | 84.32±17.2 | 53.22±21.8 |
| MG | 64.72±11.9 | 68.01±15.4 | 55.60±19.8 | 43.72±17.3 | 11.05±21.6 | 24.64±21.8 |
| SO | 76.62±13.1 | 77.03±15.5 | 71.55±17.3 | 59.27±13.3 | 13.78±24 | 27.95±24.4 |
| VM | 79.78±15.1 | 70.57±18.5 | 61.48±24 | 27.47±21.5 | 32.41±30.7 | 61.93±29.4 |
| RF | 72.31±17.2 | 70.29±18.9 | 63.10±18.8 | 41.36±16.5 | 48.72±34.1 | 58.86±28.2 |
| LH | 75.01±16.66 | 68.19±23.5 | 44.10±30.2 | 39.73±18.2 | 40.70±32.0 | 69.85±24.7 |
| MH | 82.45±16.5 | 58.74±19.6 | 34.26±24.2 | 48.79±21.4 | 69.61±27.2 | 83.24±16.8 |
| GM | 84.07±16.2 | 85.26±15.2 | 72.74±21 | 31.93±18.4 | 32.94±32.8 | 37.51±29.2 |

Table B-3. Represents percentage duration of activity of each muscle in different regions of the gait cycle in individuals walking at moderate speed (0.4-0.8m/s) pre-LT. All values are in percentages. Column 1 represents all the muscles.

| Column1 | Region 1 | Region 2 | Region 3 | Region 4 | Region 5 | Region 6 |
|---|---|---|---|---|---|---|
| TA | 56.44±17.2 | 40.42±19.6 | 27.81±17.1 | 40.25±15.3 | 89.64±12 | 67.59±20.4 |
| MG | 69.18±18 | 79.53±17.6 | 70.047±25.3 | 46.15±19.7 | 21.26±16.9 | 35.79±29.7 |
| SO | 80.10±9.1 | 89.18±12.8 | 87.01±10.6 | 58.81±12.5 | 15.9±19.4 | 19.93±14.9 |
| VM | 89.97±8.85 | 69.95±20 | 47.89±24.9 | 17.76±13.1 | 11.60±13.2 | 56.19±17.4 |
| RF | 77.93±23.4 | 62.81±31.5 | 48.56±30.7 | 45.79±13.5 | 35.65±21.6 | 38.77±19.2 |
| LH | 83.37±9.9 | 62.91±26.9 | 56.92±30.5 | 33.97±19.6 | 27.73±22.3 | 76.61±19.7 |
| MH | 93.48±9.8 | 61.16±23.5 | 40.473±26.2 | 40.00±16.1 | 44.12±26.6 | 90.88±7.9 |
| GM | 93.95±8.7 | 85.48±23.2 | 66.59±24.8 | 24.74±11.6 | 40.92±23.8 | 32.26±16.3 |

Table B-4. Represents percentage duration of activity of each muscle in different regions of the gait cycle in individuals walking at slow speed (0- <0.4m/s) post-LT. All values are in percentages. Column 1 represents all the muscles.

| **Column1** | **Region 1** | **Region 2** | **Region 3** | **Region 4** | **Region 5** | **egion 6** |
|---|---|---|---|---|---|---|
| TA | 49.91±10.5 | 39.26±15.6 | 12.32±6.1 | 23.83±10.4 | 67.92±33.1 | 60.01±20.2 |
| MG | 63.74±5.6 | 68.43±16.6 | 45.13±22.6 | 44.49±9.4 | 6.675±9.3 | 21.50±16 |
| SO | 75.03±9.1 | 72.24±4.7 | 63.66±14.1 | 68.96±8.2 | 6.68±10.4 | 17.20±8.7 |
| VM | 89.46±6.9 | 84.19±9.7 | 85.075±11.4 | 33.49±18.4 | 18.68±33.7 | 60.60±17.1 |
| RF | 81.98±8.6 | 79.50±11.3 | 68.02±22.5 | 50.04±7.3 | 28.74±22.5 | 59.92±12.6 |
| LH | 75.45±19.2 | 71.45±23.5 | 51.71±31.1 | 37.46±13.9 | 40.68±30.1 | 66.88±10.8 |
| MH | 82.40±12.2 | 52.60±23.2 | 15.38±12.4 | 39.35±25.8 | 66.74±31.4 | 77.43±15 |
| GM | 89.69±8 | 86.15±15.2 | 59.91±25.9 | 14.18±6.6 | 18.31±14.7 | 31.62±18.5 |

Table B-5. Represents percentage duration of activity of each muscle in different regions of the gait cycle in individuals walking at slow speed (0- <0.4m/s) pre-LT and moderate speed (0.4-0.8 m/s) post-LT. All values are in percentages. Column 1 represents all the muscles.

| **Column1** | **Region 1** | **Region 2** | **Region 3** | **Region 4** | **Region 5** | **Region 6** |
|---|---|---|---|---|---|---|
| TA | 62.86±18.9 | 51.42±23.3 | 29.98±21.9 | 42.65±16.7 | 76.33±25.2 | 57.74±27.3 |
| MG | 66.61±14.9 | 62.12±9.9 | 62.16±17.3 | 48.24±14.45 | 9.26±11.3 | 20.81±23.6 |
| SO | 69.14±1.8 | 70.24±14.6 | 73.55±19 | 61.25±12.6 | 13.28±2.4 | 17.44±17.6 |
| VM | 84.39±10.8 | 67.55±18.3 | 52.67±27.6 | 11.33±10 | 9.78±10.27 | 59.65±24.7 |
| RF | 69.48±16.3 | 60.35±17.9 | 54.85±26.3 | 45.57±13.8 | 46.42±28 | 47.49±28.1 |
| LH | 82.20±12.5 | 51.06±31.6 | 43.25±27.4 | 32.47±18.8 | 21.00±16.2 | 64.8±26.8 |
| MH | 88.04±7.8 | 45.81±18.7 | 29.93±14.6 | 47.93±18 | 51.69±26.9 | 78.62±19.9 |
| GM | 89.97±10.5 | 81.73±16.8 | 60.17±18.4 | 28.79±21.7 | 39.36±33 | 36.02±20.5 |

Table B-6. Represents percentage duration of activity of each muscle in different regions of the gait cycle in individuals walking at moderate speed (0.4- 0.8 m/s) pre and post-LT. All values are in percentages. Column 1 represents all the muscles.

| Column1 | Region 1 | Region 2 | Region 3 | Region 4 | Region 5 | Region 6 |
|---|---|---|---|---|---|---|
| TA | 83.74±12.4 | 58.29±27.2 | 15.35±12.9 | 20.80±5.7 | 68.10±39 | 70.00±10.8 |
| MG | 68.98±37.7 | 80.23±16.4 | 87.83±11.7 | 55.42±19.3 | 24.49±22.7 | 41.18±9.7 |
| SO | 73.47±18.8 | 84.81±8.7 | 96.66±4.7 | 59.44±25.4 | 20.92±20.5 | 28.34±30.8 |
| VM | 95.41±1.9 | 72.74±29 | 29.33±26.4 | 24.27±23.9 | 26.04±8.1 | 59.02±26.8 |
| RF | 82.91±18.2 | 67.57±29.3 | 39.21±18.3 | 42.78±10.6 | 42.68±27.7 | 34.81±17.7 |
| LH | 89.45±13 | 65.02±42.8 | 26.44±19.5 | 13.41±21.6 | 3.97±3.3 | 80.78±10.8 |
| MH | 88.42±17 | 36.89±18.7 | 10.71±8.4 | 21.5±23 | 31.74±23.2 | 93.74±7.1 |
| GM | 97.49±3.4 | 93.71±10.5 | 73.19±18.2 | 26.30±12 | 42.43±25.5 | 39.45±26.7 |

Table B-7. Represents percentage duration of activity of each muscle in different regions of the gait cycle in individuals walking at fast speed (>0.8m/s) post-LT. All values are in percentages. Column 1 represents all the muscles.

| Column1 | Region 1 | Region 2 | Region 3 | Region 4 | Region 5 | Region 6 |
|---|---|---|---|---|---|---|
| TA | 57.64±13.8 | 57.68±23.5 | 44.78±22.5 | 38.74±16.9 | 76.23±15.3 | 62.77±15 |
| MG | 49.12±15 | 72.27±13.9 | 73.23±18.2 | 38.40±8.2 | 17.94±21.5 | 7.57±8.9 |
| SO | 64.63±12.4 | 91.12±6.9 | 89.71±7.75 | 51.62±9.4 | 5.12±6.1 | 7.57±10.5 |
| VM | 82.03±15.4 | 61.46±20.4 | 41.22±17 | 20.38±11.3 | 10.85±13.4 | 42.49±19.4 |
| RF | 79.86±15.7 | 67.21±25.6 | 47.92±24.9 | 27.0672619 | 20.51±13.7 | 37.21±12.8 |
| LH | 68.88±20.7 | 43.61±22.4 | 31.26±21.5 | 28.43±15.6 | 39.05±22.1 | 55.26±20.4 |
| MH | 72.88±19.1 | 45.63±21.7 | 28.76±18.4 | 32.76±20.8 | 44.32±22.4 | 65.18±16.6 |
| GM | 80.66±11.5 | 96.94±4.54 | 88.72±8.8 | 25.63±15 | 22.15±16.6 | 20.45±14.7 |

Table B-8. Represents percentage duration of activity of each muscle in different regions of the gait cycle in healthy control individuals walking at slow speed (0.3m/s). All values are in percentages. Column 1 represents all the muscles.

| Column1 | Region 1 | Region 2 | Region 3 | Region 4 | Region 5 | Region 6 |
|---|---|---|---|---|---|---|
| TA | 57.64±13.8 | 57.68±23.5 | 44.78±22.5 | 38.74±16.9 | 76.23±15.3 | 62.77±15 |
| MG | 49.12±15 | 72.27±13.9 | 73.23±18.2 | 38.40±8.2 | 17.94±21.5 | 7.57±8.9 |
| SO | 64.63±12.4 | 91.12±6.9 | 89.71±7.75 | 51.62±9.4 | 5.12±6.1 | 7.57±10.5 |
| VM | 82.03±15.4 | 61.46±20.4 | 41.22±17 | 20.38±11.3 | 10.85±13.4 | 42.49±19.4 |
| RF | 79.86±15.7 | 67.21±25.6 | 47.92±24.9 | 27.06±19 | 20.51±13.7 | 37.21±12.8 |
| LH | 68.88±20.7 | 43.61±22.4 | 31.26±21.5 | 28.43±15.6 | 39.05±22.1 | 55.26±20.4 |
| MH | 72.88±19.1 | 45.63±21.7 | 28.76±18.4 | 32.76±20.8 | 44.32±22.4 | 65.18±16.6 |
| GM | 80.66±11.5 | 96.94±4.54 | 88.72±8.8 | 25.63±15 | 22.15±16.6 | 20.45±14.7 |

Table B-9. Represents percentage duration of activity of each muscle in different regions of the gait cycle in healthy control individuals walking at matched moderate speed (0.6m/s). All values are in percentages. Column 1 represents all the muscles.

| Column1 | Region1 | Region 2 | Region 3 | Region 4 | Region 5 | Region 6 |
|---|---|---|---|---|---|---|
| TA | 65.41±11.5 | 49.11±22 | 40.15±24.4 | 41.52±11.8 | 76.97±15.9 | 62.037±15.6 |
| MG | 44.0±17.5 | 75.39±18.8 | 88.40±13.5 | 31.35±12.3 | 22.54± | 12.68±12 |
| SO | 64.2±12.3 | 91.01±7.8 | 94.02±7.6 | 47.36±10.7 | 9.96±11.6 | 11.33±9.48 |
| VM | 90.2±7.1 | 53.40±19.5 | 23.42±16.8 | 14.94±10.4 | 13.56±10.6 | 54.35±19.3 |
| RF | 85.07±14 | 57.95±26.7 | 27.63±23.4 | 28.77±13.4 | 27.11±11.3 | 37.96±15.7 |
| LH | 70.77±15.8 | 42.09±19.1 | 26.41±20.9 | 22.59±15.0 | 42.20±24.5 | 79.86±17.3 |
| MH | 73.06±18.4 | 46.49±19.2 | 25.15±21.6 | 26.60±9 | 44.76±23.6 | 85.85±11.3 |
| GM | 89.62±8.3 | 93.57±9.7 | 73.47±18.1 | 19.88±12.7 | 26.015±16.6 | 28.91±19.3 |

Table B-10. Represents percentage duration of activity of each muscle in different regions of the gait cycle in healthy control individuals walking at matched fast speed (0.9m/s). All values are in percentages. Column 1 represents all the muscles

| Column1 | Region 1 | Region 2 | Region 3 | Region 4 | Region 5 | Region 6 |
|---|---|---|---|---|---|---|
| TA | 66.98±13.5 | 41.83±21.5 | 29.03±22.1 | 40.34±14.9 | 76.65±11.6 | 63.67±14.9 |
| MG | 46.36±16.6 | 81.40±16 | 93.41±7.4 | 29.08±10.5 | 15.38±2.2 | 15.38±15.5 |
| SO | 62.60±8.7 | 91.89±8.3 | 95.40±6.9 | 43.89±12.3 | 7.09±7.4 | 7.91±5.9 |
| VM | 92.66±4.8 | 45.08±24.3 | 18.08±13.9 | 13.90±10.8 | 15.44±11.5 | 54.45±18.5 |
| RF | 89.75±8.2 | 57.81±20.3 | 27.46±22.8 | 38.35±17.5 | 41.55±20.9 | 47.16±16.5 |
| LH | 68.85±18 | 43.1±19.8 | 21.98±18.6 | 16.43±13 | 36.20±23.7 | 85.14±17.2 |
| MH | 73.23±17.4 | 44.08±21.4 | 24.80±23.1 | 23.41±22 | 33.37±21.9 | 89.37±7.9 |
| GM | 92.25±6.1 | 91.67±10.2 | 58.42±24.6 | 16.83±11.2 | 30.54±18.3 | 34.75±20.7 |

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# BIOGRAPHICAL SKETCH

Bhavana Raja graduated from Holy Angels School in Rajpura, Punjab, India in 1996. She received her Bachelor in Physical Therapy from Guru Nanak Dev University, Amritsar, Punjab, India . Her keenness to help people with neurological impairments lead a better quality of life 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 worked under the expertise and guidance of Dr. Steve Kautz.