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Simulation and experimental analyses to assess walking performance post-stroke using step length asymmetry and module composition

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Simulation and experimental analyses to assess walking performance post-stroke using step length asymmetry and module composition

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Dissertation
Presented to the Faculty of the Graduate School of
The University of Texas at Austin
in Partial Fulfillment
of the Requirements
for the Degree of

Doctor of Philosophy

The University of Texas at Austin
August 2012
Dedication

This dissertation is dedicated to my parents, for their unconditional love and support.
Acknowledgements

I would like to thank my advisor, Dr. Richard Neptune, for providing a rich and collaborate research environment, supporting me, and helping me grow as a researcher. I would also like to thank Dr. Steven Kautz for his support of my research and insightful opinions as well as our collaborators at the University of Florida and the Medical University of South Carolina for their help with data collection and processing. I would also like to thank Dr. Lawrence Abraham, Dr. Ronald Barr and Dr. Ashish Deshpande for serving on my dissertation committee.

I would like to thank both the past and present members of the Neuromuscular Biomechanics Laboratory not only for providing their support and encouragement but also for providing a truly enjoyable and collaborative work environment. It has been a pleasure to have met and worked with such a great group of researchers and friends.

I am truly grateful to have had such great support from both my friends and family. Without them, the past few years would have been drab and dreary. To my mom, thank you for always telling me everything would be ok whenever I called (which was often). To Jennifer, thank you for providing tons of laughter and joining me on spontaneous international vacations. And to all my friends in Austin, thank you for helping make this a city full of wonderful people and one that I will always love and cherish.

Finally, I am very grateful to have received financial support from the National Science Foundation Graduate Research Fellowship Program and the Jack and Maxine Zarrow Endowed Graduate Fellowship in Engineering.
Simulation and experimental analyses to assess walking performance post-stroke using step length asymmetry and module composition

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The University of Texas at Austin, 2012

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Understanding the underlying coordination mechanisms that lead to a patient’s poor walking performance is critical in developing effective rehabilitation interventions. However, most common measures of rehabilitation effectiveness do not provide information regarding underlying coordination mechanisms. The overall goal of this research was to analyze the relationship between two potential measures of walking performance (step length asymmetry and module composition) and underlying walking mechanics.

Experimental analyses were used to analyze the walking mechanics of hemiparetic subjects grouped by step length asymmetry. All groups had impaired plantarflexor function and the direction of asymmetry provided information regarding the compensatory mechanism used to overcome this plantarflexor impairment. Those subjects who walked with longer paretic than nonparetic steps compensated using increased output from the nonparetic leg, while those with symmetric steps compensated using a bilateral hip strategy. These results suggest that step length asymmetry may provide information regarding underlying coordination mechanisms that can be used to guide rehabilitation efforts.
Another way to assess walking performance is to directly analyze deficits in muscle coordination. Recent studies have suggested that complex muscle activity during walking may be generated using a reduced neural control strategy organized around the co-excitation of multiple muscles, or modules, which may provide a useful framework for characterizing coordination deficits. Simulation analyses using modular control were performed to understand how modules contribute to important biomechanical functions of non-impaired walking and how the generation of these functions is altered in groups of post-stroke hemiparetic subjects who commonly merged different sets of non-impaired modules. The non-impaired simulation found that six modules are needed to generate the three-dimensional tasks of walking (support, forward propulsion, mediolateral balance control and leg swing control). When the plantarflexor module was merged with the module controlling the knee extensors and hip abductors, forward propulsion and ipsilateral leg swing were impaired. When the module controlling the hamstrings was merged with the module controlling the knee extensors and hip abductors, forward propulsion, body support and mediolateral balance control were impaired. These results suggest that module analysis may provide useful information regarding the source of walking deficits and can be used to guide rehabilitation efforts.
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Stroke is a leading cause of long term disability in the United States (Roger et al., 2012) that often leaves survivors with various levels of hemiparesis affecting their mobility. Post-stroke hemiparetic gait is characterized by slow walking speeds and asymmetry between the paretic and nonparetic legs (e.g., Olney and Richards 1996), which likely results from altered muscle coordination (i.e., muscle force production and timing; e.g., Den Otter et al., 2007; Turns et al., 2007). The effects of stroke are heterogeneous, with impairments and compensatory strategies varying among subjects (Knutsson and Richards 1979). Current clinical measures used to assess improvements in walking performance during rehabilitation provide limited information regarding how the improvements were actually obtained. For example, the most commonly used measure of rehabilitation effectiveness is self-selected walking speed (Bowden et al., 2012), as it is sensitive to change through recovery (Richards et al., 1995) and is a predictor of quality of life (Perry et al., 1995; Schmid et al., 2007). However, increased walking speed can be achieved through several different mechanisms (e.g., improved coordination, increased reliance on an already developed compensatory mechanism, or development of a new compensatory mechanism). In order to design rehabilitation strategies that target improving coordination instead of relying on compensatory mechanisms, identifying measures that provide information regarding mechanisms underlying impaired coordination is needed.

Measures based on gait asymmetry may provide some of this missing information. For example, post-stroke hemiparetic patients walking at similar speeds often exhibit different step length asymmetries (e.g., Balasubramanian et al., 2007; Hsu et
al., 2003; Kim and Eng 2003). This variability in asymmetry likely results from the use of different coordination mechanisms, and thus step length asymmetry may be more indicative of underlying impairments and compensatory mechanisms than measures like self-selected walking speed. A recent study found that step length asymmetry of post-stroke hemiparetic subjects negatively correlates with propulsion asymmetry (Balasubramanian et al., 2007), with those subjects showing decreased nonparetic step length compared to the paretic leg generating insufficient propulsion during paretic leg stance. Hemiparetic subjects often exhibit impairment in the paretic leg plantarflexors (e.g., Lamontagne et al., 2007; Turns et al., 2007), which is a critical muscle group for generating forward propulsion (Neptune et al., 2001), and it is likely that those subjects with longer paretic than nonparetic steps may have an active reduction in paretic leg propulsion due to plantarflexor impairment.

Conversely, subjects walking asymmetrically with shorter paretic steps generated a higher percentage of total propulsion by the paretic leg (Balasubramanian et al., 2007). While one explanation may be that the plantarflexors are unimpaired, it is more likely that these subjects generate insufficient pre-swing energy to the paretic leg (which would reduce the paretic leg swing time, thus decreasing paretic step length). The paretic hip flexors are also important for walking performance (Jonsdottir et al., 2009; Nadeau et al., 1999). They often compensate for plantarflexor impairment by advancing the leg further before its subsequent heel strike (Nadeau et al., 1999) and thus contribute to swing initiation (Neptune et al., 2004). It is likely that subjects with shorter paretic steps have reduced paretic leg advancement during swing due to impaired hip flexor activity in pre-swing. If step length asymmetry is a good indicator of walking impairment in post-stroke hemiparetic subjects, it may be useful in developing and monitoring rehabilitation programs as a simple surrogate for more complex biomechanical measures to identify
motor impairments. Therefore, the goal of the first study in Chapter 2 was to determine if post-stroke hemiparetic subjects grouped by step length asymmetry have similar walking biomechanics relative to non-impaired walkers.

Just as the same walking speed can be achieved with different step length asymmetry values, a disadvantage of asymmetry measures is that the same asymmetry value may be achieved using different underlying muscle coordination strategies. To overcome this disadvantage, another way to assess walking ability is to directly analyze muscle coordination. However, walking requires the coordination of multi-articular limbs with many degrees-of-freedom using a set of highly redundant muscles. Due to the difficulty in recording and analyzing electromyography (EMG) of multiple muscles spanning multiple joints, muscle activity has not typically been used to assess walking ability. Recent experimental work, though, has found that complex muscle activity during walking may be generated using a reduced neural control strategy organized around the co-excitation of multiple muscles, or modules (Cappellini et al., 2006; Clark et al., 2010; Ivanenko et al., 2004). These modules represent the underlying coordination patterns using a reduced number of variables, making this type of analysis potentially useful for characterizing a patient’s coordination deficits and changes throughout rehabilitation.

Recent dynamic modeling and simulation studies have examined whether modules during non-impaired walking are structured to perform the important biomechanical functions of support, forward propulsion and leg swing (McGowan et al., 2010; Neptune et al., 2009). Each module was found to be associated with specific biomechanical functions that resulted in a well-coordinated walking pattern. Module 1 (hip and knee extensors) primarily contributed to support in early stance while Module 2 (ankle plantarflexors) primarily contributed to support and propulsion in late stance. Module 3 (tibialis anterior and rectus femoris) acted to decelerate the leg in early and late
swing while generating energy to the trunk throughout swing and Module 4 (hamstrings) acted to absorb leg energy (i.e. decelerate it) in late swing while increasing the leg energy in early stance. Finally, Module 5 (hip flexors) acted to accelerate the leg forward in pre- and early swing. However, these studies used a model that was constrained to the sagittal plane, and thus the contributions of each module to mediolateral balance control and contralateral leg swing could not be assessed. Walking is a three-dimensional task in which these additional non-sagittal plane biomechanical functions are important to provide a basis of comparison for hemiparetic subjects. Therefore, the goal of the second study in Chapter 3 was to use a three-dimensional muscle-actuated forward dynamics simulation of non-impaired walking to investigate the contributions of each module to mediolateral balance control and contralateral leg swing.

Compared to non-impaired walkers, post-stroke hemiparetic subjects often utilize a reduced number of modules, with at least one of the modules resembling a merging of multiple non-impaired modules (Clark et al., 2010). As the number of independently activated modules decreases, walking ability also decreases (e.g., self-selected speed and gait symmetry; Bowden et al., 2010; Clark et al., 2010). Thus a consequence of merged modules may be the inability to successfully generate each biomechanical function during walking, resulting in poor walking performance. In addition to the number of independently activated modules, which biomechanical functions are affected likely depends on the specific modules that are merged. For example, within those subjects who merged two non-impaired modules, two common types of module compositions have been identified (Clark et al., 2010). One type appeared to merge Modules 1 and 2 such that the ankle plantarflexors and proximal extensors were co-active throughout stance. A second type appeared to merge Modules 1 and 4 such that the proximal extensors and hamstrings were co-activated from late swing into stance. It is likely that subjects using
each module composition would have different neuromotor impairments. However, it is not known specifically how important biomechanical functions during walking (e.g., support, forward propulsion, mediolateral balance control and leg swing control) are affected for each of these types of module compositions. Identifying the relationship between module composition and biomechanical function has important implications for designing locomotor therapies that target specific functional deficits. Therefore, the goal of the third study in Chapter 4 was to develop muscle-actuated forward dynamics simulations of walking for non-impaired controls and two groups of hemiparetic subjects in which different non-impaired modules are merged (Group A: merged Modules 1 and 2 and Group B: merged Modules 1 and 4) in order to identify the influence of merged modules on important biomechanical functions during walking.

The overall goal of this research was to use experimental and simulation analyses to investigate the potential of two different measures (step length asymmetry and module composition) to provide detailed information regarding walking performance. While step length asymmetry has been studied previously, its potential to classify subjects with similar walking mechanics has not been examined. In addition, this will be the first study to examine the relationship between different types of module compositions post-stroke and important biomechanical functions of walking. The results of this research can be used to develop rehabilitation interventions that target specific functional deficits, with the goal of improving walking performance and quality of life for those with post-stroke hemiparesis.
Chapter 2

Step Length Asymmetry is Representative of Compensatory Mechanisms Used in Post-Stroke Hemiparetic Walking

INTRODUCTION

Post-stroke hemiparetic subjects walk more slowly than non-impaired walkers, and self-selected walking speed is regularly used as a measure of rehabilitation outcome because it is related to functional status and quality of life (Perry et al., 1995). However, since compensatory action by the nonparetic leg can result in more effective self-selected walking speeds, speed alone is not a reliable measure of hemiparetic walking ability. While step length asymmetry has also been used as a measure of walking ability, it is weakly related to walking speed such that post-stroke hemiparetic patients walking at similar speeds may exhibit different step length asymmetries (Balasubramanian et al., 2007; Hsu et al., 2003; Kim and Eng 2003). Since this between subject variability may result from the use of different walking mechanisms, step length asymmetry may be more indicative of the underlying impairments and compensatory mechanisms used than self-selected walking speed.

An important factor in generating step length is forward propulsion, generated through the anterior-posterior ground reaction force (AP GRF) of the stance leg, which enables the trunk to progress forward while the contralateral leg is in swing (Balasubramanian et al., 2007; Roerdink and Beek 2010). A recent study found step length asymmetry of post-stroke hemiparetic subjects negatively correlates with propulsion asymmetry (Balasubramanian et al., 2007), with those subjects unable to generate sufficient propulsion during paretic leg stance showing decreased nonparetic leg step lengths compared to the paretic leg. Balasubramanian et al. (2007) suggest that
subjects who generate shorter nonparetic step lengths may have an active reduction in paretic leg propulsion generation due to ankle plantarflexor muscle impairment. The plantarflexors are important contributors to propulsion in non-impaired walking (McGowan et al., 2008; Neptune et al., 2001) and hemiparetic subjects often exhibit paretic leg plantarflexor impairment (Lamontagne et al., 2007; Turns et al., 2007). As a result, several studies have found step length and step length asymmetry to correlate with paretic leg plantarflexor impairment in post-stroke hemiparetic subjects (Hsu et al., 2003; Lin et al., 2006).

Conversely, subjects walking asymmetrically with shorter paretic steps generated a higher percentage of total propulsion by the paretic leg (Balasubramanian et al., 2007). While one explanation for this phenomenon could be that the paretic plantarflexors are unimpaired, it is more likely that these subjects do not generate sufficient pre-swing energy to the paretic leg (which would reduce the paretic leg swing time, thus decreasing paretic step length). Previous studies of hemiparetic walking have found the paretic hip flexors are also important for walking performance (Jonsdottir et al., 2009; Nadeau et al., 1999) and often compensate for plantarflexor impairment. During late stance the uniarticular hip flexors pull the leg upwards and forwards, acting to advance the leg further before its subsequent heel strike (Nadeau et al., 1999), which contributes to swing initiation (Neptune et al., 2004). Therefore it is possible that hemiparetic subjects with shorter paretic steps have reduced paretic leg advancement during swing due to impaired paretic leg hip flexor activity in pre-swing.

If step length asymmetry is a good indicator of walking impairment in post-stroke hemiparetic subjects, it may be helpful in developing and monitoring rehabilitation programs as a simple surrogate for more complex biomechanical measures to identify neuromotor impairments. While step length asymmetry has been studied previously
(Balasubramanian et al., 2007; Hsu et al., 2003; Lin et al., 2006), its potential to classify subjects into groups with similar walking mechanics has not been examined. Therefore, the goal of this study was to determine if post-stroke hemiparetic subjects grouped by step length asymmetry have similar walking biomechanics, as measured by joint kinetics from mid through late stance (when propulsion and pre-swing occur in non-impaired subjects), compared to non-impaired walkers. Compared to non-impaired walkers at a similar speed, it is hypothesized that those subjects who walk symmetrically will have similar joint moments, those who walk with longer paretic than nonparetic steps will have reduced paretic leg plantarflexor moment and those subjects who walk with shorter paretic than nonparetic steps will have a reduced paretic leg hip flexor moment.

**METHODS**

**Subjects**

Fifty-five subjects with chronic hemiparesis (28 left hemiparesis, 35 men; age 61.2 ± 11.4 years; 5.3 ± 5.7 years post stroke) and twenty-one age-matched non-impaired controls (4 men; age 65.2 ± 9.6 years) were recruited at the VA Brain Rehabilitation Research Center (Gainesville, FL). Inclusion criteria for the hemiparetic subjects included hemiparesis secondary to a single unilateral stroke, absence of significant lower extremity joint pain, limb contractures, or major sensory deficits, ability to walk independently with an assistive device over 10 m on a level surface, walk on a daily basis in the home, and absence of cardiovascular impairments contraindicative to walking. Subjects were excluded from the study if they had orthopedic or additional neurologic conditions. All subjects signed informed consent and protocol was approved by the Institutional Review Boards of the University of Florida and the University of Texas at Austin.
Experimental Setup and Procedure

Each hemiparetic subject completed three 30s walking trials on an instrumented split-belt treadmill (Tecmachine) at their self-selected treadmill walking speed without use of an assistive device. Each control subject completed trials at 0.3, 0.6 and 0.9 m/s for speed-matched comparisons. All subjects walked approximately 10 seconds prior to each data collection to ensure a steady-state walking pattern had been reached. A safety harness mounted to the laboratory ceiling protected the subjects in the event of a loss of balance. One or more practice trials were performed to ensure subjects’ comfort and safety. Retro-reflective markers were recorded using a twelve-camera system (Vicon Motion Systems) to assess bilateral 3D kinematics at 100 Hz. A modified Helen Hayes marker set that included additional marker triads attached to rigid plates located on each foot, shank and thigh segment was used to define each body segment and reduce measurement error. Bilateral GRFs were captured at 2000 Hz.

Data Analysis

Data were processed using Visual3D (C-Motion, Inc.). Marker and GRFs were low-pass filtered using a 4<sup>th</sup>-order Butterworth filter with cutoff frequencies of 6 Hz and 20 Hz, respectively. Intersegmental joint moments (normalized by subject body weight) were calculated using standard inverse dynamics analysis. Data were time normalized to 100% of the gait cycle (paretic leg for hemiparetic subjects and right leg for controls). Joint kinetics were calculated in a region approximately corresponding to the usual propulsive phase for each leg. This region was divided into late single-leg stance (i.e. second 50% of single-leg stance) and pre-swing (i.e. double support phase preceding swing). Within late single-leg stance and pre-swing, joint kinetics were calculated as the joint moment impulses (i.e. time integral of the joint moment) at the hip (flexor positive), knee (extensor positive) and ankle (plantarflexor positive). To quantify propulsion, the
AP GRF impulses (AP impulse) during late single-leg stance and pre-swing were calculated. For the control subjects, the right and left leg values were averaged.

**Statistical Analyses**

Hemiparetic subjects were grouped by PSR (calculated as the paretic step length divided by the sum of paretic and nonparetic step length) into high (PSR>0.535), symmetric (0.535≥ PSR≥ 0.465), and low (PSR<0.465) groups. This grouping criterion was derived from the range of PSR values from the non-impaired controls who were considered to walk symmetrically (Kautz et al., 2012). All statistics were performed using Matlab (MathWorks) with significance level set at 0.05. For each PSR group, non-parametric Wilcoxin signed-rank tests were used to test for differences in joint moment and AP impulses in late single-leg stance and pre-swing between each hemiparetic subject in the group and the average data of the control subjects walking at matched speed to that subject (paretic leg vs. control, nonparetic leg vs. control) based on functional walking status (Table 2.1). For variables that were significantly different from controls in all PSR groups, a nonparametric Kruskal-Wallis test with rank sums tests post hoc analyses to test for differences between each PSR group and their speed matched controls.

**Table 2.1:** For all statistical analyses, household hemiparetic walkers were matched with controls walking at 0.3 m/s, limited community walkers with controls walking at 0.6 m/s, and community walkers with controls walking at 0.9 m/s.

<table>
<thead>
<tr>
<th>Functional Walking Status</th>
<th>Hemiparetic Speed</th>
<th>Matched Control Speed</th>
<th>Total Number of Hemiparetic Subjects (low, symmetric, high PSR)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Household</td>
<td>&lt; 0.4 m/s</td>
<td>0.3 m/s</td>
<td>29 (4, 4, 21)</td>
</tr>
<tr>
<td>Limited Community</td>
<td>0.4 – 0.8 m/s</td>
<td>0.6 m/s</td>
<td>19 (4, 11, 4)</td>
</tr>
<tr>
<td>Community</td>
<td>&gt; 0.8 m/s</td>
<td>0.9 m/s</td>
<td>6 (1, 2, 3)</td>
</tr>
</tbody>
</table>
RESULTS

Nine hemiparetic subjects were in the low PSR group (i.e., shorter paretic than non-paretic steps), 17 subjects walked with a symmetric PSR, and 29 subjects were in the high PSR group (i.e., longer paretic than non-paretic steps).

All PSR groups had significantly different paretic leg AP impulses compared to speed-matched controls during paretic leg stance (Fig. 2.1). The high PSR group was most unable to generate propulsion from the paretic leg; during paretic late single-leg stance both the high and symmetric PSR groups generated less (p = 0.000, 0.031) and the low PSR group generated more (p = 0.013) paretic leg AP impulse compared to speed-matched controls. During paretic pre-swing all groups had significantly less AP impulse compared to controls (p = 0.000, 0.000, 0.002). During both paretic late single-leg stance and pre-swing the high PSR group had a significantly larger deviation between paretic leg AP impulse and controls than both the low (p = 0.001, 0.005) and symmetric (p = 0.000, 0.000) groups. During nonparetic late single-leg stance the low PSR group had less (p = 0.004) and the high PSR group had more (p = 0.041) nonparetic leg AP impulse than controls. During nonparetic pre-swing only the low PSR group had a significantly different nonparetic leg AP impulse compared to controls (reduced, p = 0.002).

All PSR groups had less paretic leg plantarflexor moment impulse compared to controls walking at similar speed (Fig. 2.2). In the high PSR group this difference was significant in both paretic late single-leg stance (p < 0.001) and pre-swing (p < 0.001). In the symmetric PSR group this difference was significant in paretic late single-leg stance (p = 0.015) and approached significance in paretic pre-swing (p = 0.068). In the low PSR group this difference approached significance in both paretic late single-leg stance (p = 0.055) and pre-swing (p = 0.055). The deviation from controls was greater in the high PSR group than both the low and symmetric group (p = 0.038 and 0.004) during late
single-leg stance. Therefore the hypothesis that hemiparetic subjects with longer paretic than nonparetic steps would show reduced paretic leg plantarflexor moment was supported. In addition, compared to controls walking at a similar speed the high PSR group had significantly more nonparetic leg plantarflexor moment impulse (Fig. 2.2, \( p = 0.007 \)) and hip flexor moment impulse (Fig. 2.4, \( p = 0.006 \)) during late single-leg stance as well as a knee extensor versus flexor moment impulse (Fig. 2.3) that approached significance (\( p = 0.065 \)) during late single-leg stance and was significant (\( p = 0.027 \)) during pre-swing.

There were several significant joint moment impulse differences in the symmetric PSR group compared to speed-matched controls. Thus the hypothesis that subjects with symmetric steps would have similar joint moment impulses as speed-matched controls was not supported. In addition to a reduced paretic leg plantarflexor moment impulse (Fig. 2.2), the symmetric PSR group had a significantly larger paretic leg hip flexor moment impulse during both paretic late single-leg stance and pre-swing (Fig. 2.4, \( p = 0.017, 0.003 \)), a larger nonparetic leg plantarflexor moment impulse (Fig. 2.2, \( p = 0.025 \)) and hip flexor moment impulse (Fig. 2.4, \( p = 0.017 \)) during nonparetic late single-leg stance, and an increase in nonparetic leg hip flexor moment impulse that approached significance (Fig 2.4, \( p = 0.055 \)) during nonparetic pre-swing.

The low PSR group had no other significant joint moment impulse differences compared to speed-matched controls. Therefore the hypothesis that subjects with shorter paretic than nonparetic steps would have reduced paretic leg hip flexor moment impulses compared to speed-matched controls was not supported.
Figure 2.1: AP impulse for control (yellow, average of right and left legs), paretic leg (green) and nonparetic leg (red) in late single-leg stance and pre-swing phases. * denotes a significant difference relative to the controls, # denotes significant a significant difference between PSR groups; all p<0.05.

Figure 2.2: Ankle (plantarflexor positive) joint moment impulses for control (yellow, average of right and left leg), paretic leg (green), and nonparetic leg (red) in their respective late single-leg stance and pre-swing phases. * and † denote significant difference relative to the controls, p<0.05 and p<0.1; # denotes a significant difference between PSR groups, p<0.05.
Figure 2.3: Knee (extensor positive) joint moment impulses for control (yellow, average of right and left leg), paretic leg (green), and nonparetic leg (red) in their respective late single-leg stance and pre-swing phases. * and † denote significant difference relative to the controls, p<0.05 and p<0.1.

Figure 2.4: Hip (flexor positive) joint moment impulses for control (yellow, average of right and left leg), paretic leg (green), and nonparetic leg (red) in their respective late single-leg stance and pre-swing phases. * and † denote significant difference relative to the controls, p<0.05 and p<0.1.
DISCUSSION

The overall goal of this study was to determine if step length asymmetry in post-stroke hemiparetic subjects is indicative of underlying differences in joint moments during walking to gain insight into the observed impairments and compensatory strategies. All hemiparetic subjects were found to have paretic leg plantarflexor impairment (i.e., reduced plantarflexor moment impulse), with the degree of plantarflexor impairment being related to the asymmetry group. In addition, PSR groups used different compensatory strategies to overcome this impairment: the high PSR group relied on the nonparetic leg and the symmetric group relied on a bilateral hip flexor strategy.

Paretic leg plantarflexor impairment is consistent across PSR groups

Paretic leg plantarflexor impairment is common among post-stroke hemiparetic subjects (Chen and Patten 2008; Lamontagne et al., 2007; Olney et al., 1994; Turns et al., 2007) and our study suggests that this impairment occurs in all step length asymmetry groups. The level of impairment, however, depends on step length asymmetry as the high PSR group had a greater paretic leg plantarflexor moment reduction compared to speed-matched controls than both the symmetric and low PSR groups. Consistent with previous studies that found the plantarflexors to be important for forward propulsion (Liu et al., 2006; McGowan et al., 2008; Neptune et al., 2001), the reduction was accompanied by a decrease in propulsion compared to speed-matched controls. Previous studies examining step length asymmetry have found both reduced paretic leg plantarflexor strength (Lin et al., 2006) and peak torque (Hsu et al., 2003) result in a greater asymmetry. These studies defined step length asymmetry using an absolute value of the deviation from symmetry and were unable to differentiate between directions of asymmetry. However, most subjects walked with longer paretic than nonparetic steps (i.e. high PSR). Therefore, their
results are consistent with ours that show a greater paretic leg plantarflexor moment impulse reduction in high PSR subjects than symmetric subjects. To our knowledge no other study has found a difference in paretic leg plantarflexor impairment between subjects who walk with different directions of step length asymmetry. This is likely because studies examining step length asymmetry in the hemiparetic subjects include fewer subjects who walk with shorter paretic than nonparetic steps (i.e. low PSR) or did not differentiate subjects based on PSR. Our study shows that paretic leg plantarflexor impairment occurs across all asymmetry groups and the degree of impairment depends on the direction of step length asymmetry.

*PSR is an indicator of compensatory mechanisms*

To compensate for the impaired paretic leg plantarflexors, the high PSR group relied primarily on the nonparetic leg. Compared to speed-matched controls, the nonparetic leg plantarflexor moment was significantly increased during late single-leg stance. This was accompanied by increased nonparetic leg AP impulse, which is consistent with the plantarflexors as main contributors to forward propulsion (Liu et al., 2006; McGowan et al., 2008; Neptune et al., 2001). By using the nonparetic leg plantarflexors to increase forward propulsion, the center-of-mass moves further forward while the paretic leg is in swing, thus increasing the paretic step length (Varraine et al., 2000). In addition, the high PSR group had a knee extensor moment during both nonparetic pre-swing and late single-leg stance. The knee extensors have previously been shown to contribute to propulsion in both mid to late-stance (Kepple et al., 1997) and pre-swing (Peterson et al., 2010a). Similar to the nonparetic leg plantarflexors, the nonparetic leg knee extensors likely increase the paretic step length due to increased propulsion.
These results are consistent with Hsu et al. (2003) who found the strength of the nonparetic leg knee extensors correlates positively with the paretic step length.

To compensate for the impaired paretic plantarflexors, the symmetric PSR group altered other joint moments in order to walk symmetrically. Therefore, the hypothesis that this group would exhibit similar joint moments as non-impaired controls was not supported. Instead, symmetric walkers appeared to utilize a bilateral hip strategy to compensate for the impaired paretic plantarflexors. This strategy utilizes the hip flexor muscles to accelerate the leg forward during pre-swing and swing (Mueller et al., 1994) and is commonly used to overcome plantarflexor weakness (Milot et al., 2007; Mueller et al., 1994; Nadeau et al., 1999; Sadeghi et al., 2001). While increased hip flexor moments act to increase swing initiation, a consequence is a reduction in propulsion as it acts to offload the leg and decrease the AP GRF impulse (Peterson et al., 2010a). However, there was no significant reduction in nonparetic leg propulsion, which was likely due to the increased nonparetic leg plantarflexor output. Paretic leg propulsion, on the other hand, was decreased which is consistent with both the reduction in paretic leg plantarflexor output and increase in hip flexor output. Based on these results, symmetric PSR subjects seem limited primarily by paretic leg plantarflexor impairment.

The AP impulses found in the low PSR group were consistent with our suggestion that these subjects are unable to generate sufficient pre-swing energy to the leg due to impaired paretic plantarflexors and hip flexors. The reduced paretic AP impulse seen during pre-swing, a result of the reduced paretic plantarflexor moment impulse, would act to decrease the energy delivered to the leg and result in shorter paretic swing time and step length. This shorter swing time would also explain the significantly reduced nonparetic leg AP impulse. Unfortunately, no other significant differences in joint moment impulses consistent with this hypothesis were found. This is likely due to the
smaller number of low PSR subjects combined with the high inter-subject variability. Further study including additional low PSR subjects is needed to elucidate any underlying joint compensation patterns in this group.

Limitations

A limitation of this study is that data was collected as subjects walked on a treadmill and previous studies have shown differences in treadmill and overground walking stride lengths (Bayat et al., 2005; Tesio and Rota 2008). However, rehabilitation using treadmill supported modalities (e.g., Hesse et al., 1995; Hornby et al., 2008) is common and thus understanding walking biomechanics on a treadmill is directly applicable to these rehabilitation methods. In addition, a recent study found no fundamental difference in AP impulses between overground and treadmill walking (Goldberg et al., 2008) and treadmill walking reveals the same motor control impairments in hemiparetic walking (Kautz et al., 2012). Therefore the treadmill should have a minimal effect on the results. Another limitation was that data was collected from subjects while walking without an assistive device regardless of whether they normally used one. However, if the overall goal is to improve walking ability such that the patient no longer has to walk with an assistive device, then understanding the impairments that are present when walking without an assistive device is important in developing rehabilitation strategies.

Conclusions

Paretic leg plantarflexor impairment (i.e., reduced net plantarflexor moment impulse during walking) consistently occurs in all post-stroke hemiparetic subjects regardless of step length asymmetry. Thus clinicians should focus on improving the
paretic leg plantarflexor output in all subjects during gait rehabilitation. The direction of asymmetry can be used to understand both the degree of paretic leg plantarflexor impairment and the compensatory mechanism used. Those subjects who walk with symmetric steps are able to compensate for plantarflexor impairment using similar output from both legs. Those subjects who walk with longer paretic than nonparetic steps, on the other hand, typically rely on the nonparetic leg, and thus improving paretic leg output in these subjects may improve their walking symmetry.
Chapter 3
Three-Dimensional Modular Control of Walking

INTRODUCTION

Previous studies suggest that non-impaired walking may be achieved using a reduced set of neural control elements or modules (e.g., Cappellini et al., 2006; Clark et al., 2010; Ivanenko et al., 2004). Recent 2D modeling and simulation studies examined whether these modules are structured to perform task-specific sagittal-plane biomechanical functions (i.e., support, forward propulsion and leg swing) (McGowan et al., 2010; Neptune et al., 2009). In these studies, four modules identified from electromyography (EMG) activity in healthy adults (Clark et al., 2010) were used to co-excite multiple muscles in a musculoskeletal model. Each module was associated with specific biomechanical functions that resulted in a well-coordinated walking pattern. Module 1 (hip and knee extensors) contributed to body support in early stance while Module 2 (ankle plantarflexors) contributed to body support and forward propulsion in late stance. Module 3 (tibialis anterior and rectus femoris) decelerated the leg in early and late swing while generating energy to the trunk throughout swing while Module 4 (hamstrings) acted to absorb leg energy (i.e. decelerate it) in late swing while increasing leg energy in early stance to provide forward propulsion. Post-hoc analysis revealed a fifth module (Module 5: hip flexors) that accelerated the leg forward in pre- and early swing. However, since the model was constrained to the sagittal plane, the contributions of each module to mediolateral (ML) balance control and contralateral leg swing (due to energy transfer through the pelvis) could not be assessed.

Walking is a 3D movement in which non-sagittal plane biomechanical subtasks must be effectively executed. In addition to providing body support and forward
propulsion, muscles must also control ML balance (Perry 1967; Winter 1995) by redirecting center-of-mass (COM) frontal plane movement. A recent 3D simulation analysis found the muscles that contribute significantly to body support and forward propulsion also regulate ML COM acceleration (Pandy et al., 2010). Furthermore, previous 3D walking simulations found that hip muscles in the ipsilateral leg are important for contralateral leg swing (Arnold et al., 2007; Hall et al., 2011; Peterson et al., 2010b). It is unclear whether a similar simple modular control framework can capture 3D walking when muscles contributing to ML balance control and contralateral leg swing are considered.

The purpose of this study was to use a 3D musculoskeletal model and forward dynamics simulation of healthy walking to gain a comprehensive view of modular control of human walking. Specifically, the modular contributions to the 3D ground reaction forces (GRFs) and power generation, absorption and transfer among body segments were analyzed. It was hypothesized that (1) a similar five module framework would be capable of driving a 3D model of walking, (2) modules important for support and forward propulsion would also be important for ML balance (e.g., Modules 1 and 2) and (3) modules that include the hip muscles (e.g., Modules 1, 4 and 5) would play a prominent role in controlling contralateral leg swing.

METHODS

Musculoskeletal Model

A previously described 3D musculoskeletal model (Peterson et al., 2010b) with 23 degrees-of-freedom was developed using SIMM (Musculographics, Inc.) and included rigid segments representing the trunk, pelvis and two legs (thigh, shank, talus, calcaneus and toes). The pelvis had six degrees-of-freedom (3 translations and 3 rotations) with the
trunk and hip joints modeled using spherical joints. The knee, ankle, subtalar and metatarsophalangeal joints were modeled as single degree-of-freedom revolute joints. The foot-ground contact forces were modeled with 31 independent visco-elastic elements attached to each foot (Neptune et al., 2000). Passive torques representing forces applied by ligaments, passive tissue and joint structures were applied at each joint (Anderson 1999). The dynamical equations-of-motion were generated using SD/FAST (PTC).

The model was driven by 38 Hill-type musculotendon actuators per leg (five smaller foot muscles from Peterson et al. 2010 were excluded in our model due to their minimal contributions to the biomechanical subtasks). Five previously identified muscle activation modules (for details see Neptune et al., 2009) describing time-varying activation patterns relative to the gait cycle were used as the muscle excitation inputs (Fig. 3.1). Modules 1-4 were derived from experimentally collected EMG data (see Experimental Data). Muscles without recorded EMGs but with similar anatomical arrangement, biomechanical function and/or EMG activity were included in these modules. Module 1 included the 3-component vastus (VAS), rectus femoris (RF), 3-component gluteus maximus (GMAX), 3-component gluteus minimus (GMIN), and 3-component gluteus medius (GMED). Module 2 included the soleus (SOL), medial and lateral gastrocnemius (GAS), tibialis posterior (TP), and flexor digitorum longus (FD). Module 3 included RF, tibialis anterior (TA), and extensor digitorum longus (ED). Module 4 included the medial hamstrings (semitendinosus = SM, semitendinosus = ST, and gracilis, GRAC), lateral hamstrings (biceps femoris long head, BFllh), and the biceps femoris short head (BFsh). Muscles within each module received the same excitation pattern and timing, but the magnitude was allowed to vary between muscles. Muscles associated with Module 5 received a bimodal excitation pattern (Hall et al., 2011). Module 5 included the iliacus and psoas (IL), pectinius (PECT), and sartorius (SAR). The
Other Muscles (Fig. 3.1) unassociated with a module also each received a bimodal excitation pattern, which allows the flexibility for two distinct peaks, one broader peak, or only one peak (while bimodal is allowed it might not be used). These muscles included the 3-component adductor magnus (AM), adductor longus (AL), adductor brevis (AB), quadratus femoris (QF), gemellus (GEM), piriformis (PIRI), and tensor fascia lata (TFL). Muscle contraction dynamics were governed by Hill-type muscle properties (Zajac 1989) and muscle activation dynamics were modeled using a non-linear first-order differential equation (Raasch et al., 1997). Polynomial equations were used to estimate musculotendon lengths and moment arms (Menegaldo et al., 2004).

Dynamic Optimization

A 3D walking simulation of 120% of a gait cycle was generated using a simulated annealing algorithm (Goffe et al., 1994) that fine-tuned the muscle excitation patterns and initial joint velocities such that the difference between the simulated and experimentally measured walking data (see below) and muscle stress was minimized. Quantities included in the cost function were muscle stress and the differences in the pelvis translations, trunk, pelvis, hip, knee and ankle joint angles and GRFs. Each bimodal excitation pattern had six optimization parameters (onset, offset and magnitude for the two modes) and each module pattern had two optimization parameters for timing (onset, offset) and a magnitude parameter for each muscle within the module. Initial parameter values were based on experimental data (joint velocities) and a previously optimized simulation (excitation parameters). To improve the tracking optimization convergence, tracking torques were applied at each joint to drive them towards desired experimental kinematic trajectories using proportional control (see Appendix C). These torques were also included in the cost function in order to drive their magnitudes to zero.
Experimental Data

The experimental data used were a subset of the data in Clark et al. (2010). Kinematic, GRF and EMG data were collected from 14 healthy adults (63.1 ± 9.1 years; 2 male) as they walked for 30s at 1.2 m/s on an ADAL split-belt treadmill (Techmachine). All subjects provided informed consent prior to data collection. Using non-negative matrix factorization (Clark et al., 2010), modules were identified from EMG data collected using bipolar Ag-AgCL surface electrodes from the tibialis anterior, soleus, medial gastrocnemius, vastus medialis, rectus femoris, medial hamstrings, lateral hamstrings and gluteus medius of each leg using a telemetered EMG acquisition system (Konigsberg Instruments). 3D body-segment kinematics were collected at 100 Hz and GRF and EMG data were collected at 2000 Hz using Vicon Workstation v4.5 software. EMG signals were high-pass filtered with a fourth-order Butterworth filter (40 Hz), demeaned, rectified and low-pass filtered with a fourth-order Butterworth filter (4 Hz). The GRFs were filtered at 20 Hz. Kinematics were low-pass filtered with a fourth-order Butterworth filter with a cutoff frequency of 6 Hz. All EMG, GRF and kinematic data were time normalized to 100% of the gait cycle. Kinematic and GRF data were averaged across subjects for the simulation tracking.

Simulation Analysis

Analyses were performed on the last 100% of the simulation (a full gait cycle starting at heel-strike) to allow the initial transients to decay. To quantify contributions of each module to the biomechanical functions of body support (vertical GRF), forward propulsion (anterior-posterior, AP GRF), ML balance control (ML GRF) and ipsilateral and contralateral leg swing, individual muscle contributions to the GRFs and body segment mechanical energetics were quantified using previously described GRF decomposition and body segment power analyses (Neptune et al., 2004). The contribution
of each module to each biomechanical function was found by summing the individual muscle contributions from those muscles associated with that module. For RF, its contribution was scaled according to the relative contribution of the two modules to the muscle’s total excitation.

RESULTS

Using the five previously identified modules as excitation inputs (plus excitation of the remaining muscles, Fig. 3.1), the simulation emulated well the group averaged walking data with average kinematic and GRF deviations of $4.6^\circ$ (experimental SD = $6.2^\circ$) and 4.3% body weight (BW, experimental SD = 2.9% BW), respectively (Fig. 3.2, Table 3.1). All tracking torques were eliminated except for small torques remaining for pelvis rotation (peak value of 3.9 Nm, average value of 0.3 Nm), which had minimal contributions to all walking subtasks.

Module 1 (gluteus muscles, vasti and rectus femoris) provided body support (positive vertical GRF, Fig. 3.3b) and acted to decelerate the body (negative AP GRF, Fig. 3.3a) during the first half of stance and accelerated the body medially (positive ML GRF, Fig. 3.3c) throughout stance. Module 1 also transferred energy from the ipsilateral leg to the contralateral leg from early to mid-stance (Fig. 3.4). Module 2 (plantarflexors) provided body support throughout stance (Fig. 3.3b), decelerated the body during the first half of stance (Fig. 3.3a), and provided forward propulsion (positive AP GRF, Fig. 3.3a) while accelerating the body laterally (negative ML GRF, Fig. 3.3c) in the second half of stance. Module 3 (ankle dorsiflexors and rectus femoris) absorbed power from the ipsilateral leg during early stance and transferred that energy to the trunk (Fig. 3.4). Module 3 also helped control ipsilateral leg swing by accelerating the leg forward during swing (i.e. generated power to the leg). Module 4 (hamstrings) provided forward
propulsion (Fig. 3.3a) and accelerated the body laterally during the first half of stance (Fig. 3.3c). Module 4 also delivered much energy to the ipsilateral leg in stance while delivering some energy to the contralateral leg prior to its swing through both power generation and power transfer from the trunk (Fig. 3.4). Finally, Module 4 also acted to decelerate the ipsilateral leg in late swing (Fig. 3.4). Module 5 (hip flexors) facilitated leg swing by generating positive power to the ipsilateral leg during swing (Fig. 3.4). Those muscles not associated with a module also contributed to walking subtasks (Figs. 3.3, 3.4). AM generated a lateral GRF in the first half of stance (Fig. 3.5a) while generating energy to the contralateral leg and trunk and absorbing energy from the ipsilateral leg (Fig. 3.5b). AL, AB and QF generated energy to the ipsilateral leg during early swing but decelerated the leg through energy transfer later in swing to assist the hamstrings (Fig. 3.5b). The hip abductors (GEM, PIRI, TFL) generated a medial GRF through mid-stance (Fig. 3.5a) while transferring power from the contralateral leg to the ipsilateral leg and trunk (Fig. 3.5b).
Table 3.1: The average difference between the experimental and simulated kinematic angles and GRFs compared to the average standard deviation (SD) of the experimental data for the original five module simulation and the final six module simulation.

<table>
<thead>
<tr>
<th>Kinematic Angles (%)</th>
<th>5 Modules</th>
<th>6 Modules</th>
<th>SD</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pelvis Obliquity</td>
<td>4.1</td>
<td>4.7</td>
<td>3.6</td>
</tr>
<tr>
<td></td>
<td>Rotation</td>
<td>5.0</td>
<td>3.7</td>
</tr>
<tr>
<td></td>
<td>Tilt</td>
<td>1.6</td>
<td>1.5</td>
</tr>
<tr>
<td>Trunk Obliquity</td>
<td>3.4</td>
<td>3.3</td>
<td>6.0</td>
</tr>
<tr>
<td></td>
<td>Rotation</td>
<td>4.4</td>
<td>4.5</td>
</tr>
<tr>
<td></td>
<td>Tilt</td>
<td>1.6</td>
<td>1.8</td>
</tr>
<tr>
<td>Ipsilateral leg</td>
<td>Hip adduction</td>
<td>5.6</td>
<td>4.6</td>
</tr>
<tr>
<td></td>
<td>Hip rotation</td>
<td>5.4</td>
<td>3.0</td>
</tr>
<tr>
<td></td>
<td>Hip flexion</td>
<td>5.5</td>
<td>4.6</td>
</tr>
<tr>
<td></td>
<td>Knee flexion</td>
<td>8.5</td>
<td>7.5</td>
</tr>
<tr>
<td></td>
<td>Ankle angle</td>
<td>6.2</td>
<td>5.4</td>
</tr>
<tr>
<td>Contralateral leg</td>
<td>Hip adduction</td>
<td>4.2</td>
<td>5.2</td>
</tr>
<tr>
<td></td>
<td>Hip rotation</td>
<td>5.3</td>
<td>3.2</td>
</tr>
<tr>
<td></td>
<td>Hip flexion</td>
<td>4.8</td>
<td>5.2</td>
</tr>
<tr>
<td></td>
<td>Knee flexion</td>
<td>4.7</td>
<td>5.9</td>
</tr>
<tr>
<td></td>
<td>Ankle angle</td>
<td>4.0</td>
<td>3.9</td>
</tr>
<tr>
<td>I ipsilateral leg</td>
<td>AP GRF</td>
<td>3.3</td>
<td>4.1</td>
</tr>
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<td></td>
<td>Vertical GRF</td>
<td>8.2</td>
<td>10.3</td>
</tr>
<tr>
<td></td>
<td>ML GRF</td>
<td>2.2</td>
<td>2.0</td>
</tr>
<tr>
<td>Forces (%BW)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Contralateral leg</td>
<td>AP GRF</td>
<td>2.8</td>
</tr>
<tr>
<td></td>
<td>Vertical GRF</td>
<td>7.9</td>
<td>8.8</td>
</tr>
<tr>
<td></td>
<td>ML GRF</td>
<td>1.6</td>
<td>1.8</td>
</tr>
</tbody>
</table>

Average Angle Difference (°) | 4.6 | 4.3 | 12.3 |
Average GRF Differences (%BW) | 4.3 | 5.0 | 5.8 |
Figure 3.1: Experimentally derived module patterns (left column, Modules 1-4) and the corresponding muscles excited by each module (rows). Module 5 and Other Muscles were each controlled using bimodal patterns.
Figure 3.2: Tracking results for the simulation controlled by five modules (plus excitation of remaining muscles). The simulated joint angles and ground reaction forces (solid lines) agree well with the experimental data (gray bars). The gray bars represent experimental means ± 2 SD.
Figure 3.3: Module contributions to the (a) anterior-posterior (AP), (b) vertical and (c) mediolateral (ML) ground reaction forces. Total is the sum of all muscles.
Figure 3.4: Mechanical power delivered to the trunk, ipsilateral and contralateral leg by each module. Total represents the sum of the mechanical power delivered to all segments. Positive and negative power values indicate a module acts to accelerate or decelerate the segments, respectively. The alternating shaded regions represent different phases of the gait cycle: 1 – 1st double support/contralateral pre-swing, 2 – first half of ipsilateral single support/contralateral swing, 3 – second half of ipsilateral single support/contralateral swing, 4 – ipsilateral pre-swing/contralateral 1st double support, 5 – first half of ipsilateral swing/contralateral single support, and 6 – second half of ipsilateral swing/contralateral single support.
Figure 3.5: Contributions from individual muscles not controlled by a module to (a) ML GRF and (b) power transfer among segments. The alternating shaded regions represent different phases of the gait cycle (see Fig. 3.4 caption).
DISCUSSION

Recent evidence supports the idea that movements are controlled through a combination of co-excitation patterns, or modules, organized around specific biomechanical functions (e.g., Clark et al., 2010; d'Avella et al., 2003; Davis and Vaughan 1993; Ivanenko et al., 2004; Neptune et al., 2009; Ting and Macpherson 2005). For example, in kicking frogs, modules have been associated with controlling kick direction (d'Avella et al., 2003) and in standing cats, modules have been associated with different hind-limb force components (Ting and Macpherson 2005). In human walking, modules have been temporally associated with particular regions of the gait cycle (Cappellini et al., 2006; Davis and Vaughan 1993; Ivanenko et al., 2004), suggesting they provide specific biomechanical functions.

When a similar five module framework was used for 3D walking, the contributions of each module to forward propulsion and body support were consistent with the previous 2D analysis (Neptune et al., 2009). Modules were also found to work synergistically throughout stance to control ML balance through their contributions to the ML GRFs. During most of stance, the net GRF is directed medially (Fig. 3.3c), first acting to decelerate the laterally moving COM in order to maintain dynamic balance and prevent falling (e.g., prevent the COM from moving beyond the base of support) and then to help shift the body weight to the contralateral side in preparation for the next stance phase. This medial GRF is generated primarily by Module 1, which dominates the lateral contributions from the hip adductors (primarily AM, Fig. 3.5a) and Module 4 (Fig. 3.3c). The medial GRF from Module 1 is generated by the gluteus muscles (GMED, GMIN and GMAX), which dominate the small offsetting lateral contribution from VAS (Fig. 3.6). These opposing contributions of muscles within Module 1 are consistent with a previous simulation analysis that found similar opposing COM accelerations (Pandy et al., 2010).
Figure 3.6: ML GRF contributions from individual muscles in Module 1.

This co-activation is needed for body support. Thus, more demanding ML tasks (e.g., turning) may reweight the relative muscle contributions within Module 1 to emphasize one of these opposing contributions to a greater degree (e.g., similar to the reweighting of Module 2 muscles that were found when body weight and mass were independently manipulated, McGowan et al., 2010). Near the end of stance, the direction of the net GRF switches from medial to lateral as Module 2’s (plantarflexors) lateral contribution dominates the medial contributions by Module 1 and the hip abductor muscles (Fig. 3.3c, 3.5a). In support of the hypotheses, Modules 1 and 2 appear to be the primary modules contributing to ML balance control, which is consistent with a previous simulation analysis that found VAS, SOL, GAS and GMED are the primary contributors to ML COM acceleration (Pandy et al., 2010). The hip ab/adductors, primarily AM in early stance, also play an important role in ML balance control, which suggests that the current five module framework is insufficient for 3D walking and that an additional neural control element may be needed to control hip ab/adduction.

In addition to controlling ipsilateral leg swing, modules also contributed to contralateral leg swing. In partial support of the hypothesis that modules containing the hip muscles would contribute to contralateral leg swing, Modules 1 and 4 generated energy to the contralateral leg (i.e. accelerated the leg into swing): Module 1 in early
contralateral swing and Module 4 in contralateral pre-swing and swing (Figs. 3.4, 3.7). The magnitude of the power delivered by these modules to the contralateral leg is of similar magnitude as the power delivered to the ipsilateral leg by Module 5 during ipsilateral swing (Fig. 3.4), which highlights the important role Modules 1 and 4 have in controlling contralateral leg swing. While the energy transfer to the contralateral leg was not observed in Neptune et al. (2009), the present 3D model included additional degrees-of-freedom at the trunk, pelvis and hip, allowing for more energy transfer from the stance leg to the contralateral swing leg through pelvis rotation. These results are consistent with previous modeling studies that found power transfer to the contralateral leg from several of the muscles within these modules (e.g. GMED, GMIN, hamstrings) (Arnold et al., 2007; Hall et al., 2011; Peterson et al., 2010b). These results are also consistent with previous studies deriving modules from bilateral EMG (Dominici et al., 2011; Olree and Vaughan 1995). These studies identified a module active primarily around contralateral swing that includes several of the ipsilateral muscles that we include in our Modules 1 and 4 (e.g. GMED, VAS, TFL) and contralateral muscles that we include in our Module 5 (e.g. AL) that are important for leg swing. These results also provide support for the importance of bilateral coordination during walking.

The remaining muscles (Other Muscles), all hip ab/adductor muscles, were important for ML balance control (see above) and both ipsilateral and contralateral leg swing. Some hip adductors (AL, AB and QF) accelerated the ipsilateral leg forward during swing, similar to Module 5, and then decelerated the leg in late swing prior to heel-strike (Fig. 3.5b). The remaining hip adductor (AM) transferred energy from the ipsilateral leg to the contralateral leg prior to contralateral swing (Fig. 3.5b). One question that naturally arises is whether these muscle contributions could be mapped into existing modules. To address this question, post-hoc analyses were performed in which
the remaining muscles were placed into different modules. Simulations were generated with each of the remaining muscles placed into one of the original five modules rather than allowing them to be independently controlled. The hip abductors (GEM, PIRI and TFL) were placed into Module 1 due to the similarity in their optimized excitation patterns with Module 1. Similarly, AL, AB and QF were placed in Module 5 with the hip flexors due to their similarity in excitation timing to Module 5. The optimized excitation pattern for AM had similarities to both Module 1 and 4 patterns, therefore two simulations were generated with AM placed in either Module 1 or 4. Neither optimization eliminated the tracking torques while successfully reproducing the walking pattern. Thus, the hypothesis that a five module framework can successfully drive a 3D model was not supported.

The primary source of difficulty with using the five module framework was eliminating the hip adduction/abduction tracking torques, which suggests that an additional module may be critical for non-sagittal hip motion control. Since AM was found to be a powerful contributor to ML balance control (Fig. 3.5a), another optimization was performed in which a sixth module was used to control all three components of AM. The
Figure 3.8: Module 6 (AM) (a) generated a medial GRF during the beginning of stance and (b) decelerated the ipsilateral leg (negative power) during late swing and early stance while generating energy to the contralateral leg and trunk (positive power). The alternating shaded regions represent different phases of the gait cycle (see Fig. 3.4 caption).

optimization found that these six modules were able to successfully reproduce the 3D walking pattern with all tracking torques eliminated except for minimal torques for pelvis rotation (mean torque of 0.4 Nm, mean joint angle error of 4.3°, experimental SD of 6.2°, and mean GRF error of 5.0% BW, experimental SD of 2.9% BW, Table 3.1). The biomechanical functions of Modules 1-5 remained the same while the new Module 6 (AM) generated a laterally directed GRF during early stance (Fig. 3.8a) and transferred
energy from the ipsilateral leg to the contralateral leg prior to contralateral swing (Fig. 3.8b).

A potential limitation is that modules were derived from only 8 muscles per leg while the model was driven by 38 per leg. Remaining muscles were each placed into a module based on results from previous studies (see Appendix B). However, a number of muscles in our model have not been included in any previous experimental module study (with many of these being muscles that contribute to non-sagittal plane motion). Future research recording EMG from a larger dataset of muscles is needed to support our results. Given that an arrangement of all muscles into a five module framework could not be found, it seems likely that at least one additional module primarily composed of hip abductor or adductor activity exists. In addition, except for RF, each muscle was controlled using only one module even though each muscle is excited to some degree by each module (e.g., Clark et al., 2010). An area of future work will look at the effect of allowing each module to contribute to each muscle’s excitation pattern. However, assuming that each muscle has been placed in its correct dominant module, the overall modular function should remain unchanged with only slight differences in the magnitude and timing.

In conclusion, while a five module framework can successfully simulate 2D walking (Neptune et al., 2009) it does not appear to provide the additional control needed for 3D walking. The results of this study suggest that a sixth module organized around the subtasks of ML balance control and contralateral leg swing can provide the additional non-sagittal plane control. The fact that the simulation emulated remarkably well the measured kinematics and GRFs using a reduced set of neural control elements provides further evidence that modular control strategies organized around task-specific biomechanical functions may be used to control complex human movements. Future
work will be directed at understanding how execution of these biomechanical functions is altered in neurologically impaired populations due to impaired modular control.
Chapter 4

The Influence of Merged Muscle Excitation Modules on Post-Stroke Hemiparetic Walking Performance

INTRODUCTION

Stroke is a leading cause of long-term disability in the United States that often leaves survivors with various levels of hemiparesis affecting their mobility. These individuals typically walk at slower speeds (e.g., Perry et al., 1995) due to impaired muscle coordination (Den Otter et al., 2007; Turns et al., 2007). However, the type of coordination impairments varies among individuals (e.g., De Quervain et al., 1996; Knutsson and Richards 1979; Shiavi et al., 1987). Recent studies have suggested that complex muscle activity during walking may be generated using a reduced neural control strategy organized around the co-excitation of multiple muscles, or modules (e.g., Cappellini et al., 2006; Clark et al., 2010; Ivanenko et al., 2004). Furthermore, the coordination impairments observed in post-stroke hemiparetic subjects manifests in different modular patterns (Clark et al., 2010). Recent studies have proposed analyzing modules as a promising technique to identify and monitor muscle coordination impairments and their influence on locomotor performance (Clark et al., 2010; Safavynia et al., 2011). Understanding how different module compositions influence specific measures of walking performance can potentially provide clinicians with detailed information to tailor rehabilitation to a patient’s specific coordination deficits.

Four modules have been identified previously in non-impaired walking (Clark et al., 2010) that each have distinct contributions to important biomechanical functions during walking (e.g., support, forward propulsion, mediolateral balance control and leg swing: Allen and Neptune 2012; McGowan et al., 2010; Neptune et al., 2009). Module 1
(an early stance module with hip abductor and knee extensor activity) and Module 2 (a late stance module with plantarflexor activity) provide support during early and late stance, respectively. These two modules, combined with Module 4 (a late swing into early stance module composed of hamstrings activity) work together to maintain both forward propulsion and mediolateral balance. Modules 4 and 3 (an early swing module with dorsiflexor and biarticular knee extensor activity) are both important for controlling ipsilateral leg swing, while Modules 1 and 4 are both important for controlling contralateral leg swing. Since these four modules synergistically control important biomechanical functions during non-impaired walking, altered module compositions may adversely affect walking performance.

Studies have shown that instead of independently activating the same four modules found during non-impaired walking, post-stroke hemiparetic subjects often utilize a reduced number of modules, with at least one of the modules resembling the merging of multiple non-impaired modules (Clark et al., 2010). As the number of independently activated modules decreases, walking performance (e.g. self-selected speed, bilateral symmetry, etc.) also decreases (Bowden et al., 2010; Clark et al., 2010). A consequence of merged modules may be a reduced ability to successfully generate each biomechanical function, resulting in poor walking performance. Which specific functions are affected likely depends on which modules are merged. For example, within those subjects who could independently activate three modules, two common types of module compositions were found (Clark et al., 2010). One type merged Modules 1 and 2 such that the proximal extensors and plantarflexors were co-activated throughout stance. A second type merged Modules 1 and 4 such that the proximal extensors and hamstrings were co-activated from swing into late stance. It is likely that subjects with these two
types of module compositions would have different neuromotor impairments and benefit from different rehabilitation strategies.

The purpose of this study was to develop muscle-actuated forward dynamics simulations of walking for non-impaired control subjects and two groups of hemiparetic subjects in which different non-impaired modules are merged (Group A: merged Modules 1 and 2; Group B: merged Modules 1 and 4) in order to identify the potential influence of merged modules on post-stroke hemiparetic walking performance (as measured by contributions to specific biomechanical functions of walking). Based on the function of each module in non-impaired walking, we expect that Group A will exhibit deficits in support, forward propulsion and mediolateral balance control while Group B will be primarily impaired in the non-sagittal plane biomechanical functions (e.g., mediolateral balance control and contralateral leg swing). Identifying the relationship between module composition and biomechanical functions of walking will provide evidence-based rationale for rehabilitation programs designed to target the deficits associated with specific module compositions.

METHODS

Experimental Data

The experimental data used were a subset from Clark et al. (2010) (i.e., those that fell into Group A or Group B). Kinematic, GRF and EMG data were collected from 11 chronic post-stroke hemiparetic subjects walking at their self-selected speed. All subjects provided informed consent prior to data collection. 3D body-segment kinematics were collected at 100 Hz and GRF and EMG data were collected at 2000 Hz using Vicon Workstation v4.5 software while subjects walked for 30s on an ADAL split-belt treadmill (Techmachine). Kinematics and GRF data were low-pass filtered with a fourth-order
Butterworth filter with a cutoff frequency of 6 Hz and 20 Hz, respectively. EMG signals were high-pass filtered with a fourth-order Butterworth filter (40 Hz), demeaned, rectified and low-pass filtered with a fourth-order Butterworth filter (4 Hz). All EMG, GRF and kinematic data were time normalized to 100% of the paretic gait cycle. Using non-negative matrix factorization (Clark et al., 2010, see Appendix A for details), modules were identified from EMG data collected using bipolar Ag-AgCL surface electrodes from the tibialis anterior, soleus, medial gastrocnemius, vastus medialis, rectus femoris, medial hamstrings, lateral hamstrings and gluteus medius of each leg using a telemetered EMG acquisition system (Konigsberg Instruments).

Of the these subjects, seven were in Group A and four in Group B. Walking trials of a representative subject from each group (Group A: male, right hemiparesis, age = 57, time post-stroke = 3 years 3 months, self-selected treadmill speed = 0.75 m/s; Group B: male, right hemiparesis, age = 82, time post-stroke = 2 years 5 months, self-selected treadmill speed = 0.70 m/s) were selected for the simulation analysis based on similar walking speeds and minimum difference between kinematics and GRFs compared to their group average data (see Appendix D for details). Both subjects exhibited similar gait asymmetry to their respective group (Table 4.1). The individual gait cycle for each subject with the minimum difference in kinematics and GRFs compared to that subject’s average data was then used to generate the simulations.
Table 4.1: Subject and group average asymmetry measures for the post-stroke hemiparetic module composition groups. The simulated subjects (shaded regions) exhibit asymmetry measures within one standard deviation of the group average values. PSR (paretic step ratio) is a measure of the asymmetry in step lengths and PP (percent paretic propulsion) is a measure of the asymmetry in propulsion generated from the paretic and nonparetic legs. Both measures are defined as the paretic value divided by the total from the paretic and nonparetic leg (a symmetric value is equal to 0.5).

<table>
<thead>
<tr>
<th>Subject</th>
<th>Speed</th>
<th>PSR</th>
<th>PP</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>0.8</td>
<td>0.513</td>
<td>0.266</td>
</tr>
<tr>
<td>2</td>
<td>0.3</td>
<td>0.522</td>
<td>0.193</td>
</tr>
<tr>
<td>3</td>
<td>0.45</td>
<td>0.518</td>
<td>0.128</td>
</tr>
<tr>
<td>4</td>
<td>0.4</td>
<td>0.514</td>
<td>0.054</td>
</tr>
<tr>
<td>5</td>
<td>0.75</td>
<td>0.501</td>
<td>0.382</td>
</tr>
<tr>
<td>6</td>
<td>0.95</td>
<td>0.409</td>
<td>0.539</td>
</tr>
<tr>
<td>7</td>
<td>0.5</td>
<td>0.578</td>
<td>0.191</td>
</tr>
<tr>
<td><strong>Average</strong></td>
<td><strong>0.59</strong></td>
<td><strong>0.508</strong></td>
<td><strong>0.250</strong></td>
</tr>
<tr>
<td><strong>SD</strong></td>
<td><strong>0.24</strong></td>
<td><strong>0.050</strong></td>
<td><strong>0.164</strong></td>
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<th>PSR</th>
<th>PP</th>
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<td>0.7</td>
<td>0.443</td>
<td>0.548</td>
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<tr>
<td>2</td>
<td>0.4</td>
<td>0.409</td>
<td>0.740</td>
</tr>
<tr>
<td>3</td>
<td>0.4</td>
<td>0.661</td>
<td>0.054</td>
</tr>
<tr>
<td>4</td>
<td>0.3</td>
<td>0.512</td>
<td>0.695</td>
</tr>
<tr>
<td><strong>Average</strong></td>
<td><strong>0.45</strong></td>
<td><strong>0.506</strong></td>
<td><strong>0.509</strong></td>
</tr>
<tr>
<td><strong>SD</strong></td>
<td><strong>0.17</strong></td>
<td><strong>0.112</strong></td>
<td><strong>0.314</strong></td>
</tr>
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</table>
Musculoskeletal Model

A previously described 3D musculoskeletal model (see Chapter 3) with 23 degrees-of-freedom was developed using SIMM/Dynamics Pipeline (Musculographics, Inc.) and included rigid segments representing the trunk, pelvis and two legs (thigh, shank, talus, calcaneus and toes). The pelvis had six degrees-of-freedom (3 translations, 3 rotations) with the trunk and hip joints modeled using spherical joints. The knee, ankle, subtalar and metatarsophalangeal joints were modeled as single degree-of-freedom revolute joints. The foot-ground contact forces were modeled with 31 independent viscoelastic elements attached to each foot (Neptune et al., 2000). Passive torques representing forces applied by ligaments, passive tissue and joint structures were applied at each joint (Anderson 1999). The dynamical equations-of-motion were generated using SD/FAST (PTC).

The model was driven by 38 Hill-type musculotendon actuators per leg. The modular excitation patterns identified through NNMF were used as excitation inputs to the corresponding muscles (i.e., within a given module, if the average weighting for an individual muscle was >40% across subjects, then that muscle was excited by that module, Table 4.2). Muscles without recorded EMGs but with similar anatomical arrangement, biomechanical function and/or EMG activity were included in these modules (see Appendix B). Muscles within each module received the same excitation pattern and timing, but the magnitude was allowed to vary between muscles. All other muscles (Table 4.2) were driven using individual bimodal Henning patterns (Hall et al., 2011). Muscle contraction dynamics were governed by Hill-type muscle properties (Zajac 1989) and the activation dynamics were modeled by a first-order differential equation (Raasch et al., 1997). Polynomial equations were used to estimate musculotendon lengths and moment arms (Menegaldo et al., 2004).
Dynamic Optimization

Forward dynamics simulations of 115% of a gait cycle (15% of the gait cycle prior to paretic heel-strike to the next paretic heel-strike) were generated for both hemiparetic groups. A simulated annealing algorithm (Goffe et al., 1994) fine-tuned the muscle excitation patterns and initial joint velocities such that the difference between the simulated and experimentally measured kinematics and GRF walking data were minimized. Quantities included in the cost function were differences in the pelvis translations, trunk, pelvis, hip, knee and ankle joint angles and GRFs. Total muscle stress (muscle force/cross-sectional area of muscle) was also included in the cost function to minimize unnecessary co-contraction. Each bimodal excitation pattern had six optimization parameters (onset, offset and magnitude for the two modes) and each module pattern had two optimization parameters for timing (onset, offset) and a magnitude parameter for each muscle within the module.

To improve the tracking optimization convergence, tracking torques were applied at each joint to drive them towards desired experimental kinematics using proportional control (see Appendix C). The tracking torques were also included in the cost function in order to minimize their magnitudes. The tracking differences, muscle stress and tracking torques were initially weighted equally in the cost function.

Simulation Analyses

Analyses were performed on a previously optimized control simulation (see Chapter 3) and the two hemiparetic simulations for a full gait cycle starting at paretic/left leg heel-strike. The influence of each module was assessed by quantifying the potential of muscles within that module to contribute to forward propulsion (anterior-posterior, AP, GRFs), body support (vertical GRFs), mediolateral balance control (ML GRFs) and leg swing (average power delivered to the leg prior to and during swing) using a perturbation
analysis, similar to previously described GRF decomposition and body segmental power analyses (Neptune et al., 2004). First, the total GRF and segmental powers were calculated at time step $i$. Then, at time step $i-1$, the muscle force of interest was perturbed 1N and the GRFs and segmental powers were recomputed. That muscle’s per unit force contributions to the GRFs and segmental powers were approximated by the difference between the unperturbed and perturbed values. The process was then repeated for each muscle. These per unit force contributions were then scaled by each muscle’s respective experimentally measured module timing pattern to give the potential of a muscle to contribute to each biomechanical function over the gait cycle.
Table 4.2: The 38 individual muscles per leg are combined into 34 groups based on anatomical function. The excitation pattern for each muscle group was either module based or defined by a bimodal Henning pattern. Each of the hemiparetic groups had three modules (M1a-M3a, M1b-M3b) and all muscles within a module received the same timing but the magnitude was allowed to vary.

<table>
<thead>
<tr>
<th>Muscle Name</th>
<th>Muscle Group</th>
<th>Excitation Pattern Type</th>
<th>Group A</th>
<th>Group B</th>
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<tr>
<td>Iliacus</td>
<td>IL</td>
<td>Bimodal</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Psoas</td>
<td>IL</td>
<td>Bimodal</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Adductor Longus</td>
<td>AL</td>
<td>Bimodal</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Adductor Brevis</td>
<td>AB</td>
<td>Bimodal</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Pectineus</td>
<td>PECT</td>
<td>Bimodal</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Quadratus Femoris</td>
<td>QF</td>
<td>Bimodal</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Superior Adductor Magnus</td>
<td>AM</td>
<td>Bimodal</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Middle Adductor Magnus</td>
<td>AM</td>
<td>Bimodal</td>
<td></td>
<td></td>
</tr>
<tr>
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<tr>
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<td>Bimodal</td>
<td></td>
<td></td>
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<tr>
<td>Rectus Femoris</td>
<td>RF</td>
<td>M1a, M2a, M2b, M3b</td>
<td></td>
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<td>MVAS</td>
<td>M1a, M3b</td>
<td></td>
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<tr>
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<td>LVAS</td>
<td>M1a, M3b</td>
<td></td>
<td></td>
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<tr>
<td>Vastus Intermedius</td>
<td>LVAS</td>
<td>M1a, M3b</td>
<td></td>
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<td>GMED1</td>
<td>M1a, M1b, M3b</td>
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<td>M1a, M1b, M3b</td>
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<td>GMED3</td>
<td>M1a, M1b, M3b</td>
<td></td>
<td></td>
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<tr>
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<td>PIR1</td>
<td>Bimodal</td>
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<td>Gemellus</td>
<td>GEM</td>
<td>Bimodal</td>
<td></td>
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<tr>
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<td>M1a, M1b, M3b</td>
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<td>M1a, M1b, M3b</td>
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<td>M1a, M1b, M3b</td>
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RESULTS

Forward Propulsion

In the non-impaired control subjects, the plantarflexors (SOL+GAS) and knee extensors (RF+VAS) both had substantial potential to generate braking (negative AP GRF, Fig. 4.1c) while the hamstrings (HAM), SOL+GAS, and gluteus medius (GMED) had substantial potential to generate forward propulsion (positive AP GRF, Fig. 4.1c).

When Modules 1 and 2 were merged in Group A, the potential of SOL+GAS to generate braking was notably increased while the potential to generate forward propulsion was decreased (compare Figs. 4.1a and 4.1c, impulses). Instead of generating braking primarily during mid-stance, SOL+GAS generated braking from early stance well into late stance (compare Figs. 4.1a and 4.1c, dotted line in timing traces). Similarly, RF+VAS had an increased potential to generate braking (compare Figs. 4.1a and 4.1c, impulses) due to extended braking throughout most of stance compared to controls (compare Figs. 4.1a and 4.1c, solid line in timing traces).

Several notable differences occurred when Modules 1 and 4 were merged in Group B. Similar to Group A, SOL+GAS in Group B had an increased potential to generate braking and a decreased potential to generate forward propulsion (compare Figs. 4.1b and 4.1c, impulses). Again, the braking from SOL+GAS occurred earlier and extended later in stance (compare Figs. 4.1b and 4.1c, dotted line in timing traces). Both HAM and GMED had the potential to generate a larger amount of forward propulsion (compare Figs. 4.1b and 4.1c, impulses). Similar to controls, HAM generated this forward propulsion mostly from early to mid-stance (compare Figs. 4.1b and 4.1c, dash-dot line in timing traces). GMED generated forward propulsion from early to mid-stance in Group B, but in the controls GMED generated forward propulsion primarily during
mid-stance (compare Figs. 4.1b and 4.1c, dashed line in GRF traces). Finally, the braking generated by RF+VAS was decreased (compare Figs. 4.1b and 4.1c, impulses).

**Figure 4.1:** AP GRF impulses (left side) and traces (right side) during the stance phase for the (a) Group A, (b) Group B and (c) non-impaired control simulations. Hemiparetic values correspond to muscle groups on the paretic leg.

**Body Support**

In the non-impaired controls, GMED, RF+VAS and SOL+GAS each had substantial potential to generate body support (positive vertical GRF) (Fig. 4.2c), RF+VAS and GMED during early stance and SOL+GAS from mid- to late stance. In
addition, HAM had a small potential to generate body support at the very beginning of stance.

Similar to controls, when Modules 1 and 2 were merged in Group A, SOL+GAS, RF+VAS and GMED each had substantial potential to generate body support (Fig. 4.2a, impulses). Instead of generating body support starting in mid-stance, the potential of SOL+GAS to generate body support started in early stance (compare Figs. 4.2a and 4.2c, dotted line in GRF traces). Similar to controls, RF+VAS and GMED primarily generated body support in early stance. However, RF+VAS also generated negative body support near mid-stance (compare Figs. 4.2a and 4.2c, solid line in GRF traces). Finally, HAM no longer generated body support near the beginning of stance.

SOL+GAS, RF+VAS and GMED also had substantial potential to generate body support when Modules 1 and 4 were merged in Group B (Fig. 4.2b, impulses), although there were differences in timing. SOL+GAS contribution to body support began during early stance instead of mid-stance (compare Figs. 4.2b and 4.2c, dotted line in GRF traces) and both RF+VAS and GMED generated body support further into stance (compare Figs. 4.2b and 4.2c, solid and dashed lines in GRF traces). However, the total body support provided by GMED was reduced. Finally, HAM generated negative body support from early to mid-stance (Fig. 4.2b) instead of positive body support (Fig. 4.2c).
Mediolateral Balance Control

In the non-impaired controls, GMED generated a medial GRF throughout stance, HAM generated a lateral GRF at the beginning of stance and SOL+GAS generated a lateral GRF during late stance.

While GMED still had a large potential to generate a medial GRF when Modules 1 and 2 were merged in Group A (Fig. 4.3a, impulses), its potential extended further into stance (compare Figs. 4.3a and 4.3c, dashed line in GRF traces). The plantarflexors
generated a medial GRF near the beginning of stance and then a lateral GRF from early into late stance (Fig. 4.3a, dotted line in GRF traces).

When Modules 1 and 4 were merged in Group B, GMED had a similar potential to generate a medial GRF compared to controls (compare Figs. 4.3b and 4.3c). The lateral contribution from HAM, however, was extended throughout stance (compare Figs. 4.3b and 4.3c, dash-dot line in GRF traces), opposing the medial contribution from GMED. Finally, the plantarflexors generated a medial GRF during late stance instead of a lateral GRF (compare Figs. 4.3b and 4.3c, dotted line in GRF traces).
Leg Swing Control

In the non-impaired controls, only HAM and SOL+GAS contributed to ipsilateral leg swing control (Fig. 4.4a); the plantarflexors delivered energy to the leg in pre-swing while HAM absorbed energy from the leg during late swing prior to heel-strike. When Modules 1 and 2 were merged in Group A, HAM function remained the same while those muscles in the merged module (GMED, SOL+GAS and RF+VAS) all absorbed energy from the leg during pre-swing. When Modules 1 and 4 were merged in Group B, SOL+GAS function remained the same while HAM absorbed less energy from the leg, GMED absorbed energy from the leg and RF+VAS generated energy to the leg during late swing.

Both HAM and GMED delivered energy to the contralateral leg during pre-swing and into swing in all simulations (Fig. 4.4b). HAM primarily delivered energy to the contralateral leg prior to swing in controls, but in both Group A and Group B this was extended throughout swing. GMED delivered energy to the contralateral leg during pre-swing and early swing in controls and also when Modules 1 and 2 were merged in Group A. However, when Modules 1 and 4 were merged in Group B, GMED delivered less energy to the contralateral leg during pre-swing and had minimal effect during swing. In all groups SOL+GAS had minimal contributions to contralateral leg swing control while RF+VAS absorbed energy from pre-swing into late-swing.

DISCUSSION

Muscle activity post-stroke can be explained using fewer modules than found in non-impaired control subjects, with two or more of the non-impaired modules often merged together (Clark et al., 2010). Moreover, the different coordination impairments observed post-stroke have been found to manifest in different types of module compositions. Understanding how different module compositions post-stroke influence
walking performance has important implications for the design of targeted rehabilitation interventions aimed at improving rehabilitation outcome. This study examined the effects of two commonly merged modules on walking performance post-stroke: the merging of (a) the gluteus medius and knee extensors of Module 1 with the plantarflexors of Module 2 and (b) the gluteus medius and knee extensors of Module 1 with the hamstrings of Module 4.

**Group A (merged Modules 1 and 2)**

When Modules 1 and 2 were merged in Group A, a major difference compared to non-impaired controls was the reduced ability to generate forward propulsion while the ability to generate body support and control mediolateral balance did not appear...
Figure 4.5: Module timing for the Group A simulation. (a) The merged module in Group A controls the plantarflexors (SOL+GAS) and the gluteus medius (GMED) and knee extensors (RF+VAS) which are separately controlled in the non-impaired control simulation. (b) Even though the hamstrings (HAM) are controlled with their own module in Group A, the excitation pattern is extended further into stance compared to non-impaired controls.

impaired. This reduced ability to generate forward propulsion is consistent with the reduced paretic leg propulsion found experimentally within this group of subjects (Clark et al., 2010) and was a combination of both increased braking from the muscle groups in the merged module and an impaired capacity of the plantarflexors to generate propulsion in late stance (Fig. 4.1).

The increased braking was a result of the altered excitation timing of the merged module compared to the two non-impaired modules. The knee extensors and gluteus medius in Module 1 are normally active only in early stance, but in the merged module their activity was extended into late stance (Fig. 4.5a). Similarly, the plantarflexors of Module 2 are normally activated beginning in mid-stance but in the merged module they were instead activated beginning in early stance (Fig. 4.5a). As a result, the braking
generated by the knee extensors that normally occurs during early stance in non-impaired walking was prolonged throughout the stance phase (Fig. 4.1). Similarly, the braking generated by the plantarflexors that occurs during mid-stance in non-impaired walking began much earlier (Fig. 4.1). Consequently, the overall braking in Group A was much larger than in the control subjects.

The plantarflexors also had a reduced capacity to generate forward propulsion during late stance. This muscle group is critical for forward propulsion in non-impaired controls (e.g., Liu et al., 2006; Neptune et al., 2001) and their reduced potential to generate forward propulsion in Group A is consistent with recent muscle-actuated simulations of post-stroke hemiparetic walking that found reduced forward propulsion from the plantarflexors (Hall et al., 2011; Peterson et al., 2010b). Forward propulsion is not only a function of muscle force production but also depends on where the foot is relative to the center-of-mass (Peterson et al., 2010a). Without proper leg positioning, the mechanical advantage to propel the center-of-mass forward is diminished. In Group A the paretic leg hip and knee angles were at a nearly neutral position during late stance, likely due to extended activity of the knee and hip extensors, leaving the paretic leg in a poor configuration to generate forward propulsion. Therefore, the plantarflexors were unable to produce forward propulsion to the extent found in controls.

Another consequence of merging Modules 1 and 2 was inhibited ipsilateral leg swing control (Fig. 4.4a). In non-impaired walking the gluteus medius and knee extensors are not active during the contralateral swing phases and thus have minimal contributions to leg swing control. However, their activity was extended into pre-swing in Group A, causing them to absorb leg energy prior to swing. In addition, the plantarflexors also had altered contributions to leg swing control. In non-impaired walking the biarticular plantarflexors (GAS) are important for swing initiation (e.g., Neptune et al., 2004) and
Figure 4.6: Average power delivered to the ipsilateral leg, contralateral leg and trunk during pre-swing by the uniarticular (SOL) and biarticular (GAS) plantarflexors for (a) non-impaired controls and (b) Group A. Total represents the average power delivered to the ipsilateral leg, contralateral leg and trunk.

transfer energy to the leg during pre-swing (Fig. 4.6a) such that the overall energy transferred from both plantarflexors is positive (Fig. 4.4a). In Group A, however, due to kinematic differences GAS no longer generated energy to the leg during pre-swing and instead absorbed leg energy similar to the uniarticular plantarflexors (SOL, Fig. 4.6b). If no other muscle groups compensate for these negative plantarflexor, gluteus medius and knee extensor contributions to ipsilateral leg swing initiation in Group A, those with merged Modules 1 and 2 would have impaired ipsilateral leg swing control.

While the hamstrings module remained independently activated in Group A, its timing differed slightly from the hamstrings module in controls and its high activity was extended further into stance (Fig. 4.5b). Prolonged hamstrings activity has previously been documented in post-stroke hemiparetic subjects (e.g., Den Otter et al., 2007; Knutsson and Richards 1979; Shiavi et al., 1987), with Den Otter et al. (2007) suggesting that the prolonged activity, in conjunction with prolonged knee extensor activity, may be a compensatory mechanism used to overcome insufficient plantarflexor strength and provide additional body support. While the hamstrings do provide support in non-
impaired walking at the beginning of stance, due to kinematic differences they have very minor contributions to support in Group A. However, their contribution to forward propulsion is extended, which suggests that they may be utilized as a compensatory mechanism to provide needed forward propulsion and counteract the increased braking from the knee extensors and plantarflexors that in during early stance (Fig. 4.1).

**Group B (merged Modules 1 and 4)**

When Modules 1 and 4 were merged together in Group B, forward propulsion, body support, and mediolateral balance were all affected. In non-impaired walking the hamstrings are only activated into early stance where they generate body support (Fig. 4.2). However, in the merged module of Group B, this high hamstrings activity was extended further into stance with the knee extensors (Fig. 4.7a), which is a common co-activation pattern among post-stroke hemiparetic subjects (e.g., Knutsson and Richards 1979; Shiavi et al., 1987). Instead of the hamstrings generating body support later into stance as would be expected, they acted to impede body support (i.e., negative body support, Fig. 4.2). While not intuitive, this was due to altered kinematics that put the hamstrings in a position where they acted to flex the knee more than extend the hip, which has the overall effect of lowering the center-of-mass and compromising body support. Furthermore, the hamstrings also had altered contributions to mediolateral balance control. During non-impaired walking, the hamstrings, gluteus medius and plantarflexors work synergistically to control mediolateral balance (Fig. 4.3). In Group B the lateral contribution from the hamstrings in early stance is extended throughout much of stance, opposing the important medial contribution from the gluteus medius, which may result in compromised mediolateral balance control.
Figure 4.7: Module timing for the Group B simulation. (a) The merged module in Group B controls the hamstrings (HAM) and gluteus medius (GMED) and knee extensors (RF+VAS) which are separately controlled in the non-impaired control simulation. (b) Even though the plantarflexors (SOL+GAS) are controlled with their own module in Group B, the excitation pattern differs from controls and the plantarflexors are excited earlier in stance.

The contributions to both ipsilateral and contralateral leg swing were also affected when Modules 1 and 4 were merged. In the control subjects the gluteus medius and knee extensors were activated only near the very end of swing and have little to no contributions to ipsilateral leg swing control. Consequently, when they were merged with Module 4, their activity began earlier in swing (Fig. 4.7a), and as a result their contributions to ipsilateral leg swing were altered: gluteus medius acted to decelerate the leg (absorb energy) while the knee extensors acted to accelerate the leg (generate energy) prior to heel-strike. However, the overall effect was still to decelerate the leg prior to heel-strike, and therefore the merging of Modules 1 and 4 does not appear to impair ipsilateral leg swing control. Similarly, while individual contributions to contralateral leg swing were altered, it does not appear that contralateral leg swing is impaired when
Modules 1 and 4 were merged. While the gluteus medius had less potential to transfer energy to the contralateral leg prior to and during swing than in controls, the hamstrings compensated through their extended activity into swing (Fig. 4.4b).

Despite their independent recruitment, the plantarflexors in Group B were prematurely activated earlier in stance than in controls (Fig. 4.7b), which is a common abnormality following stroke (Den Otter et al., 2007; Knutsson and Richards 1979). This premature activity could be due to either impaired plantarflexor recruitment or a compensatory strategy to overcome the effects of merging Modules 1 and 4. Given that the plantarflexors are critical generators of body support, it is possible that they were activated earlier to counteract the negative body support from the hamstrings in order to provide needed body support. Similar to the plantarflexors in Group A, the plantarflexors in Group B also had a reduced potential to generate forward propulsion due to poor mechanical advantage. Therefore, while the muscles in the merged module did not impair forward propulsion generation, the overall effect left the body in such a position that the plantarflexors were still unable to generate proper forward propulsion during late stance.

**Limitations**

One of the limitations of this study was that the tracking torques in both the Group A and Group B simulations were not completely eliminated. However, the unit force perturbation analysis used in this study is insensitive to the value of the tracking torques. This kind of analysis produces the *potential* of a muscle to contribute to a biomechanical function rather than its absolute contribution. While the total effect from all muscles within a merged module muscles depends on the relative amount to which they are recruited, their individual function would remain the same.
CONCLUSIONS

During non-impaired walking muscles are combined into modules that exploit their unique functional roles to produce well-coordinated walking. When modules are merged post-stroke this uniqueness is no longer exploited and walking performance is impaired. The plantarflexors had reduced potential to generate forward propulsion regardless of module composition, which is consistent with the results of Chapter 2 (Allen et al., 2011) that found the plantarflexors were impaired in all subjects regardless of step length asymmetry (the subject in the Group A simulation had a symmetric PSR of 0.501 while the subject in the Group B simulation had a low PSR of 0.443). This suggests that improving forward propulsion from the plantarflexors should be a focus of rehabilitation in both groups of subjects. However, while the plantarflexors are independently activated by their own module in Group B, they are co-excited with the knee extensors and gluteus medius in Group A. Therefore, this group of subjects would also likely benefit from rehabilitation programs that include tasks that separately focus on each muscle group.

The remaining functional consequences depended on the specific modules that were merged, which highlights how subjects with each of these common types of merged modules would benefit from different types of rehabilitation efforts. Group A also exhibited impaired ipsilateral leg swing and, therefore, rehabilitation that focuses on improving the energy delivered to the leg prior to swing may be beneficial for this group. In Group B both body support and mediolateral balance control were compromised, and these subjects may benefit from rehabilitation programs that include more focus on improving these biomechanical functions. Future study should be performed on other types of merged module compositions, such as those who could only independently
activate two modules, to understand their different effects on walking performance and provide guidelines for rehabilitation strategies to improve their walking performance.
Chapter 5

Conclusions

The overall goal of this research was to use experimental and simulation analyses to investigate the potential of two different measures (step length asymmetry and module composition) to provide information regarding walking performance in individuals with post-stroke hemiparesis. Experimental analyses were performed on hemiparetic subjects grouped by step length asymmetry to analyze the difference in joint mechanics compared to non-impaired controls (Chapter 2). Simulation analyses were performed to understand how modules contribute to important biomechanical functions during non-impaired walking (Chapter 3) and how the generation of these functions is altered when post-stroke hemiparetic subjects merge different groups of non-impaired modules (Chapter 4). The results of this research provide two potential measures that can be used to develop rehabilitation interventions that target specific functional deficits in the post-stroke hemiparetic population.

The experimental analyses used in Chapter 2 grouped hemiparetic subjects by step length asymmetry and found that paretic plantarflexor impairment consistently occurred in all subjects regardless of step length asymmetry. Thus, clinicians should focus on improving paretic leg plantarflexor output in all subjects during rehabilitation. The direction of asymmetry can be used to understand both the degree of paretic leg plantarflexor impairment and the compensatory mechanisms used. Those subjects who walked with symmetric steps were able to compensate for plantarflexor impairment using similar output from both legs. Those subjects who walked with longer paretic than nonparetic steps, on the other hand, relied on the nonparetic leg, and improving paretic leg output in these subjects may improve their walking symmetry. These results suggest
that step length asymmetry may be a useful measure in assessing underlying coordination mechanisms that can be use to guide rehabilitation efforts.

In Chapter 3, the contributions of modules identified during non-impaired walking to important 3D biomechanical functions were analyzed using simulation analyses. Previous simulation studies have shown that five modules satisfy the sagittal-plane biomechanical functions of 2D walking. The results of this study show that a sixth module, which contributes primarily to mediolateral balance control and contralateral leg swing, was needed to satisfy the additional non-sagittal plane demands of 3D walking. Body support was provided by Module 1 (hip and knee extensors, hip abductors) in early stance and Module 2 (plantarflexors) in late stance. In early stance, forward propulsion was provided by Module 4 (hamstrings), but net braking occurred due to Modules 1 and 2. Forward propulsion was provided by Module 2 in late stance. Module 1 accelerated the body medially throughout stance, dominating the lateral acceleration in early stance provided by Modules 4 and 6 (adductor magnus) and in late stance by Module 2, except near toe-off. Modules 3 (ankle dorsiflexors, rectus femoris) and 5 (hip flexors and adductors except adductor magnus) accelerated the ipsilateral leg forward in early swing whereas Module 4 decelerated the ipsilateral leg prior to heel-strike. Finally, Modules 1, 4 and 6 accelerated the contralateral leg forward prior to and during contralateral leg swing. These results provide further evidence that a simple neural control strategy involving muscle activation modules organized around task-specific biomechanical functions may be used to control complex human movements and provide the basis for further analyses of impaired module composition in impaired populations.

Chapter 4 investigated how impaired module composition post-stroke affects the generation of important biomechanical functions during walking. Specifically, two common and distinct types of module composition were analyzed: (a) merged Modules 1
and 2 and (b) merged Modules 1 and 4. Similar to the results of Chapter 2, the ankle plantarflexors appeared impaired regardless of which modules were merged. Specifically, the ankle plantarflexors exhibited reduced potential to generate forward propulsion in both groups, which suggests that both groups would benefit from rehabilitation targeted at improving plantarflexor function. In Group A, however, the plantarflexors were co-activated with the knee extensors and gluteus medius, therefore subjects in this group may also benefit from rehabilitation programs that include tasks which separately stress these muscle groups. The remaining biomechanical functions affected differed between groups: Group A had impaired ipsilateral leg swing control while Group B exhibited compromised body support and mediolateral balance control. These results highlight how the merging of specific modules has different functional outcomes and provides rationale for the specific biomechanical functions that should be targeted during rehabilitation for these specific types of module compositions.
Chapter 6

Future Work

This work can be expanded in several different areas. A key finding in this research was evidence that both PSR and module composition appear to be useful measures of walking performance for developing rehabilitation interventions. While the results of these studies suggest that PSR and module composition can be used to target specific deficits and thus improve underlying coordination mechanisms, rehabilitation interventions utilizing these measures are needed to support these results. Therefore, future studies are warranted to evaluate the effectiveness of both PSR and module composition as measures of improved walking performance throughout rehabilitation.

In addition, future study simulating additional types of module compositions is necessary in order to expand its usefulness as a measure of walking performance. The simulation analyses performed in Chapter 4 were performed for only two types of merged modules that were identified in the post-stroke hemiparetic population. While these were the most common types among those who merged only two non-impaired modules, additional module compositions were found within this group of hemiparetic subjects (Clark et al., 2010). In addition, some hemiparetic subjects merged more than two non-impaired modules (Clark et al., 2010). Additional simulation analyses of the different types of module compositions should be performed in order to increase its usefulness as a measure of walking performance in the general post-stroke population.

Due to the heterogeneity of the stroke population, variability still existed even within each group of subjects studied (both in the PSR groups in Chapter 2 and in the module composition groups in Chapter 4). For example, in Chapter 2 the variability of joint moments in the low PSR group was large enough such that no significant results
were found at joints other than the paretic ankle. A low PSR is more uncommon and thus this group contained the fewest number of subjects. Future work examining a larger number of low PSR subjects is needed to determine if there are any underlying impairments in this subgroup. In addition, even though specific deficits and compensations were identified in the symmetric and high PSR groups, performing subject-specific simulations may provide further information on how PSR affects walking performance.

In the same way, while module composition was similar between subjects in each group in Chapter 4, the individual components of module composition (e.g., muscle weights and module timing patterns) varied slightly among subjects. The simulations in Chapter 4 were performed for a representative subject from each group and future work should focus on developing subject-specific simulations to gain insight in how this variability may affect the influence of the merged modules on biomechanical function within each group.

While subject-specific simulations are ideal, the current forward dynamics simulation framework requires large amounts of computation time to converge upon a solution, making the application toward subject specific simulations impractical. Various techniques have been developed that attempt to solve this issue (e.g., Damsgaard et al., 2006; Delp et al., 2007), each with their own limitations. Future work developing a faster optimization approach to develop subject-specific simulations is needed. To this effect, I have begun developing a technique that combines inverse dynamics static optimization with forward dynamics (similar to van der Helm 2011), and also takes into account our dynamic ground-foot contact model (see Appendix E for details).

Finally, in addition to understanding how modules synergistically control the biomechanical demands of straight-line walking at self-selected speed (Chapter 3),
understanding how modules are used to control other tasks (i.e. standing balance, turning, stair-stepping, accelerating/decelerating, etc) is also important for improving the quality of life in impaired populations. While improving straight-line walking performance is critical, many additional tasks are also contained during activities of daily living. For example, walking across the street may involve other tasks such as stepping up/down a curb, acceleration/deceleration and turning. While previous experimental studies have identified modules in non-impaired subjects during some tasks other than walking (e.g, walking with voluntary movements such as kicking or reaching, Ivanenko et al., 2005; standing balance, Torres-Oviedo et al., 2006), no study has yet examined how the identified modules contribute to the biomechanical demands necessary for successfully performing each task. Understanding how non-impaired modules during various tasks affect their necessary biomechanical functions will allow us to understand how altered module composition in impaired populations adversely affects task performance. This information could then be used to develop rehabilitation interventions that target specific deficits in a variety of tasks, not just straight-line walking.
Appendix A

Non-negative Matrix Factorization

Given a nonnegative matrix \( V \), the objective of non-negative matrix factorization (NNMF) is to find nonnegative matrix factors \( W \) and \( H \) such that \( V \approx WH \) (Lee and Seung 1999, 2001). Given a set of \( m \)-dimensional data vectors, the vectors are placed in the columns of an \( m \times t \) matrix \( V \) where \( t \) is the number of examples in the data set. This matrix is then approximately factorized into an \( m \times n \) matrix \( W \) and an \( n \times t \) matrix \( H \), where \( n \) is chosen to be smaller than \( m \), so that the product \( WH \) represents a compressed version of the original data matrix, \( V \). The \( n \times t \) matrix \( H \) is a set of \( n \) basis functions of length \( t \) and \( W \) specifies the relative weighting of each \( m \) original data vectors to the \( n \) basis functions. When multiplied together the result is an approximation of the original data matrix. The matrices \( H \) and \( W \) are optimized until the error between \( WH \) and \( V \) is minimized.

For each subject, the NNMF algorithm presented above was used to decompose the EMG from the eight leg muscles into a set of basis patterns, or modules, and the corresponding weights which represent the relative weighting of each muscle in each module (Clark et al., 2010). For each subject, EMG was combined into an \( m \times t \) matrix \( V \) where \( m \) was equal to the number of muscles \( (m = 8) \) and \( t \) is the time base \( (t = \text{number of strides} \times 101) \). A priori, the number of modules \( (n) \) was specified and the NNMF algorithm was used to find the best \( W \) and \( H \) matrices, where \( H \) specifies the activation timing of each module over each of the gait cycles and \( W \) specifies the relative weighting of a muscle in each module, with each muscle weight invariant across all gait cycles. The Matlab function NNMF was used to find the optimal \( W \) and \( H \) matrices.
No a priori assumptions were made regarding the number of modules \((n)\) that would be required to adequately reconstruct the original EMG signals. Therefore, separate NNMF analyses were performed with the output constrained to one, two, three, four and five modules \((n = 1,2,3,4 \text{ and } 5)\). The ratio of the sum of the squared error to the sum of the squared original EMG was used to quantify the variability accounted for (VAF) for a given number of modules.

\[
\text{VAF} = 1 - \frac{(\text{EMG}_o - \text{EMG}_r)^2}{\text{EMG}_o^2}\quad \text{Eq. A1}
\]

where \(\text{EMG}_r\) is the reconstructed EMG and \(\text{EMG}_o\) is the original EMG. The VAF was calculated across all gait cycles as well as within different regions of the gait cycle. The analysis began with \(n = 1\) and the number of modules was increased until all muscles in all regions achieved \(\text{VAF} \geq 0.9\) or until adding an additional module did not increase VAF by \(> 0.05\) for the muscle(s) and/or region(s) with the lowest VAF.
Appendix B
Simulation Module Composition

Non-Impaired Simulation

The NNMF algorithm was performed on eight muscles per leg and four modules were identified in the non-impaired control group (Fig. B1). A fifth module was also included to account for the hip flexors (Neptune et al., 2009). Muscles with similar anatomical function for which EMG data were not recorded were included in each module according to the rationale as described below. This resulted in 30 of the 38 muscles per leg being included in one of five modules (Fig B2).

Figure B1: Module weighting (a) and activation timing profiles for the non-impaired control subjects during self-selected (b) and at fixed walking speeds (c). The black lines/bars represent the group average and the gray bars/lines represent each individual subject. (Clark et al., 2010)
Based on the experimentally measured EMG, Module 1 was found to include the gluteus medius (GM), rectus femoris (RF) and vastus medialis (VM). Similar to the previous 2D study (Neptune et al., 2009), the vastus intermedius and lateralis were included in Module 1 due to their similar anatomical arrangement and action to vastus medialis. Gluteus maximus was also successfully included in this module in the previous 2D simulation, and was included because Ivanenko and colleagues (2004) found that it was strongly associated with Module 1. This is consistent with the gluteus maximus acting co-functional with the vasti to provide body support in early stance (Neptune et al., 2004; Zajac et al., 2003). Finally, gluteus minimus was included in Module 1 because of its anatomical arrangement and its similar excitation timing as the other muscles in Module 1 (Sutherland 1984).

Module 2 was experimentally found to include the soleus (SO) and medial gastrocnemius (MG). The lateral gastrocnemius, flexor digitorum longus and tibialis posterior were also included in this module due to similar anatomical arrangement and potential to plantarflex the ankle.

Module 3 was experimentally found to include rectus femoris and tibialis anterior. (TA) The extensor digitorum longus was included in this module since it also acts to dorsiflex the ankle.

Experimentally, Module 4 was found to have high representation from the medial (semimembranosus) and lateral (biceps femoris-long head) hamstrings (MH, LH). The semitendinosus and gracilis were included due to their similar anatomical arrangement and excitation timing (Sutherland 1984). The biceps femoris-short head was also included because it is often co-excited with the hamstrings and was successfully included in a module with these muscles in the previous 2D simulation (Neptune et al., 2009).
Ivanenko and colleagues (2004) identified a fifth module which was associated primarily with iliopsoas and erector spinae muscle activity, which were not collected in this study. The previous 2D simulation study successfully included this fifth module which consisted of the iliacus and psoas (Neptune et al., 2009). In our current 3D model, sartorius and pectineus were also included in Module 5 since they both can act as hip flexors.

The remaining hip muscles were initially not included in any of the 5 modules. While the adductor brevis, adductor longus and adductor magnus can also act to flex the hip similar to the muscles in Module 5, they were not initially included because of their high potential to adduct the thigh. Similarly, quadratus femoris, piriformis, gemellus, and tensor fascia lata were initially given individual excitation patterns because of their main action to abduct and/or adduct the thigh, with minimal hip flexor/extensor potential.

Figure B2: Muscles within each module for the non-impaired control simulation.
Impaired Simulations

Two simulations were generated to represent two commonly observed sets of merged modules. Three modules were identified in both Group A (Fig. B3) and Group B (Fig. B4). To determine which muscles would be controlled by a module in the simulation, a cut-off of 0.4 was used on the module weighting value. In addition, muscles with similar anatomical function for which EMG data were not recorded were included in each module.

Group A Simulation

Module M1a was found to include the soleus, medial gastrocnemius, vastus medialis, rectus femoris and gluteus medius. The lateral gastrocnemius was also included in this module due to similar anatomical arrangement and potential to plantarflex the ankle. Gluteus maximus and minimus were also included due to their anatomical arrangement to gluteus minimus. Module M2a was found to include the tibialis anterior and rectus femoris. Module M3a was found to include lateral (biceps femoris-long head) and medial (semimembranosus) hamstrings. The semitendinosus and gracilis were included due to their similar anatomical arrangement. The biceps femoris-short head was also included because it is often co-excited with the hamstrings.

Group B Simulation

Module M1b was found to include the soleus and medial gastrocnemius. The lateral gastrocnemius was also included in this module due to similar anatomical arrangement and potential to plantarflex the ankle. Module M2a was found to include the tibialis anterior and rectus femoris. Module M3a was found to include vastus medialis, rectus femoris, gluteus medius, lateral (biceps femoris-long head) and medial
(semimembranosus) hamstrings. Gluteus maximus and minimus were also included due to their similar anatomical arrangement to gluteus minimus. The semitendinosus and gracilis were included due to their similar anatomical arrangement to the other hamstrings. The biceps femoris-short head was also included because it is often co-excited with the hamstrings.
Figure B3: Module weighting and activation timing profiles for the Group A subjects during self-selected walking speeds. The black lines/bars represent the group average and the gray bars/lines represent each individual subject. (Clark et al., 2010)

Figure B4: Module weighting and activation timing profiles for the Group B subjects during self-selected walking speeds. The black lines/bars represent the group average and the gray bars/lines represent each individual subject. (Clark et al., 2010)
Appendix C

Simulation Tracking Torques

The tracking torque at each joint was based on the difference between the simulated and experimental angles. A logistic function was used to ensure a smooth transition of torque application. The torque was only applied once the joint angle error was greater than two standard deviations of the experimental joint angle as follows:

\[
T_i(t) = A \times [q_i(t) - \hat{q}_i(t)] \frac{1}{1 + \exp \left( \frac{B}{2SD_i(t)} |q_i(t) - \hat{q}_i(t)| + C \right)}
\]  

Eq. C1

where \( T_i(t) \) is the applied torque at time \( t \) for joint \( i \), \( \hat{q}_i(t) \) is the desired joint angle position at time \( t \) for joint \( i \), \( q_i(t) \) is the simulated joint angle at time \( t \) for joint \( i \), \( SD_i(t) \) is the standard deviation at time \( t \) for joint \( i \), \( A \) is the proportional multiplier, and \( B \) and \( C \) are the logistic function parameters and are equal to 91.9 and 87.3, respectively. The values for \( B \) and \( C \) were chosen such that the logistic function multiplier is zero until the joint error equals 90% of two standard deviations of the experimental data and is equal to one when the error is equal to two standard deviations of the experimental data (see Figs C1 and C2).
Figure C1: The exponential multiplier is zero until the error is equal to 1.8 standard deviations and is equal to one after the error reaches two standard deviations.
Figure C2: Example of tracking torque calculation. (a) Experimental (solid) and simulated (dashed) joint angle. Gray region represents ±2 SD. (b) Error between the experimental and simulated joint angle normalized by SD (blue solid line). The tracking torque does not turn on until the error reaches 1.8 SD (dotted black line) and reaches its full value at 2 SD (solid black line). (c) Value of the exponential multiplier (blue line) and the resulting feedback torque (green line)
Appendix D

Representative Subjects for Hemiparetic Simulations

The hemiparetic simulations performed in Chapter 4 were developed for a representative subject in each group, where the representative subjects were chosen based on similar walking speeds and minimum difference between kinematics and GRFs compared to their group average data. The individual gait cycle used for each subject with the minimum difference in kinematics and GRFs compared to that subject’s average data was then used to generate the simulations.

Group A (merged Module 1 and 2)

Seven subjects were in Group A (58 ± 10.4 years; 4 male; 3 right side hemiparesis; 3.6 ± 2.5 years post-stroke; self-selected treadmill speed = 0.59 ± 0.24 m/s). Based on the difference between the group average kinematics/GRFs and subject values, subject 42 was chosen to simulate (see Table D1 and Figs. D1 and D2).

Group B (merged Module 1 and 4)

Four subjects were in Group B (69.5 ± 11.23 years; 3 male; 3 right-side hemiparesis; 3.4 ± 3.4 years post-stroke; self-selected treadmill speed = 0.45 ± 0.17 m/s). Based on the difference between the group average kinematics/GRFs and subject values, subject 14 was chosen to simulate (see Table D2 and Figs. D3 and D4).
Table D1: Subject average difference (kinematic and GRF) from group average data for hemiparetic Group A. Subject 42 (shaded gray) was chosen as the representative subject due to minimal difference from the group average data.

<table>
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<th>26</th>
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Figure D1: Paretic leg individual subject kinematics and group average ± two standard deviations for Group A.
**Figure D2:** Nonparetic leg individual subject kinematics and group average ± two standard deviations for Group A.
Table D2: Subject average difference (kinematic and GRF) from group average data for hemiparetic Group B. Subject 14 (shaded gray) was chosen as the representative subject due to minimal difference from the group average data.

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Average Angle Difference (radians) | 4.8 | 5.3 | 4.6 | 7.0 |

Forces (%BW) |         |   |   |   |
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Average GRF Differences (%BW) | 2.8 | 2.7 | 6.0 | 2.9 |
Figure D3: Paretic leg individual subject kinematics and group average ± two standard deviations for Group B.
Figure D4: Nonparetic leg individual subject kinematics and group average ± two standard deviations for Group B.
Appendix E

Inverse/Forward Optimization Approach

The generation of subject-specific simulations holds much promise to provide individualized recommendations for rehabilitation. However, a large computational effort is required to solve large-scale musculoskeletal models using forward dynamics techniques. This results in an inordinate amount of computation time, making the use of forward dynamic optimization for subject-specific simulation challenging. Recently, a “fast, near optimal” forward dynamic simulation of shoulder and elbow motion has been developed using a technique that combines inverse static optimization with forward dynamics (van der Helm 2011). The following presents a method based upon that of van Der Helm et al. (2011) which can be used to simulate the lower extremity during walking and incorporates a ground contact model.

Musculoskeletal Model

The model used was a planar bipedal musculoskeletal model previously developed in SIMM with 13 degrees-of-freedom (MusculoGraphics, Inc, Neptune et al., 2001). The model consisted of rigid segments that represented the trunk and two legs, where the trunk segment included the mass and inertial characteristics of the pelvis, torso, head and arms and each leg consisted of a thigh, shank, patella rear-foot, mid-foot and toes. Musculoskeletal model geometry was based on Delp et al. (1990). The trunk had three degrees-of-freedom (2 translations and 1 rotation) and single degree-of-freedom revolute joints were used to model the hip and ankle. The knee (tibiofemoral) joint was modeled with a moving center-of-rotation for flexion-extension specific as functions of knee flexion angle, with the patella constrained to move along a prescribed trajectory...
relative to the femur as a function of knee flexion angle (Yamaguchi and Zajac 1989). Passive torques were applied at the hip, knee and ankle to model the forces applied by ligaments, passive tissues and joint structures (Anderson 1999; Davy and Audu 1987). Contact between the foot and ground was modeled using 31 visco-elastic elements with coulomb friction attached to each foot (Neptune et al., 2000). The dynamical equations-of-motion were generated using SD/FAST (Symbolic Dynamics, Inc.). The model was driven by 25 individual Hill-type actuators per leg: soleus, tibialis posterior, medial and lateral gastrocnemius, tibialis anterior, peroneus tertius, peroneus longus, peroneus brevis, flexor digitorum longus, flexor hallucis longus, extensor digitorum longus, extensor hallucis longus, biceps femoris long head, medial hamstrings, biceps femoris short head, rectus femoris, vastus medialis, vastus lateralis, vastus intermedius, psoas, iliacus, gluteus maximus, adductor magnus and the anterior and posterior compartments of the gluteus medius.

**Optimization Method**

An overview of the method is depicted in Figure E1. The inputs to the algorithm are the experimentally measured joint kinematics \((q_E)\) and joint torques \((T_{inv})\) derived using inverse dynamics. The outputs of the algorithm are the individual muscle forces, activations and excitations that result in the optimal tracking of the input experimental data. The algorithm is performed at each time step and includes two phases: an inverse dynamics phase and a forward dynamics phase.
Figure E1: Detailed schematic of the inverse/forward optimization technique. For each time step, the experimentally measured joint kinematics (q_E) and joint torques (T_{inv}) are input into a static optimization to solve for the muscle forces (F_{opt}) that are required to achieve the joint torques given the corresponding muscle moments arms. The optimal forces are bounded by upper and lower bounds based upon a forward muscle model which converts lower (u=0) and upper (u=1) to forces given the current state of the muscle (i.e., fiber length and velocity). The optimal forces are then input into an inverse muscle model which converts the optimal forces to corresponding optimal activations (a_{opt}) and neural excitations (u_{opt}). The excitations are then input into the model and the equations-of-motion are integrated forward one time step. Feedback torques are then calculated based on the error between the input experimental and output simulation kinematics. These torques are added to the experimental joint torques and the process is repeated until the tracking torques are reduced to zero.
The inverse dynamics phase uses a static optimization to solve the muscle redundancy problem in order to find the muscle forces ($F_{opt}$) that would result in the measured joint torques. The optimization also includes an objective function of minimizing the sum of squares of muscle stress in order to minimize unnecessary muscle co-contraction.

$$\text{minimize: } J(F_{MT}) = \sum_{i=1}^{\# \text{muscles}} \frac{F_{MT,i}}{PCSA} \quad \text{Eq. E1}$$

subject to:  

$$R(q)F_{MT} = T_{inv}$$

$$F_{LB} \leq F_{MT} \leq F_{UB}$$

where:  

- $PCSA$ = physiological cross-sectional area
- $F_{MT}$ = muscle forces
- $R(q)$ = matrix of muscle moment arms
- $T_{inv}$ = joint torques
- $F_{LB}, F_{UB}$ = muscle force lower and upper bounds

The lower and upper bounds for the muscle forces ($F_{LB}$ and $F_{UB}$) are calculated using a forward muscle model. Minimal and maximal neural excitations are input into the activation-dynamics equations to solve for the lower and upper activation bounds given the level of activation from the previous time step. The minimal and maximal neural excitations are 0 and 1, respectively (or they can be set to desired levels in order to bound the excitations). The lower and upper activation bounds are then used in the force model, which takes into account activation, the current state of the system, and the intrinsic muscle force-length-velocity relationships to solve for the lower and upper bounds on the individual muscle forces. The pseudo-code for the force model is described below.
**Force model:**

1. calculate musculotendon velocity
2. guess fiber length
3. calculate pennation angle and cosine factor
   \[ \text{cosine factor} = \cos(\text{pennation angle}) \]
4. calculate tendon length
   \[ \text{tendon length} = \text{musculotendon length} - \text{cosine factor} \times \text{fiber length} \]
5. normalize fiber and tendon lengths by optimal fiber length
6. calculate fiber velocity
   \[ \text{fiber velocity} = \text{cosine factor} \times \text{musculotendon velocity} \]
7. calculate tendon force
   1. calculate tendon strain
   2. interpolate tendon strain – force curve to get tendon force
8. calculate fiber force
   1. interpolate force-velocity relationship to get velocity factor
   2. interpolate active force-length curve to get active component
   3. interpolate passive force-length curve to get passive component
   4. \[ \text{fiber force} = \text{max isometric force} \times \text{cosine factor} \times (\text{active component} \times \text{activation} \times \text{velocity factor} + \text{passive component}) \]
9. calculate error between tendon and fiber force
10. if error \(< 0.00001\), end
11. else, update fiber length, go to 3

The optimal forces from the static optimization \(F_{\text{opt}}\) are then input into an *inverse muscle model*, which takes into account force-length-velocity relationships to calculate the muscle excitations that are necessary to generate the muscle forces from the static optimization. The inverse muscle model consists of two sub-models: a force to activation sub-model and an activation to excitation sub-model. First, the muscle forces solved for in the static optimization are input into the force to activation sub-model, which takes into account the force-length-velocity relationships to calculate the activation required to produce the desired force given the current state of the system. These activations are then fed into the activation to excitation sub-model to solve for the excitation that would produce the desired activation taking into account activation dynamics. The pseudo code of the two sub-models is presented below.
**Force to Activation:**
1. make initial guess of activation level
2. calculate muscle force using force model
3. calculate error between desired and calculated muscle force
4. if error < 0.001N, end
5. else, guess new activation level and go to 2

**Activation to Excitation:**
1. calculate desired activation rate ($\Delta a_{des}$)
   \[
   \Delta a_{des} = [a(i) - a(i - 1)] \frac{c_r}{\Delta t}
   \]  \hspace{1cm} \text{Eq. E2}
   where: \quad a(i) = activation at current time step
   \quad a(i-1) = activation at previous time step
   \quad c_r = activation timescale = 0.1s^{-1}

2. make initial guess of excitation ($u$)
3. calculate activation rate ($\Delta a_{calc}$) using activation dynamics equations
   if $u \geq a(i - 1)$  \hspace{1cm} \text{Eq. E3}
   \[
   \Delta a_{calc} = [u - a(i - 1)] * (c_1 u + c_2)
   \]
   else
   \[
   \Delta a_{calc} = [u - a(i - 1)] * c_2
   \]
   where: \quad $c_1 = \tau_{act}^{-1} - \tau_{deact}^{-1}$
   \quad $c_2 = \tau_{deact}^{-1}$
   \quad $\tau_{act} =$ activation time constant
   \quad $\tau_{deact} =$ de-activation time constant
4. calculate error in activation rate, abs($\Delta a_{calc} - \Delta a_{des}$)
5. if error < 0.00001, end
6. else, guess new excitation and go to 3
The excitations from the inverse dynamics phase are then input into a forward dynamics simulation, which integrates forward one time step. Due to slight differences in the muscle models between the inverse and forward dynamics phases and the use of discrete time steps, there exist small errors between the measured motion and the simulated motion from the forward dynamics phase that propagate over time. To account for these errors, feedback joint torques are calculated used to drive the simulation back to the desired experimental angles (see Appendix C). These feedback torques are added to the joint torques from the inverse dynamics phase and the inverse/forward process is performed again until the tracking torques are reduced to zero.

**Testing**

The output simulation results from a previous 2D forward dynamics optimization were used to test the new algorithm. Since the muscle forces, activations and excitations for the previous simulation output motion were available, this allowed the direct comparison of the new optimized muscle forces, activations and excitations with values known to produce the same motion. A feedback torque multiplier of 100 was used and the torques turned on when the error was 1° (see Appendix C for details on the feedback torque model).

First, the algorithm was run with excitations bounds based on the optimal excitations from the forward dynamics simulation (excitation ± 0.1). The result was a simulation that emulated well the kinematics (Fig. E2) and GRFs (Fig. E3), with comparable muscle activations (Figs. E4 and E5).

Next, the algorithm was run without excitation bounds to determine if the new inverse/forward algorithm would result in similar muscle activation patterns. This optimization resulted in good tracking of both kinematics (Fig. E6) and GRFs (Fig. E7).
However, while the activation of some of the muscles compared well with the activations from the forward dynamics optimization (Figs. E8 and E9), there were several muscles with very different activation patterns (e.g. BFsh, VAS1, GMEDa, etc). Therefore, it was clear that muscle redundancy allowed different solutions to emerge when the excitations were left unbounded.

Next, the excitations were bounded based on on/off timing from the forward dynamics simulation. The excitations were bounded by values of 0 and 0.01 when the forward dynamics optimal excitations were not on, and bounded by 0 and 1 otherwise. Again, the simulations resulted in good tracking of both kinematics (Fig. E10) and GRFs (Fig. E11). While some of the muscle activations were closer to the forward dynamics optimal excitations than when the excitations were left unbounded, differences still existed, especially at the smaller muscles of the foot (Figs. E12 and E13).

The time required to complete each optimization of 130% of a gait cycle, where each time step was equal to 1% of the gait cycle, was approximately 15 minutes. An area of future work will include testing different optimization algorithms (e.g., gradient based vs. global methods) to examine their optimization performance. The current results suggest that this type of inverse/forward dynamics algorithm is a promising technique to develop subject-specific muscle-actuated simulations.
Figure E2: Inverse/Forward optimization kinematic tracking when excitations were bound based on ±0.1 the original optimal forward excitation values.
Figure E3: Inverse/Forward optimization ground reaction force tracking when excitations were bound based on ±0.1 the original optimal forward excitation values. Green is ± 2 standard deviations of the experimental data.
Figure E4: Inverse/Forward optimization right leg muscle activations when excitations were bound based on $\pm 0.1$ the original optimal forward excitation values.
Figure E5: Inverse/Forward optimization left leg muscle activations when excitations were bound based on ±0.1 the original optimal forward excitation values.
Figure E6: Inverse/Forward optimization kinematic tracking when excitations were unbounded.
Figure E7: Inverse/Forward optimization ground reaction force tracking when excitations were unbounded. Green is ± 2 standard deviations of the experimental data.
Figure E8: Inverse/Forward optimization right leg muscle activations when excitations were unbounded.
Figure E9: Inverse/Forward optimization left leg muscle activations when excitations were unbounded.
Figure E10: Inverse/Forward optimization kinematic tracking when excitations were on/off bounded based on optimal forward dynamics excitations.
Figure E11: Inverse/Forward optimization ground reaction force tracking when excitations were on/off bounded based on optimal forward dynamics excitations.
Figure E12: Inverse/Forward optimization right leg muscle activations when excitations were on/off bounded based on optimal forward dynamics excitations.
Figure E13: Inverse/Forward optimization left leg muscle activations when excitations were on/off bounded based on optimal forward dynamics excitations.
References


Vita

Jessica Lynn Allen was born in Jacksonville, Florida and attended Allen D. Nease High School in Saint Augustine, FL. After graduating in 2002, she entered the University of Florida in Gainesville, Florida where she majored in Mechanical Engineering with a minor in Business Administration and earned a Bachelor of Science degree in December 2006. From 2004 to 2006 she performed undergraduate research in the Orthopeadic Biomechanics Lab at the University of Florida. During the summer and fall of 2005, she worked at Zimmer, GmbH in Winterthur, Switzerland. During the summer of 2007, she worked in the Human Motor Performance Lab at the University of Florida before entering the Graduate School at the University of Texas at Austin in September 2007 in the Neuromuscular Biomechanics Laboratory. In August of 2009, she earned a Master of Science in Engineering. Jessica’s graduate research has focused on using experimental and simulation analyses to understand impaired muscle coordination during walking post-stroke.

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