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Muscle Contributions to Balance Control, Propulsion and Leg Swing during Healthy and Post-Stroke Walking

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Muscle Contributions to Balance Control, Propulsion and Leg Swing during Healthy and Post-Stroke Walking

by

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Dedication

This dissertation is dedicated to my husband and my family.

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I would like to thank Dr. Neptune for not only helping me grow as a researcher but also caring about me as a person. I am grateful to work in such a supportive lab environment where I have had opportunities to be creative and explore areas that are interesting to me while receiving unparalleled training for my future career.

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Abstract

Muscle Contributions to Balance Control, Propulsion and Leg Swing during Healthy and Post-Stroke Walking

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Human walking requires complex muscle coordination to produce important biomechanical functions such as balance, forward propulsion and leg swing. For healthy individuals, these tasks are often accomplished effortlessly. However, for individuals post-stroke, balance, propulsion and leg swing can be compromised. Thus, the overall goal of this research was to understand how healthy individuals respond to altered balance control via mediolateral foot placement perturbations and how specific muscles contribute to propulsion and leg swing deficits in individuals post-stroke.

Controlling mediolateral foot placement is critical to maintaining balance in the frontal plane, but can be difficult post-stroke. Thus, we investigated how healthy individuals maintain their balance after mediolateral foot placement perturbations to compare to individuals post-stroke. We found that participants responded to medial foot placement perturbations using lateral hip and ankle strategies and lateral foot placement perturbations using a lateral ankle strategy, but did not use a plantarflexion strategy following either perturbation. Modeling and simulation analyses further revealed changes in hip and trunk muscle contributions to foot placement, suggesting a coordinated response

of the trunk and bilateral hip abductor muscles. On average, changes in muscle contributions to mediolateral ground reaction forces, torso power, and frontal plane external moments were small. These results highlight the responses of healthy individuals to altered balance control via foot placement perturbations.

Individuals post-stroke often experience propulsion and knee flexion deficits, leading to slow walking speeds and stiff-knee gait. These deficits may have several underlying causes. Thus, modeling and simulation analyses of individuals post-stroke were used to identify muscle contributions to propulsion deficits, including excess braking from the vasti, plantarflexor braking, low plantarflexor output and reliance on compensatory mechanisms. Moreover, higher vasti contributions to braking in pre-swing predicted lower knee flexion. While the rectus femoris and iliopsoas did not directly contribute to lower knee flexion acceleration in pre-swing compared to contributions from the vasti, in some cases, the rectus femoris absorbed more power and the iliopsoas contributed less power to the paretic leg. These results highlight the heterogeneity of the post-stroke population and the need to identify individual causes of walking deficits to improve rehabilitation outcomes.

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Chapter 1: Introduction

During human walking, muscles coordinate to perform essential biomechanical subtasks such as body support, forward propulsion and leg swing (Neptune et al., 2001, 2004). In addition, walking is inherently unstable in the frontal plane and requires active balance control (e.g. MacKinnon and Winter, 1993). Usually, healthy individuals can accomplish these complex tasks easily. However, individuals post-stroke have reduced muscle coordination complexity which correlates with deficits in walking performance (Clark et al., 2010) and balance control (e.g. Brough et al., 2019). Studying the underlying muscle and joint level contributions to biomechanical subtasks and balance control in individuals with and without neurological injury can provide insight into developing evidence based rehabilitation practices.

Control of mediolateral foot placement is a critical to maintaining balance in the frontal plane (Bauby & Kuo, 2000; Patla, 2003). Healthy individuals control foot placement based on the dynamic state of the body (e.g. Bruijn and van Dieën, 2018; Rankin et al., 2014). While individuals with mild impairment post-stroke may still be able to modulate foot placement to recovery from balance perturbations (Haarman et al., 2017), individuals post-stroke demonstrate less active control of paretic foot placement than healthy controls based on observed correlations with mediolateral pelvis position (Stimpson et al., 2019) and stance-limb mediolateral position and velocity (Dean & Kautz, 2015). Individuals post-stroke walk with wider steps, which are associated with poor dynamic balance (Vistamehr et al., 2016, 2018) and are used by individuals who are at a greater risk of falling (e.g., Dean et al., 2007; Frame et al., 2020). Therefore, impaired control of foot placement may make individuals post-stroke more susceptible to falls.

Perturbation-based training can reduce fall risk in older adults (e.g. Gerards et al., 2017; Pai et al., 2014) and improve clinical balance measures among individuals post-stroke (Schinkel-Ivy et al., 2019), but may not effectively reduce falls outside the clinic (Mansfield et al., 2018). Thus, more research is needed to identify rehabilitation targets by understanding the specific mechanisms used for balance recovery from foot placement errors in healthy individuals. While some responses to mediolateral foot placement perturbations have been described (Miller et al., 2018; Rankin et al., 2014; Segal et al., 2015), most studies using mechanical perturbations have used surface (e.g. Afschrift et al., 2018; Madehkhaksar et al., 2018) or pelvis (e.g. Haarman et al., 2017; Hof et al., 2010; Vlutters et al., 2018) translations. Therefore, the goal of the study in Chapter 2 was to characterize the hip and ankle balance response mechanisms of healthy individuals to foot placement perturbations designed to simulate an error in foot placement that might occur during activities of daily living. The results from this work will provide insight into the complex mechanisms used to control balance in the frontal plane during walking.

The force from an external mechanical perturbation can cause changes to the observed kinetic and kinematics measurements, such as ground reaction forces and foot placement location, that are not due to active balance responses from muscles. Moreover, understanding muscle function is often counterintuitive due to dynamic coupling in which muscles can accelerate joints and body segments they do not cross (Zajac and Gordon, 1989). Modeling and simulation can be used to estimate individual muscle contributions to the biomechanical subtasks of walking (e.g. Zajac et al., 2002, 2003). Chapter 3 will use modeling and simulation to determine muscle contributions to balance responses following mediolateral foot placement perturbations, including mediolateral ground reaction forces and foot placement during subsequent recovery steps, as well as muscle contributions to

the overall balance response. Together, the results of Chapters 2 and 3 will serve as a basis for comparison with individuals post-stroke in future studies.

In addition to maintaining balance, successful walking requires generating sufficient forward propulsion. Increases in propulsion from the paretic leg following a stroke predict improvements in walking speed (Bowden et al., 2013; Hsiao et al., 2016). Walking speed is a critically important predictor of overall health outcomes and has even been called the "sixth vital sign" (Fritz & Lusardi, 2009). Moreover, increasing walking speed from velocities that allow only household mobility to those velocities that allow limited (>0.4 m/s) or full (>0.8 m/s) community ambulation result in improved quality of life (Schmid et al., 2007). Thus, measuring propulsion and propulsion symmetry can help identify how well the paretic limb recovers and is contributing to changes in walking speed (Bowden et al., 2006). Analyzing propulsion asymmetry helps identify the use of compensatory mechanisms to achieve a particular walking speed, which may limit long-term recovery. However, force plate measure of paretic propulsion alone cannot determine the underlying mechanisms for why propulsion deficits occur. For example, some individuals may even have higher paretic than nonparetic propulsion due to other compensation strategies (Awad et al., 2020). In contrast, modeling and simulation can identify individual muscle contributions to propulsion to determine the underlying causes of propulsion deficits in individuals post-stroke, which is a major focus of Chapter 4.

Following propulsion, individuals must initiate leg swing and produce sufficient knee flexion to achieve toe clearance during swing. However, reduced knee flexion is common following a stroke and may require compensatory movements at the hip to achieve toe clearance (Balaban & Tok, 2014). Compensations for reduced knee and ankle movement are metabolically costly (McCain et al., 2021) and failure to achieve toe clearance may result in a trip and fall. Low knee flexion is often attributed to pre-swing

and swing-phase overactivity of knee extensors (e.g. Akbas et al., 2020; Lewek et al., 2007), although stiff knee gait may also be caused by low push-off forces from the plantarflexors (Apti et al., 2016; Wang et al., 2017), low knee flexion velocity at toe-off (Anderson et al., 2004; Apti et al., 2016; Campanini et al., 2013; Goldberg et al., 2003) and braking forces in late stance (Dean et al., 2020). Thus, insufficient propulsion or inappropriate braking forces in late stance may not only affect walking speed but also knee flexion during swing (Dean et al., 2020). However, the contributions of individual muscles to reduced knee flexion and stiff knee gait are still unknown, as previous simulation studies have investigated how muscles to accelerate the knee during late stance (Goldberg et al., 2004; Reinbolt et al., 2008) and swing (Anderson et al., 2004; Piazza & Delp, 1996) but have not investigated differences in individuals post-stroke with and without stiff knee gait. Thus, a second focus of Chapter 4 is to investigate muscle contributions to pre-swing knee flexion in individuals post-stroke with and without stiff knee gait and healthy controls. This study is unique because of the sample size of participants post-stroke (n=15), which is uncommonly high for simulation studies and allows us to investigate a variety of causes of their observed walking deficits. These results will highlight the heterogeneity of the post-stroke population and provide a foundation for developing individualized interventions.

The overall goal of this research was to use both experimental and modeling framework to understand the mechanisms used to control balance, foot placement, propulsion and leg swing during walking. Understanding these mechanisms has implications for improving the design of evidence-based interventions for individuals post-stroke and with other types of neurological injury.

Chapter 2: Biomechanical Response to Mediolateral Foot Placement Perturbations during Walking

INTRODUCTION

The ability to control and maintain dynamic balance is critical to participating in activities of daily living.[1] A fear of falling is linked to reduced activity levels, depression and anxiety among older adults (Painter et al., 2012). Dynamic balance has been shown to be maintained passively in the sagittal plane, but active control is required to maintain balance in the frontal plane, largely through control of foot placement (e.g., Bauby and Kuo, 2000; MacKinnon and Winter, 1993). A number of studies have focused on the role of the swing leg gluteus medius in foot placement (e.g., Dean and Kautz, 2015; Rankin et al., 2014). However, a recent simulation study demonstrated that both swing and stance leg muscles are utilized to control foot placement (Roelker, Kautz, et al., 2019), highlighting the complex muscle coordination needed from both legs to control balance.

Foot placement has an important role in regulating whole-body angular momentum (*H)*, which is a measure used to assess dynamic balance (Neptune & Vistamehr, 2019). In the frontal plane, foot placement affects both the mediolateral and vertical moment arms through which the ground reaction forces (GRFs) produce an external moment about the center of mass, which is equal to the time rate of change of *H*. Frontal-plane *H* is tightly regulated during unimpaired walking (Herr & Popovic, 2008), but has a higher range (Nott et al., 2014; Vistamehr et al., 2016) and is less tightly regulated during single-leg stance among individuals with impaired balance control (Nott et al., 2014). Unlike clinical balance

[1] This chapter is based on the previous published work: **Brough, L. G**., Klute, G. K., & Neptune, R. R. (2021). Biomechanical response to mediolateral foot-placement perturbations during walking. *Journal of Biomechanics*, 116, 110213. I contributed to designing the study, collecting data, analyzing data and writing the manuscript. RN and GK contributed to conceptualizing the study and preparing the manuscript.

measures, *H* provides insights into the underlying biomechanical factors influencing dynamic balance, including foot-placement, GRFs and body segment motion (Neptune & Vistamehr, 2019).

Variations in mediolateral foot placement can lead to a loss of balance, but healthy individuals have the ability to compensate for altered foot-placement to maintain their balance. A combination of strategies can be used depending on the timing and severity of the balance perturbation, including stance leg lateral ankle, hip and ankle push-off strategies (Reimann, Fettrow, & Jeka, 2018). The lateral ankle strategy acts to shift the center of pressure (COP) location quickly to correct small errors in foot placement (Hof et al., 2007). Shifting the COP provided the fastest response following a visual perturbation (Reimann, Fettrow, Thompson, et al., 2018a), while foot placement could only be altered on the subsequent step. Using a passive prosthesis, individuals with lower-limb amputations cannot generate active ankle moments to shift their COP shift in response to foot-placement perturbations. As a result, amputees often use a hip strategy during single-leg stance to modulate their GRFs to maintain balance after a foot placement perturbation (Miller et al., 2018; Segal et al., 2015). Similarly, individuals use a hip strategy to maintain balance when they are prevented from using a COP shift (Otten, 1999). Others have shown during steady-state walking, even when a lateral ankle strategy is possible, the hip strategy still plays a dominant role in frontal-plane balance control (MacKinnon & Winter, 1993; Winter, 1995).

In addition to lateral ankle and hip strategies, an ankle push-off strategy can also be used to control balance (Reimann, Fettrow, Thompson, et al., 2018a). The ankle plantarflexors are important contributors to controlling frontal-plane balance during steady-state walking through their contributions to the GRFs (Neptune & McGowan, 2016). Impaired plantarflexor coordination is a predictor of poor balance control among

individuals post-stroke (Brough et al., 2019). In response to visual perturbations, an ankle push-off strategy was shown to be an effective mechanism to help restore balance (Reimann, Fettrow, Thompson, et al., 2018a). However, it is unclear if such a strategy would be used following foot-placement perturbations because of the altered interactions between foot placement and GRF generation by the plantarflexors. For example, the lateral moment arm of the vertical GRF would be decreased with a medial foot placement perturbation, thus reducing its potential to help control frontal plane *H*.

The purpose of this study was to identify the biomechanical responses of healthy individuals to medial and lateral foot-placement perturbations during walking. The perturbations were generated using a custom pneumatic device which moved the foot medially or laterally just prior to heel strike. We hypothesized that on the perturbed leg, individuals would compensate for medial (lateral) foot-placement perturbations with 1) a lateral (medial) COP shift, 2) a decreased (increased) hip abduction moment impulse, and/or 3) an increased (decreased) ankle plantarflexion moment impulse. We hypothesized that these responses would occur during single-leg stance. Characterizing responses to foot placement perturbations in healthy individuals can provide a basis of comparison for those with neurological deficits and impaired balance control.

METHODS

Data Collection

Fifteen young adults without neurological injury or mobility impairments gave informed consent to an IRB-approved protocol (Table 2.1). To determine their self-selected over-ground walking speed, participants performed three trials of a 10-meter walk test at their "comfortable, typical walking speed." Kinematic data were collected at 120 Hz using

a 10-camera motion capture system (Vicon, Oxford, UK) and a full-body set of 65 reflective markers. Kinetic data were collected at 960Hz from a split-belt instrumented treadmill (Motek, Amsterdam, Netherlands). Participants performed ten 30-45 second walking trials at a standard speed of 1.0 m/s and ten trials at their self-selected over-ground walking speed (20 trials total). Ten trials (five at each speed) included foot-placement perturbations. During the perturbation trials, two medial and two lateral perturbations were applied to random steps, resulting in 20 medial perturbations and 20 lateral perturbations for each subject. All trial conditions (speeds and perturbations) were randomized.

Table 2.1: Participant demographics and self-selected walking speeds.

Subject	Height (cm)	Mass (kg)	Age (years)	Gender	Self-Selected Speed (m/s)
1	161.0	58.8	24	F	1.28
2	166.0	62.4	25	F	1.49
3	166.0	56.9	25	F	1.48
4	180.0	75.3	27	M	1.38
5	171.5	60.8	28	F	1.50
6	188.0	83.0	27	M	1.46
7	179.5	68.7	28	F	1.20
8	191.0	67.7	26	M	1.14
9	184.0	71.5	26	M	1.21
10	183.0	80.4	26	M	1.31
11	157.0	46.9	20	F	1.24
12	189.0	78.4	32	M	1.19
13	161.5	51.9	19	F	1.32
14	178.5	64.1	21	F	1.38
15	174.0	72.8	22	F	1.40
Average (SD)	175.3 (11.0)	66.6 (10.6)	25.1 (3.4)		1.33 (0.12)

Perturbations

Foot-placement perturbations were performed using a custom pneumatic device (Segal & Klute, 2014). A compressed air tank was connected to the ankle via flexible tubes (Figure 2.1). An inertial measurement unit and microprocessor (Sparkfun, Niwot, CO) were used to identify gait events. Based on the average cadence of ten unperturbed steps, the microprocessor triggered solenoid valves (ASCO) to release compressed air 140ms prior to the expected timing of the perturbed heel strike. Air exited through elbow joints for 180ms, or until after heel strike occurred, producing a medial or lateral force of ~15N on the ankle to perturb foot-placement. The original system was modified with an additional valve so the participants were unaware of the direction or timing of the perturbations.

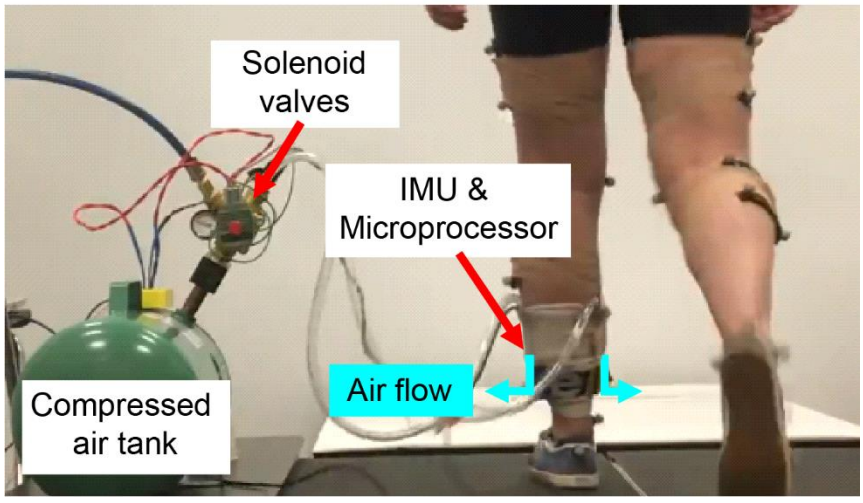

Figure 2.1: The perturbation device consisted of a compressed air tank with solenoid valves, flexible hoses and elbow joints that released air to produce a medial or lateral force on the ankle.

Data Analysis

Marker and force plate signals were low-pass filtered at 6Hz and 15Hz, respectively. A 13-segment inverse dynamics model was created for each subject (Visual 3D, C-Motion, Germantown, MD). Net internal joint moments generated by muscles were calculated using inverse dynamics. *H* was calculated by summing the angular momentum of each body segment about the whole-body center of mass. GRF impulses were calculated by integrating the GRF signals over stance and normalizing by body weight. Moment impulses were calculated by integrating the joint moments over stance and within four regions of stance (first double support, early ipsilateral single-leg stance, late ipsilateral single-leg stance, second double support). Joint moments were normalized by subject mass and *H* was normalized by subject mass, walking speed and leg length. Perturbation distance was defined peak divergence of the heel marker from the average unperturbed heel trajectory in the mediolateral direction following heel strike. COP excursion was calculated as the maximum lateral difference between the heel marker and COP location over stance in the lab coordinate frame. Cross-over steps were identified and removed from kinetic analyses. Kinetic and kinematic measures exceeding three standard deviations of each subject's average trajectory during that condition were considered outliers and excluded from further analysis.

Differences between perturbed and unperturbed steps were evaluated using linear mixed effects models. Separate models were created for medial and lateral perturbations throughout stance and each region of stance. Gait cycles began at the perturbed-side heel strike immediately following the perturbation. After confirming there were no significant differences in outcome measures, self-selected and standardized walking speeds were pooled for statistical analyses. Fixed effects were perturbation condition (perturbed or

unperturbed) and random effects were study subjects. Outcome measures included frontal-plane range of *H* (H_R), ankle inversion moment impulse, ankle plantarflexion moment impulse, hip abduction moment impulse, COP excursion and ground reaction force moment impulses. A linear mixed effects model was also created to test for correlation between ankle inversion moment impulse and COP excursion. For this model, fixed effects were inversion moment and random effects were study subjects. Statistical analyses were performed in MATLAB (Mathworks, Natick, MA).

RESULTS

Perturbation effect

Relative to the average unperturbed heel trajectory, perturbations caused an average of 3.6 ± 2.8 cm more medial and 3.6 ± 2.2 cm more lateral foot-placement compared to average unperturbed foot placement (Figure 2.2).

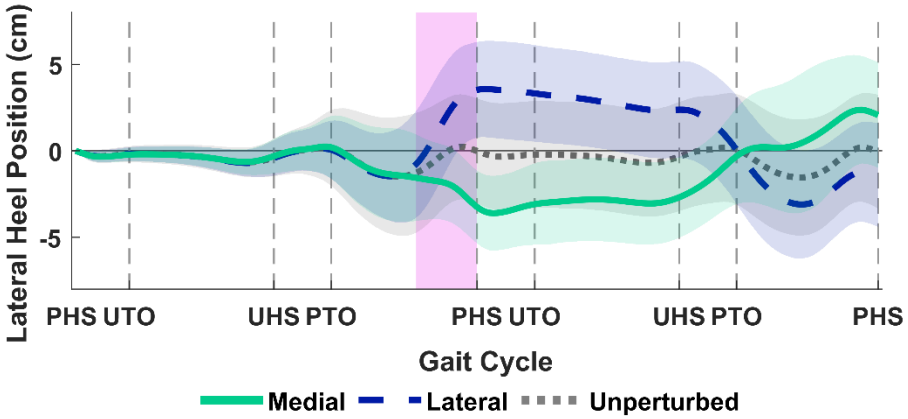

Figure 2.2: Average perturbed-side (left leg) lateral (+) and medial (-) heel marker position relative to unperturbed heel trajectory ± 1 SD before and after medially perturbed, laterally perturbed and unperturbed steps. The vertical shaded region indicates perturbation duration. Vertical dashed lines indicate perturbed-side heel strike (PHS) and toe-off (PTO) and unperturbed-side heel-strike (UHS) and toe-off (UTO).

Dynamic balance

H_R was 0.026 higher after medial perturbations (p<.001) and 0.002 lower after lateral perturbations (p=.002) compared to an unperturbed value of 0.048 (non-dimensional units; Figure 2.3). See Appendix Table A1 for results of all dependent measures.

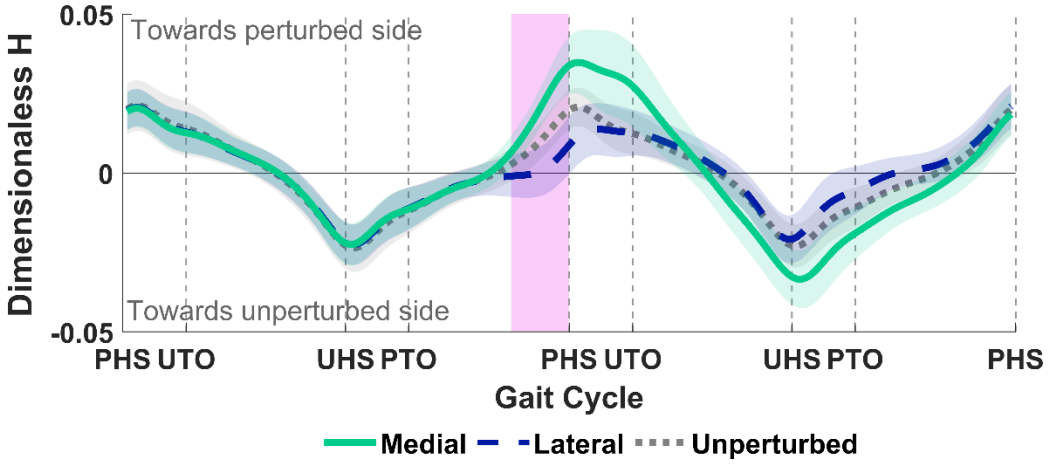

Figure 2.3: Average frontal-plane H ± 1 SD during the gait cycle before and after medially perturbed, laterally perturbed and unperturbed steps. The vertical shaded region indicates perturbation duration. Vertical dashed lines indicate perturbed-side heel strike (PHS) and toe-off (PTO) and unperturbed-side heel-strike (UHS) and toe-off (UTO).

Ground reaction forces

Over stance, the mediolateral GRF impulse decreased by an average of 0.58% BW-s after medial perturbations and increased by 0.92% BW-s after lateral perturbations compared to 3.30% BW-s for unperturbed mediolateral GRF impulse (p<.001 for both), while the vertical GRF impulse was not significantly different for any conditions (Figure 2.4).

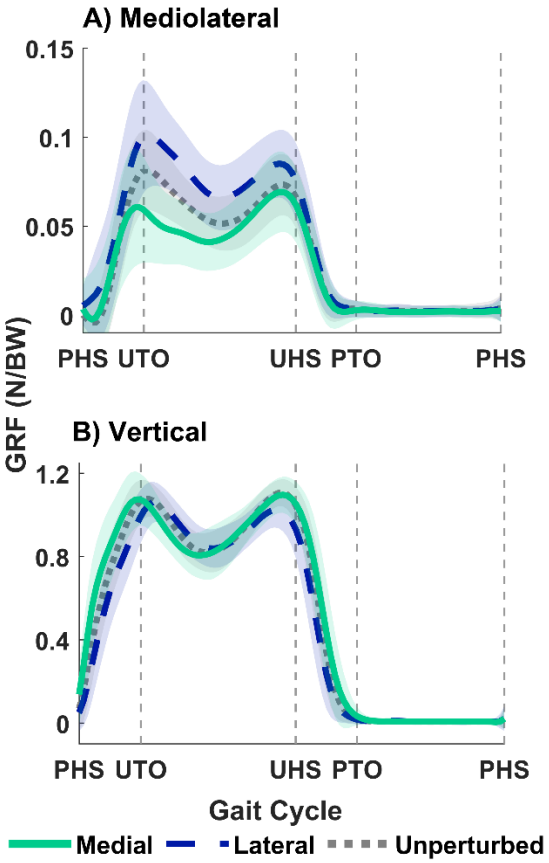

Figure 2.4: Average perturbed-side A) mediolateral and B) vertical GRFs ± 1 SD during the gait cycle after medially perturbed, laterally perturbed and unperturbed steps. Vertical dashed lines indicate perturbed-side heel strike (PHS) and toe-off (PTO) and unperturbed-side heel-strike (UHS) and toe-off (UTO).

Lateral ankle strategy

Over stance, total stance leg ankle inversion moment impulse increased by an average of 0.039 N-m-s/BW after medial perturbations and decreased by 0.032 N-m-s/BW after lateral perturbations compared to -0.001 N-m-s/BW for unperturbed moment impulse. Inversion moment impulses for medially and laterally perturbed steps were significantly different for all four regions of stance compared to unperturbed walking (all $p<.01$) (Figure

2.5A). COP excursion was 1.9 cm more lateral after medial perturbations and 2.3 cm more medial after lateral perturbations compared to 0.6 cm for unperturbed steps (p<.001 for both) (Figure 2.5B). The increases in ankle inversion moment were correlated to greater lateral COP excursion (p<.001, R^2 = 0.79) (Figure A1).

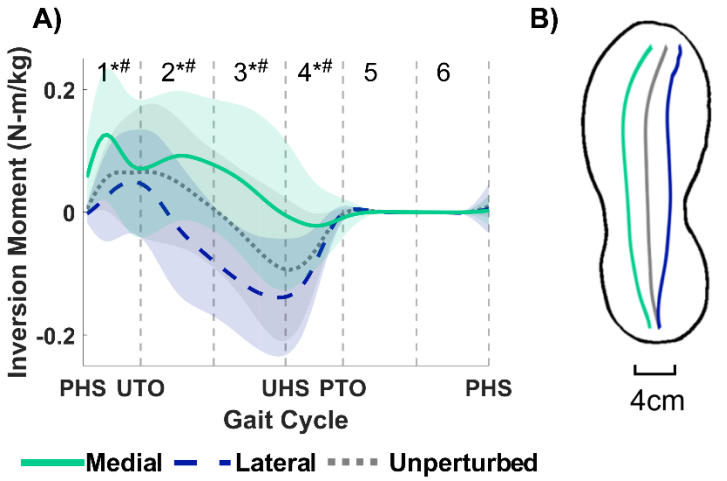

Figure 2.5: A) Average stance leg ankle inversion (+) and eversion (-) moment ± 1 SD for medially perturbed, laterally perturbed and unperturbed steps. Vertical lines indicate the six phases of the phases of the gait cycle. '*' denotes significance for medial perturbations and '#' denotes significance for lateral perturbations for moment impulse within each gait phase. Vertical dashed lines indicate perturbed-side heel strike (PHS) and toe-off (PTO) and unperturbed-side heel-strike (UHS) and toe-off (UTO). B) Average COP excursion relative to the heel marker for each perturbation condition.

Hip strategy

Over stance, the perturbed hip abduction moment impulse decreased by 0.07 N-m-s/kg after medial perturbations and decreased by 0.04 N-m-s/BW after lateral perturbations compared to unperturbed walking at 0.43 N-m-s/BW (p<.001 for both). Compared to unperturbed gait regions, the hip abduction moment impulse decreased during single-leg

stance and second double support after medial perturbations (p<.001 for all) and decreased slightly during in all regions after lateral perturbations (p<0.05 for all) (Figure 2.6A).

Ankle push-off strategy

The ankle plantarflexion moment impulse decreased by an average of 0.035 N-m-s/BW after medial perturbations (p<.001) and decreased slightly by 0.016 N-m-s/BW after lateral perturbations (p=.05) during stance compared to unperturbed walking at 0.440 N-m-s/BW (Fig. 6B). After lateral perturbations, plantarflexion moment impulses were slightly higher after lateral perturbations during the first two regions of stance and slightly lower for the second two (p<0.05 for all*)* and lower after medial perturbations during second double support (p<.001) (Figure 2.6B).

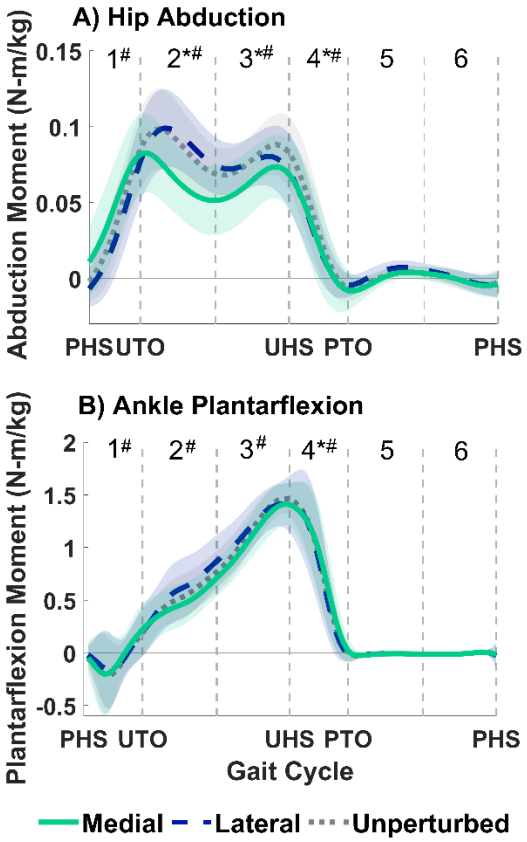

Figure 2.6: A) Average hip abduction (+) and adduction (-) moment, and B) average ankle plantarflexion (+) and dorsiflexion (-) moment ± 1 SD for medially perturbed, laterally perturbed and unperturbed steps. Vertical lines indicate phases of the gait cycle. '*' denotes significance for medial perturbations and '#' denotes significance for lateral perturbations for moment impulse within each gait phase. Vertical dashed lines indicate perturbed-side heel strike (PHS) and toe-off (PTO) and unperturbed-side heel-strike (UHS) and toe-off (UTO).

DISCUSSION

The purpose of this study was to identify the biomechanical responses of healthy individuals to medial and lateral foot-placement perturbations during walking. In agreement with our hypothesis, participants compensated for medial (lateral) perturbations with a lateral (medial) COP shift and decreased hip abduction moment impulse after medial

perturbations. However, contrary to our hypothesis the ankle plantarflexion moment impulse decreased slightly after medial perturbations and hip abduction moment impulse decreased slightly after lateral perturbations. While balance responses primarily occurred during single-leg stance as hypothesized, other small changes in ankle and hip responses occurred during first and second double support.

Perturbation effect on dynamic balance

Frontal-plane H_R increased (decreased) after medial (lateral) perturbations, which was consistent with previous work (Miller et al., 2018). The time rate of change of H ($\dot{H}$) is equal to the sum of the external moments acting about the body's center of mass (COM). During gait, these external moments are generated by the GRFs acting at a perpendicular distance away from the COM (i.e., the moment arm) and can be modulated by adjusting mediolateral and vertical GRF magnitude and moment arms (Figure 2.7). During steady-state walking, the external moments produced by each leg counteract the H produced by the opposite leg and rotate the body towards the contralateral side, keeping the integral of H over the gait cycle close to zero to prevent a lean. Thus, a change in H caused by a foot placement perturbation requires altered balance control to compensate for increases or decreases to net H.

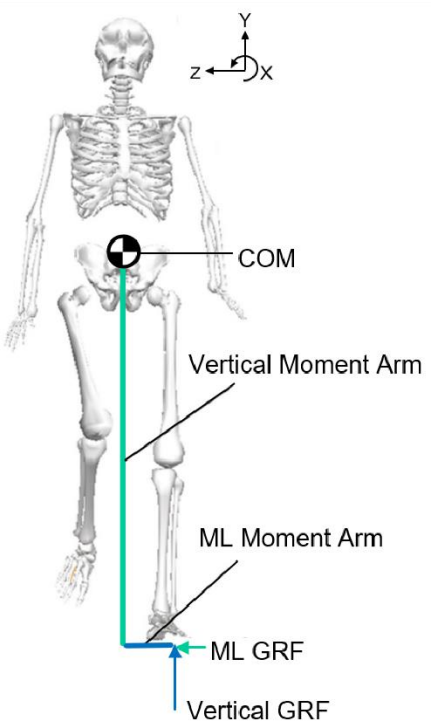

Figure 2.7: Vertical and mediolateral (ML) forces and moment arms contributing to frontal-plane H about the center of mass (COM). Adapted from Miller et al. (2018).

Higher H_R levels are indicative of balance deficits (Vistamehr et al., 2016), thus the increase in H_R after medial perturbations suggests a disruption to dynamic balance. Compared to unperturbed walking, more medial foot placement reduces the mediolateral distance between the COP and COM, resulting in a shorter mediolateral moment arm. The shortened mediolateral moment arm may cause an increase in H_R by reducing the potential of the vertical GRF to counteract the momentum generated by the unperturbed leg. In response, participants used a lateral ankle strategy to lengthen the mediolateral moment arm and a hip abduction strategy to reduce the external moment towards the perturbed side generated by the mediolateral GRF.

In contrast, H_R decreased after the lateral foot placement perturbations. Lower values of H_R are associated with better clinical balance scores (Vistamehr et al., 2016) and the wider base of support created by the lateral perturbation was unlikely to challenge balance. However, the change in *H* still required a response to keep net *H* over the gait cycle close to zero. Previous work suggested that a decrease in H_R after lateral foot-placement perturbations was caused by an increase in perturbed leg mediolateral GRF (Miller et al., 2018). While we also observed an increased perturbed-side mediolateral GRF after lateral perturbations (Figure 2.4A), the decrease in *H* occurred before heel strike and *H* did not continue to decrease during stance (Fig. 3), indicating that the perturbation itself caused *H* to decrease rather than the increased mediolateral GRF. Lateral perturbations also caused wider steps, which are associated with higher external moments and thus higher H_R (Nott et al., 2014) and are used by individuals at a greater risk of falling (e.g., Dean et al., 2007; Frame et al., 2020). Thus, a lateral perturbation would likely cause H_R to increase during the subsequent step unless active control occurred. However, participants were able to maintain low levels of H_R to control their balance in part by using a lateral ankle strategy.

Lateral ankle strategy

Studies have shown individuals may shift the location of the COP to compensate for a step that is too medial or lateral (Hof et al., 2007, 2010; Segal et al., 2015) in order to correct the mediolateral moment arm of the vertical GRF. This COP shift can be accomplished via an ankle inversion or eversion moment (Segal et al., 2015). Shifting the COP medially (laterally) shortens (lengthens) the mediolateral moment arm, thus increasing (decreasing) the external moment produced by the perturbed-side vertical GRF. As expected, subjects responded to medial (lateral) perturbations with an increased (decreased) ankle inversion moment impulse over stance and a correlated COP shift

opposite the perturbation direction. Small but significant ankle inversion moment responses occurred immediately after heel strike during double support. This early reaction despite the neural and electromechanical delays in muscular response to balance disruptions (Pijnappels et al., 2005) indicates that a reflex response occurred or that participants initiated an active response during the perturbation.

Hip strategy

COP shifts produced by the lateral ankle strategy are limited by the surface area of the foot. Thus, a hip strategy was also necessary after medial perturbations to restore pre-perturbation gait patterns. Lower hip abduction moments correlated with higher frontal plane H_R in previous work (Silverman et al., 2012), which suggested that increasing hip abduction moment might be an effective strategy to reduce H_R. In contrast, these participants decreased their hip abduction moments during single-leg stance.

This reduction in abduction moment counteracts the increased H_R following medial perturbations. Hip abductor muscles produce medially directed GRFs that rotate the body towards ipsilateral side in early and late stance (Neptune & McGowan, 2016). When the shortened mediolateral moment arm reduces the H generated by the vertical GRF towards the contralateral side, decreasing the mediolateral GRFs would counteract that change by decreasing H in the opposite direction (Figure 2.7). Because the hip abductors are the primary contributors to the mediolateral GRF during single-leg stance (John et al., 2012), reducing the hip abduction moment could accomplish this decrease in mediolateral GRFs. Indeed, we observed corresponding decreases in hip abduction moments and mediolateral GRFs after medial perturbations (Figure 2.4A).

Lateral perturbations also produced a slight decrease throughout double support and single-leg stance (Figure 2.6A). However, the mediolateral GRF increased despite the decrease in hip abduction moment (Figure 2.4), possibly due to the perturbation force or an unmeasured response. The small change opposite the direction hypothesized suggests that the lateral perturbation did not challenge balance to the extent of the medial perturbation. Previous work reported an increase in positive and decrease in negative hip abduction work after lateral perturbations and the opposite for medial perturbations (Miller et al., 2018), suggesting that a measure of work may identify some responses that were not clear in the moment impulse.

Ankle push-off strategy

Modifying the vertical GRF could be an additional strategy to modulate *H*. The ankle plantarflexors are primary contributors to vertical GRFs in late stance (Neptune et al., 2001; Anderson & Pandy, 2003) and frontal plane *H* (Neptune and McGowan, 2016). Thus, we hypothesized that ankle plantarflexor moments would be used to adjust vertical GRFs and compensate for altered mediolateral moment arms after perturbations. To overcome the shorter (longer) moment arm after medial (lateral) perturbations, we expected to see increased (decreased) ankle plantarflexion moments to increase (decrease) the vertical GRF. However, the opposite occurred after medial perturbations and minimal decreases were observed after lateral perturbations. Because there were no changes to the vertical GRFs and changes in the ankle plantarflexion moment were small (Figure 2.6B), we suspect that they occurred as a byproduct of the lateral ankle strategy: i.e., the ankle everter and inverter muscles used to accomplish the COP shifts also have plantarflexion moment arms, and vice versa for ankle plantarflexor muscles (Lee & Piazza, 2008). Thus, we do not believe that an ankle plantarflexor strategy was intentionally used to respond to

these perturbations. Unlike the ankle push-off strategy used to recover from frontal plane visual perturbations (Reimann, Fettrow, Thompson, et al., 2018a) and trips in the anterior direction (Pijnappels et al., 2005), an ankle push-off strategy may not be an efficient way to recover from a foot placement perturbation due to the relatively small moment arm and high force of the vertical GRF. Moreover, changing ankle push-off moments could interfere with anterior-posterior balance control. Future modeling work will investigate individual muscle contributions to the observed perturbation responses.

A potential limitation to this study was that the perturbation was intended to produce an imposed error in foot placement, which would subsequently require a balance response. However, based on the measured H values, the perturbation itself affected dynamic balance prior to foot placement. Moreover, because EMG data were not collected and causal relationships were not analyzed, we could not determine whether some changes to ground reaction forces and H were caused by the perturbation itself or by an active balance response. In future studies, modeling and simulation techniques should be used to determine individual muscle contributions to these biomechanical responses. Another limitation was that a learning effect could occur throughout data collection. In a post-hoc test, linear mixed effects models were created for medial and lateral perturbations to evaluate the relationship between H_R and step sequence. A group effect was found for medial perturbations (p=.04) and separate linear regressions were created for each subject, with four of fifteen participants demonstrating learning effects (p<.05). There were no group or individual learning effects for lateral perturbations. Because the effect was small and the majority of the subjects had no learning effect, this likely had little impact on our results and conclusions. Finally, medial perturbations were more likely to cause a cross-over step. However, due to the large number of steps that were analyzed (225 medial and

279 lateral), we do not believe the removal of cross-over steps affected our primary conclusions.

In summary, participants used both hip and ankle strategies to control H in response to medial and lateral foot placement perturbations. Medial perturbations caused H_R to increase, which produced a lateral ankle and hip response. Lateral perturbations caused H_R to decrease and primarily produced a lateral ankle response. Medial perturbations did not produce the expected ankle push-off response, instead producing a slight response in the opposite direction. Together, these results highlight the complex responses healthy individuals use to recover from foot placement perturbations and can provide a baseline for comparing balance recovery mechanisms for those with neurological deficits and impaired balance control.

Chapter 3: Muscle Response to Mediolateral Foot Placement Perturbations During Walking

INTRODUCTION

Walking requires the successful control of balance in the frontal plane, which is primarily accomplished via the generation of appropriate ground reaction forces and mediolateral foot placement (Bruijn & van Dieën, 2018; Neptune & McGowan, 2016). Previous work found that young healthy individuals react to an imposed error in foot placement with lateral hip and ankle strategies on the stance leg (Brough et al., 2021). After these initial stance leg reactions, further corrections may take place by altering the subsequent mediolateral foot placement (Hof et al., 2010). Both strategies work to maintain a low net moment about the body's center of mass, and subsequently a low range of frontal plane angular momentum, which are observed during healthy walking (Herr & Popovic, 2008). There is evidence that these responses are complementary strategies (Leeuwen et al., 2021) since contributions to foot placement are produced by both stance and swing leg muscles (Roelker, Kautz, et al., 2019).

Due to the mechanical effects of a foot placement perturbation, it is difficult to differentiate between muscle responses to the perturbation versus passive mechanical effects. For example, an observed decrease in angular momentum following lateral foot placement perturbations was originally attributed to a decrease in medial ground reaction forces (GRFs) modulated by hip abductor muscles (Miller et al., 2018), but was later determined to be caused by the perturbation itself (Brough et al., 2021). Likewise, changes to foot placement following a perturbation may be due to a combination of active and passive effects. Previous studies have attempted to separate passive and active responses by analyzing EMG (Hof & Duysens, 2013; Reimann, Fettrow, Thompson, et al., 2018a), sensory perturbations (e.g. Reimann et al., 2018) and simulation approaches (e.g Afschrift

et al., 2018). Simulation approaches provide estimates of muscle forces that cannot be measured experimentally and can be used to determine individual muscle contributions to movement (e.g. Zajac et al., 2003, 2002).

Previous simulation work has found that bilateral gluteus medius activity contributes to active changes in step width following medial and lateral treadmill perturbations at various points in the gait cycle (Afschrift et al., 2018). However, visual perturbations simulating a lateral fall at heel strike triggered a foot placement response that did not appear to be caused by a change in hip abduction angle or gluteus medius activity (Reimann et al., 2018). Unlike previous research investigating response to actual foot placement perturbations (Brough et al., 2021), participants responded to the visual perturbation by increasing the plantarflexion angle during perceived falls towards the stance leg and vice versa for perceived falls towards the swing leg (Reimann et al., 2018). These results highlight the specificity of balance responses to the type of perturbation. However, because these studies only examined a limited number of muscles, the contribution of other muscles to balance control is unclear.

The stance leg gluteus medius muscles are primary contributors to mediolateral GRFs (John et al., 2012) and act to rotate the body toward the ipsilateral side during stance (Neptune and McGowan, 2016). The swing and stance leg gluteus medius are also important contributors to foot placement (Roelker, Kautz, et al., 2019) and have been the focus of a number of perturbation studies (Afschrift et al., 2018; Hof & Duysens, 2013; Rankin et al., 2014). However, the contralateral plantarflexors, core muscles (e.g. erector spinae, obliques), and swing leg iliopsoas also play important roles in transferring power to the foot and pelvis during swing, both of which influence foot placement (Roelker, Kautz, et al., 2019). Thus, it is unknown how the swing and stance leg gluteus medius

coordinate with other muscles to perform both the lateral hip strategy and control foot placement.

The purpose of this study was to investigate individual muscle contributions to balance recovery strategies following medial and lateral foot placement perturbations. Specifically, we determined muscle contributions to mediolateral center of mass acceleration during stance and foot placement during the perturbed stance phase and subsequent step. We also determined muscle contributions to the overall balance response by calculating their contributions to the whole-body external moment ($\dot{\vec{H}}$). We hypothesized that: 1) the contralateral gluteus medius will contribute to the foot placement response, but the contralateral plantarflexor contribution will be negligible, 2) the stance leg gluteus medius would be the primary contributor to modulating the mediolateral GRF after medial perturbations and 3) the stance leg gluteus medius will contribute less to frontal plane angular momentum after medial perturbations only. The outcomes of this analysis will help identify balance response strategies among healthy individuals to provide the foundation for future studies analyzing balance control deficits in those at risk of falling and inform targeted interventions.

METHODS

Experimental data

Previously collected kinematic and kinetic data from ten young healthy participants (six female, mass = 64 ± 13 kg, age = 25.6 ± 3.8 years) were used to create the musculoskeletal models and simulations. As described previously, each participant performed 10 treadmill walking trials which included two medial and two lateral perturbations (Brough et al., 2021). The timing of the perturbation was determined by measuring the average cadence from ten previous steps to calculate the expected timing of

the next heel strike. The perturbation was applied 140ms before the estimated heel strike timing and the voltage signal triggering the perturbation was recorded to measure the actual timing. The force onset was determined from the controller voltage signal sent to the data acquisition system and synchronized with motion capture and force plate data. Representative medially perturbed, laterally perturbed and unperturbed gait cycles at each participant's self-selected speed (1.33 ± 0.14 m/s) were selected for further analysis (Sangeux & Polak, 2015), resulting in three simulations for each participant for a total of thirty simulations.

Musculoskeletal model and simulation

Using OpenSim 4.1 (Delp et al., 2007; Seth et al., 2018), a musculoskeletal model with 23 degrees of freedom and 92 muscle actuators on the lower body was scaled to fit the anthropometry of each participant. After the model scaling, an inverse kinematics analysis determined joint angles by minimizing the difference between experimental and model body segment markers. The unperturbed gait cycles were analyzed from left heel strike to left heel strike. For laterally and medially perturbed gait cycles, the previous left swing phase was simulated which included the perturbation before heel strike. The external perturbation force was added to the model at the measured vertical offset from the medial and lateral malleoli markers.

A residual reduction algorithm (RRA) reduced dynamic inconsistencies between model and experimental data by determining the joint moments required to track the joint coordinates calculated from inverse kinematics while adjusting the center of mass position, segment masses and joint kinematics to reduce residual forces and moments (Delp et al., 2007). RRA results were evaluated by comparing joint position results with inverse kinematics and evaluating the maximum and root mean square force and moment residuals

to ensure they were within acceptable ranges (Hicks et al., 2015). Static optimization was then used to estimate muscles forces that reproduced joint accelerations while minimizing the sum of muscle activations squared. Muscles were combined into groups for further analysis (Table 3.1).

Table 3.1: Muscle analysis groups

Name	Abbreviation	Muscles Included
Iliopsoas	IL	Iliacus, Psoas
Adductors	ADD	Adductor Longus, Adductor Brevis, Pectineus, Quadratus Femoris, Superior, Middle and Inferior Adductor Magnus
Erector Spinae	ERSPIN	Erector Spinae
Internal Obliques	INTOB	Internal Obliques
Rectus femoris	RF	Rectus femoris
Gluteus Medius	GMED	Anterior and Middle Gluteus Medius, Anterior and Middle Gluteus Minimus, Posterior Gluteus Medius, Posterior Gluteus Minimus
Biarticular Hamstrings	HAM	Semimembranosus, Semitendinosus, Biceps Femoris Long Head, Gracilis
Gastrocnemius	GAS	Medial Gastrocnemius, Lateral Gastrocnemius
Soleus	SOL	Soleus, Tibialis Posterior, Flexor Digitorum Longus
Tibialis Anterior	TA	Tibialis Anterior
Vasti	VAS	Vastus Intermedius, Vastus Lateralis, Vastus Medialis
Tensor Fasciae Latae	TFL	Tensor Fasciae Latae

Muscle contributions to balance strategies

Foot placement and hip strategies modulate $\dot{\vec{H}}$ (eq. 1) by altering the mediolateral moment arms and GRFs that determine the external moment about the body COM (Brough et al., 2021). Thus, we examined muscle contributions to each of these strategies. To

quantify muscle contributions to foot placement, a segment power analysis (Fregly and Zajac, 1996) was used to determine mediolateral muscle power delivered to the foot segment relative to the pelvis during representative perturbed and unperturbed steps (Roelker et al., 2019), which was integrated over the swing phase to determine each muscle's net mechanical work done on the foot. To quantify muscle contributions to the mediolateral GRF, muscle contributions to COM mediolateral acceleration were integrated over stance in representative unperturbed and perturbed gait cycles. A segmental power analysis was also used to determine muscle contributions to torso power.

To perform the induced acceleration and segment power analyses, a surface rolling constraint was applied to the feet during stance (Hamner et al., 2010). Then, the potential of each muscle to accelerate each joint, segment and body center of mass per unit force was calculated and multiplied by the corresponding muscle force determined from static optimization. Because foot placement was defined relative to the pelvis, muscle contributions to foot placement were defined as the difference between power contributions to the calcaneus and pelvis (Roelker, Kautz, et al., 2019). Muscle contributions to the net external moment in the frontal plane were calculated to determine their overall contribution to balance control as:

$$\dot{\vec{H}} = \vec{r}\, x\, \vec{F}_{\mathrm{GRF}} = \overrightarrow{M}_{EXT} \quad (1)$$

where $\dot{\vec{H}}$ is the time rate of change of whole-body angular momentum, $\vec{r}$ is the moment arm between the body center of mass and the center of pressure, $\vec{F}_{\mathrm{GRF}}$ are the ground reaction forces, and $\overrightarrow{M}_{EXT}$ is the external moment (Neptune and McGowan, 2016). Spatiotemporal measures including stance time and step width were also calculated during the representative gait cycles.

Statistical analysis

Paired t-tests were used to assess changes in summary dependent measures (step width, stance time, muscle work on foot placement) between unperturbed and medially perturbed gait cycles and between unperturbed and laterally perturbed gait cycles. Differences in continuous variables over time (muscle contributions to mediolateral GRFs, torso power and frontal plane external moments) were assessed using the one-dimensional statistical parametric mapping parallel of a paired t-test (Pataky, 2012), which identified normalized time points where variables from medial or laterally perturbed gait cycles differed significantly from variables from unperturbed gait cycles. A Bonferroni correction was used to correct for multiple comparisons, resulting in a significance level of $\alpha = 0.025$.

RESULTS

Spatiotemporal

Participants took a 3.4 ± 2.7 cm narrower step relative to the contralateral foot following medial perturbations (p=0.003) and a 6.4 ± 3.3 cm wider step in the first recovery step following lateral perturbations (p<0.001) compared to unperturbed steps (Figure 3.1). Relative to the COM position, participants had a 3.2 ± 1.5 cm narrower step in the first recovery step following medial perturbations (p<0.001) and a 3.5 ± 1.9 cm wider step following lateral perturbations (p<0.001) compared to unperturbed steps (Figure 3.1). In the second recovery step following medial perturbations, step width relative to the COM was not significantly different. However, in the second step following lateral perturbations foot placement relative to the COM was 2.7 ± 2.3 cm wider compared to unperturbed steps (p=0.005). Participants had a 32 ± 22 ms longer stance time following medial perturbations (p=0.001) and 33 ± 19 ms shorter stance time following lateral perturbations (p<0.001).

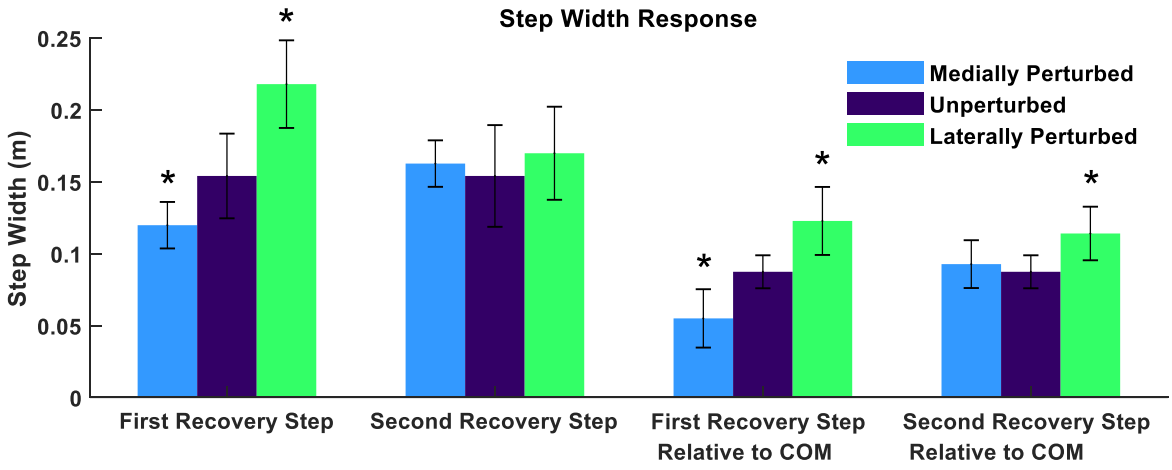

Figure 3.1: Step widths in the first and second recovery steps after the perturbed step relative to the opposite foot and relative to the center of mass. '*' indicates that the step is significantly wider or narrower than unperturbed steps.

Muscle contributions to foot placement

After medial perturbations, the ipsilateral gluteus medius did slightly less lateral work on the foot relative to the pelvis in the second recovery step only ($p<0.001$)(Figure 3.B). The ipsilateral erector spinae did less lateral work on the foot during both the first ($p=0.01$) and second ($p<0.001$) recovery steps (Figure 3.2A & B). The contralateral iliopsoas did less lateral work on the foot during the first recovery step only ($p<0.001$)(Figure 3.2A). The contralateral erector spinae did less medial work after both the first ($p=0.003$) and second ($p=0.009$) recovery steps (Figure 3.2A & B). No significant changes to work done by the ipsilateral internal obliques, tibialis anterior, iliopsoas, hamstrings, adductors, or contralateral gluteus medius were observed during either recovery step following medial perturbations (Figure 3.2A & B). The net muscle-tendon work done on the foot relative to the pelvis was unchanged during the first recovery step and less medial during the second recovery step ($p=0.006$)(Figure 3.2A & B).

After lateral perturbations, the ipsilateral gluteus medius did more lateral work on the foot relative to the pelvis during the first (p=0.004) and second (p<0.001) recovery steps (Figure 3.2A & B). The ipsilateral erector spinae did more lateral work on the foot during the first recovery step only (p=0.01) (Figure 3.A). The internal obliques did more lateral work during the first (p=0.001) and second (p=0.025) recovery steps (Figure 3.A & B). The ipsilateral tibialis anterior did more lateral work during the second recovery step only (p=0.009) (Figure 3.B). The ipsilateral hamstrings did more medial work during the second recovery step (p=0.017) (Figure 3.B). The ipsilateral adductors performed more medial work during both the first (p=0.025) and second (p<0.001) recovery steps (Figure 3.A & B). Finally, the contralateral gluteus medius performed more medial work on the foot during the first recovery step only (p=0.018)(Figure 3.A). There were no significant change in work performed by the contralateral erector spinae. The net muscle-tendon work done on the foot relative to the pelvis was more medial during the first recovery step and unchanged during the second recovery step (Figure 3.A & B). For all muscle contributions to foot placement, see Table B1 in Appendix B.

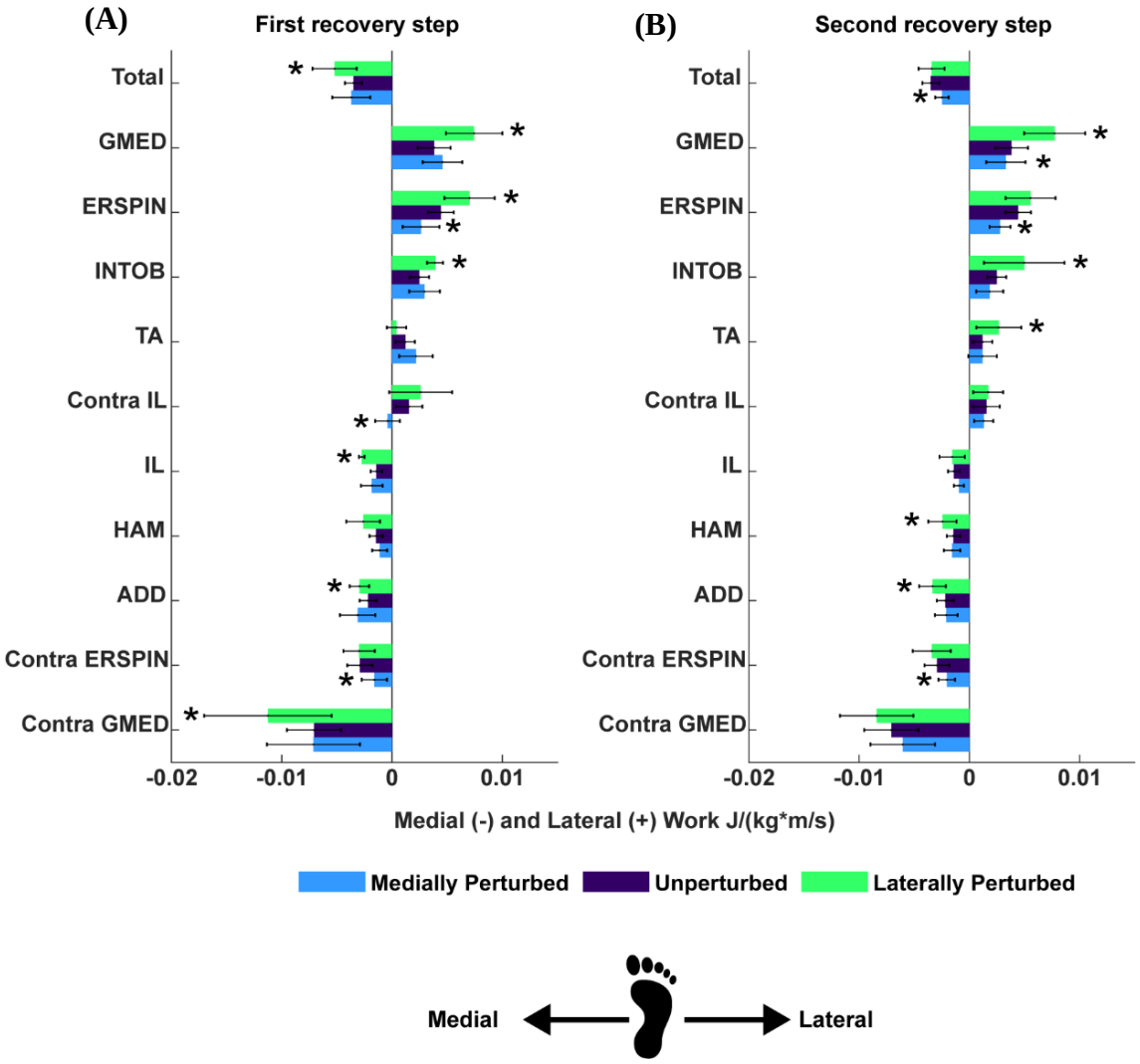

Figure 3.2: Primary contributors to muscle work on medial (-) and lateral (+) foot placement relative to the pelvis during swing. Error bars represent one standard deviation. Muscles are ordered from most medial to most lateral work on average from all the gait cycles analyzed. Contralateral muscles are on the stance leg side, while all other muscles are on the swing leg side. '*' indicates a significant difference between the perturbed and unperturbed condition ($p<0.025$).

Muscle contributions to mediolateral GRFs

There were no significant differences in muscle contributions to mediolateral GRFs in the stance phase following medial or lateral perturbations from major muscle contributors (Figure 3.3).

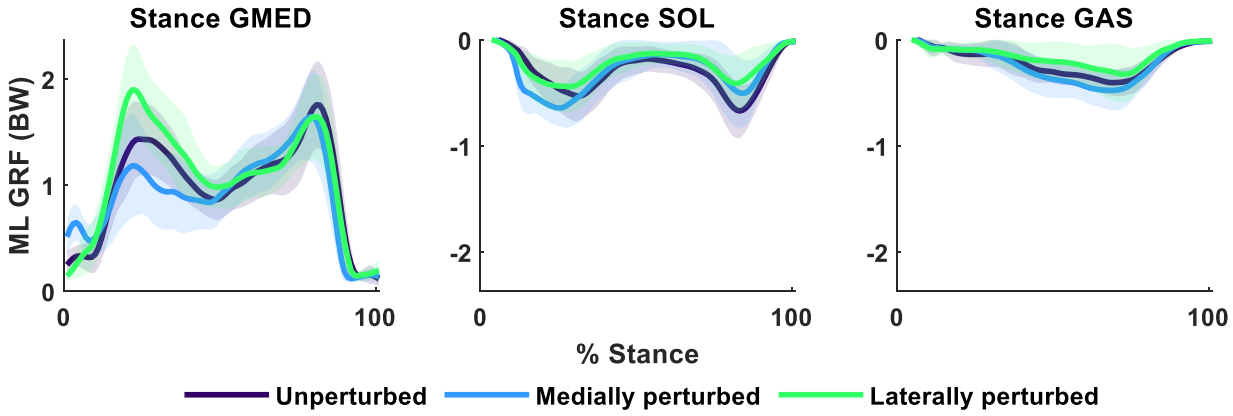

Figure 3.3: Muscle contributions to the mediolateral GRFs during the stance phase immediately following the medial and lateral perturbations for the three major contributors to the mediolateral GRF.

Muscle contributions to torso power

Stance leg soleus power delivered to the torso increased briefly in early stance following medial perturbations ($p<0.001$) and swing leg gluteus medius power increased briefly during the first double support phase ($p=0.012$) (Figure 3.4).

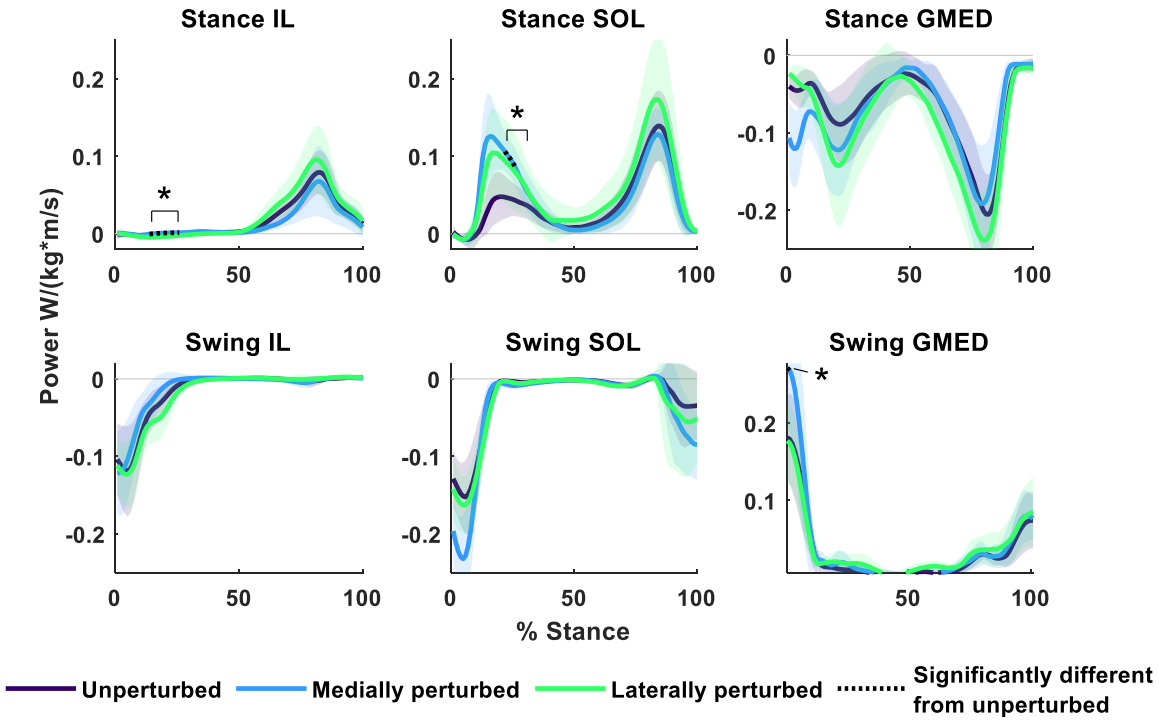

Figure 3.4: Individual muscle power delivered to the torso during the stance phase following medial and lateral perturbations from the six primary contributors to torso power. '*' and a dotted line indicate the period where the power after medial or lateral perturbations is significantly different from the unperturbed power.

Muscle contributions to frontal plane angular momentum

Following medial perturbations, there was a decrease in soleus contributions to the frontal plane external momentum during the second half of single leg stance compared to unperturbed steps (p<0.001)(Figure 3.5). There were no significant changes in muscle contributions to the frontal plane external moment between laterally perturbed and unperturbed steps.

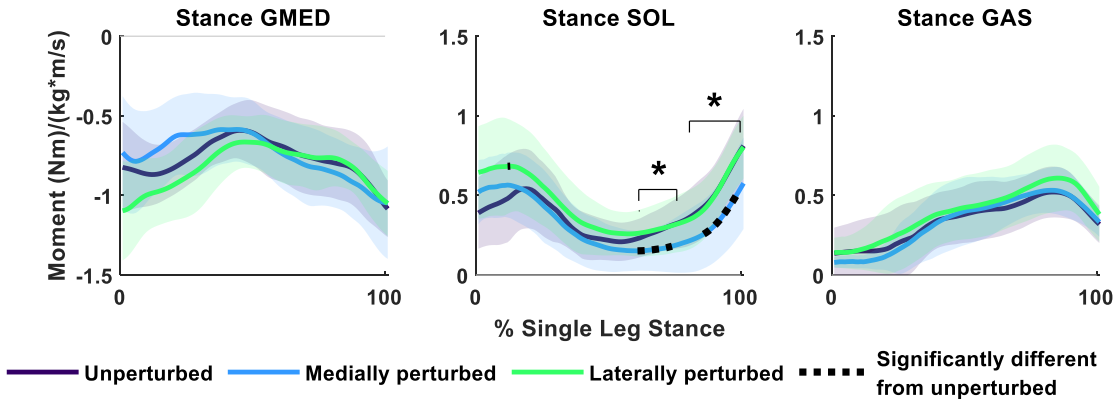

Figure 3.5: Individual muscle contributions to the frontal plane external moment during the stance phase immediately following medial and lateral perturbations for the three primary contributors to frontal plane angular momentum. '*' and a dotted line indicate a period where the external moment after medial or lateral perturbations is significantly different from the unperturbed external moment.

DISCUSSION

The purpose of this study was to investigate individual muscle contributions to overall balance control and recovery strategies following medial and lateral foot placement perturbations. Specifically, we determined muscle contributions to foot placement, mediolateral GRFs, trunk mechanical power and the frontal plane external moment.

Muscle Contributions to Foot placement

Participants took narrower and wider steps following medial and lateral steps, respectively (Figure 3.1), in agreement with previous work on foot placement perturbations (Segal & Klute, 2014). However, the net muscle work done on the foot was more medial after lateral perturbations during the first recovery step, and less medial after medial

perturbations during the second recovery step (Figure 3.2), suggesting that changes in step width were not due to active muscle control. Alternatively, because foot placement control depends on trailing leg position relative to the COM (Rankin et al., 2014), small changes to mediolateral COM position caused by the perturbations may cause swing phase muscles to work to bring the swing leg more medial after lateral perturbations and more lateral after medial perturbations.

We hypothesized that the stance gluteus medius but not the stance plantarflexor muscles would contribute to changes in foot placement following the perturbations. This hypothesis was partially supported, as the stance gluteus medius did more medial work on the foot after lateral perturbations, but there were no changes from the stance gluteus medius after medial perturbations (Figure 3.2A). The swing leg gluteus medius also did more lateral work on the foot after lateral perturbations. These results align with other studies demonstrating the importance of the swing and stance gluteus medius muscles in controlling foot placement (Afschrift et al., 2018; Hof & Duysens, 2013; Rankin et al., 2014; Stokes et al., 2017).

Another noteworthy result was the role of the trunk muscles in modulating foot placement. The swing side erector spinae increased lateral work on the foot after lateral perturbations and decreased lateral work after medial perturbations, and the swing side internal oblique increased lateral work on the foot after lateral perturbations (Figure 3.2). A lack of trunk control predicts poor balance and walking performance (Verheyden et al., 2006) and because of the significant mass of the trunk, trunk movement accounts for a large portion of whole body angular momentum in the frontal plane (e.g. Begue et al., 2021), but it appears that trunk control also influences balance control by affecting foot placement. A previous study using mediolateral perturbations to optical flow observed increases in trunk muscle activity (ipsilateral external oblique and external spinae) along

with concurrent increases in gluteus medius activity. The authors suggested that a postural adjustment was made by trunk muscles and the gluteus medius then compensated for changes in foot placement caused by the postural adjustment (Stokes et al., 2017). The results of the present study support the existence of a concurrent trunk and foot placement response, as there were changes in the work done by trunk muscles on the foot following the perturbations, but those changes were not in the direction of the net change in work. Thus, other muscles may have coordinated a response to counteract changes to foot placement caused by a trunk response and produce the appropriate foot placement to maintain balance.

In agreement with previous work (Roelker, Kautz, et al., 2019), the swing gluteus medius, erector spinae and internal obliques were primary contributors to lateral work on the foot relative to the pelvis and the stance gluteus medius and erector spinae were primary contributors to medial work on the foot relative to the pelvis. However, unlike Roelker et al. (2019), we found that the stance leg plantarflexors performed very little work on the foot and the swing iliopsoas was also a relatively small contributor. Differences between studies can be attributed to participant age (25.6 ± 3.8 versus 53.7 +/- 8.7 years) and walking speed (1.33 ± 0.14 m/s versus to 0.8 +/- 0.3 m/s), and we found that these results did not depend on whether static optimization (present study) or computed muscle control (Roelker et al., 2019) methods were used to estimate muscle activations. In addition, there was considerable variability in contralateral plantarflexor contributions to mediolateral foot work among participants in Roelker et al. (2019), with some having low contributions similar to the participants in the present study.

Muscle Contributions to Mediolateral GRF

We hypothesized that the stance leg gluteus medius would be a primary contributor to modulating the mediolateral GRF following the perturbations. We found that the stance gluteus medius, gastrocnemius and soleus were primary contributors to mediolateral GRFs in agreement with previous work (John et al., 2012; Pandy et al., 2010). However, while gluteus medius contributions to mediolateral GRFs trended lower after medial perturbations and higher after lateral perturbations in early single leg stance as expected (Figure 3.3), these reactions were not statistically significant and there was substantial variability between participants. We also considered changes in muscles contributions to trunk power, which like mediolateral GRFs, were largely insignificant. Thus, while we previously found a clear decrease in mediolateral GRFs after medial perturbations and increase after lateral perturbations (Brough et al., 2021), individual muscle contributions to changes in mediolateral GRFs were not evident.

Muscle Contributions to Frontal Plane External Moment

While contributions from the stance leg gluteus medius to the external moment (i.e., the time rate of change of whole body angular momentum) in the frontal plane trended towards decreasing after medial perturbations and increased after lateral perturbations in single leg stance, which mirrored the contributions to the mediolateral GRF, these results were not significant and not all participants demonstrated this response (Figure 3.5). Interestingly, only the soleus muscles had a significantly different contributions to the external moment after medial perturbations despite the altered mediolateral moment arm resulting from both medial and lateral perturbations, possibly because after corresponding center of pressure and COM adjustments, the moment arm was only slightly different from unperturbed walking.

Individual responses

Previously, we showed that on average, participants responded to medial perturbations with lateral ankle and hip strategies and lateral perturbations with a lateral ankle strategy, but neither perturbation produced a significant plantarflexor strategy (Brough et al., 2021). However, in a post-hoc analysis we observed distinct balance responses that did not conform to the average responses. For example, Subject 1 used the expected hip adduction strategy after medial perturbations to reduce the mediolateral GRF, but also increased the gastrocnemius contribution to the vertical GRF and also demonstrated a hip response after lateral perturbations (Figure 3.6). Subject 2 did not use the expected hip strategy after medial perturbations, instead increasing the gastrocnemius contribution to the vertical ground reaction force. Finally, Subject 8 demonstrated the expected response of a hip adduction strategy after medial perturbations and minimal stance leg changes after medial perturbations. Subjects 1 and 2 also had an increase in soleus contributions to the vertical GRF after both medial and lateral perturbations during early stance, which may reflect a reflex response to stiffen the joint. Responses for all participants can be found in Appendix B. Future work is needed to further understand why young, healthy participants would choose different balance response strategies, as we found they were not predicted by subject mass or walking speed.

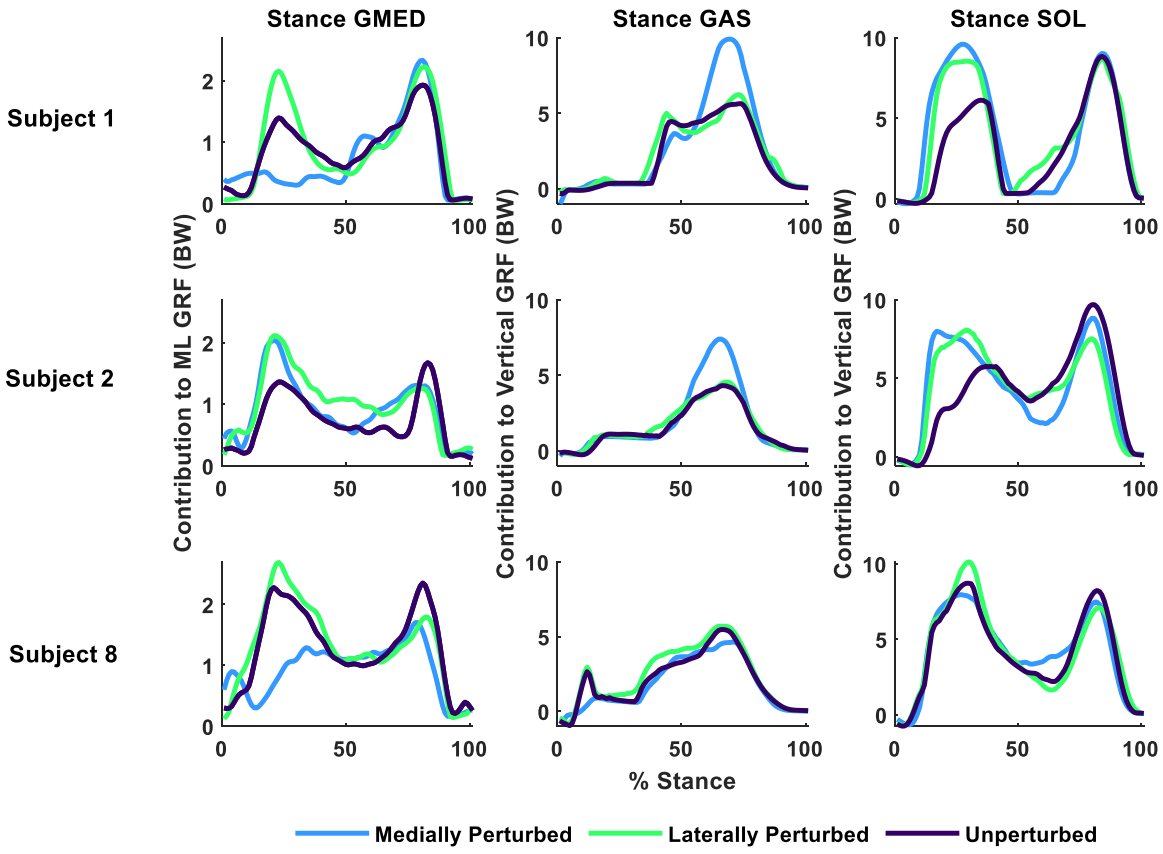

Figure 3.6: Muscle contributions to mediolateral and vertical GRFs from primary muscle contributors for three participants with different balance response strategies.

Limitations

A limitation to this study is that the time consuming nature of simulating 30 gait cycles limited the number of subjects and steps analyzed per subject. Despite choosing representative gait cycles for each participant, there was significant variability between participants. This variability limited our ability to generalize muscle contributions to the mediolateral GRFs, torso power and whole body angular momentum following the

perturbations. However, having fewer gait cycles made it clear that participants did not consistently use the same responses, which understanding why is an important topic for future study. In addition, the low number of trunk muscles in this model limits our ability to deduce the individual roles of trunk muscles in foot placement and balance control. However, similar to previous work on muscle contributions to trunk acceleration (Klemetti et al., 2014), we can interpret the roles of the internal obliques and erector spinae as the net effect of all the trunk muscles. Another limitation to this study is the use of static optimization, which does not include activation-deactivation dynamics, passive muscle forces or tendon compliance. Despite these shortcomings static optimization is robust and sufficient for estimating muscle forces during slow, low force activities such as walking. Further, we compared the results of static optimization and computed muscle control for this data and chose to use static optimization because computed muscle control estimated unrealistically high muscle activations during the swing phase, which would require placing limits on muscle activations without having EMG to inform those constraints.

Conclusions

In summary, participants responded to medial foot placement perturbations by reducing the lateral work done on the foot by the ipsilateral erector spinae and decreasing the medial work done on the foot by the contralateral erector spinae, but there were no clear foot placement responses from the ipsilateral and contralateral gluteus medius. After lateral perturbations, work done on the foot by the ipsilateral gluteus medius, erector spinae and internal obliques increased, and medial work done by the contralateral gluteus medius also increased. The net work on the foot was less medial following medial perturbations during the second recovery step and more medial following lateral perturbations during the first recovery step despite opposite changes in foot placement. There were no significant

changes in muscle contributions to mediolateral ground reaction forces, torso power, or frontal plane angular momentum, although we observed a number of different balance response strategies observed among participants. These results highlight the important role of the trunk muscles in controlling foot placement and suggest that trunk muscles play a multifaceted role in maintaining balance.

Chapter 4: Muscle Contributions to Pre-Swing Biomechanical Tasks Influence Swing Leg Mechanics in Individuals Post-Stroke during Walking

INTRODUCTION

Over 795,000 people in the United States experience a stroke each year and over half of stroke survivors over age 65 have reduced mobility (Virani et al., 2020). Regaining walking ability is an important goal of rehabilitation, as walking speed is a critical predictor of long-term health (Fritz & Lusardi, 2009) and stroke survivors who achieve limited or full community walking speeds report an overall higher quality of life than those who remain household ambulators (Schmid et al., 2007). Successful walking requires the execution of the critical pre-swing biomechanical subtasks of body propulsion and leg swing initiation, which are often impaired post-stroke (Bowden et al., 2006; Hall et al., 2011).

Deficits in these late stance subtasks may also have important implications for achieving adequate knee flexion during the swing phase. For example, a lack of push-off acceleration rather than over-activity of the knee extensors may be the primary cause of stiff knee gait in some individuals post-stroke (Campanini et al., 2013) and stimulating the plantarflexors in pre-swing increased peak knee flexion (Kesar et al., 2009). In addition, late stance phase braking forces in stroke survivors predict less knee flexion during swing, and late braking and low propulsion have been shown to predict circumduction (Dean et al., 2020). However, interestingly the total propulsive force is not correlated to knee flexion (Dean et al., 2020) and it is unknown if the relationships between braking, propulsion and swing phase kinematics are causal or correlative.

Braking and propulsion deficits are common in individuals post-stroke (Bowden et al., 2006). While healthy individuals produce braking only in the first half of stance and

propulsion in the second half, stroke survivors often have prolonged braking and low propulsion output, which predicts slower walking speed (Awad et al., 2020; Roelker, Bowden, et al., 2019). The plantarflexor muscles are primary contributors to propulsion (McGowan et al., 2009; Neptune et al., 2001) and decreased plantarflexor contributions to propulsion have been observed in stroke survivors (Allen et al., 2014; Nadeau et al., 1999; Peterson, Hall, et al., 2010), with post-training increases in walking speed being predicted by greater plantarflexor contributions to propulsion (Knarr et al., 2013). However, a lack of propulsion can occur not only because of low plantarflexor output, but propulsion can be offset by prolonged activity from the vasti, which are primary contributors to braking forces (Neptune et al., 2004). On average, individuals with impaired plantarflexor coordination do not have lower propulsion, but rather greater braking than individuals with normal plantarflexor coordination (Brough et al., 2019), which is likely due to co-activation of the plantarflexors and vasti muscles. Due to the characteristically high variability between stroke survivors, there are a number of mechanisms that can cause the propulsion deficits.

Stroke survivors also experience deficits in leg movement throughout swing (Balaban & Tok, 2014; Olney & Richards, 1996), with modeling studies having identified knee flexion velocity at toe-off as the primary contributor to peak knee flexion during swing (Anderson et al., 2004; Goldberg et al., 2003; Piazza & Delp, 1996). Knee velocity at toe-off may be diminished by late braking forces because muscles such as the vasti and rectus femoris that contribute to braking also contribute to knee extension and oppose leg swing initiation in late-stance (Neptune et al., 2004). Deficits in leg swing initiation may also be caused by decreased gastrocnemius contributions to leg-swing initiation, leading to lower knee velocity at toe-off and consequently less knee flexion during swing. For example, medial gastrocnemius contribution to knee flexion acceleration increased after

gait retraining (Knarr et al., 2013). However, a representative stroke survivor with a limited community walking speed had similar contributions from the gastrocnemius and vasti compared to a healthy control walking at the same speed but less power delivered to the leg in pre-swing by the iliopsoas (Peterson, Hall, et al., 2010). Thus, the high degree of variability in stroke survivors makes these results difficult to generalize and it is unknown how these deficits in leg-swing initiation impact swing phase kinematics.

Impaired knee flexion is often attributed to rectus femoris activity (Balaban & Tok, 2014; Tenniglo et al., 2014). In a previous modeling study, eliminating rectus femoris activity in pre-swing was more effective than eliminating rectus femoris activity in early swing for improving knee flexion (Reinbolt et al., 2008). The gluteus medius, vasti, and rectus femoris have the greatest potential to decrease knee flexion velocity in late stance, while the sartorius, gracilis, biceps femoris short head, gastrocnemius, iliopsoas and hamstrings have the greatest potential to increase knee flexion velocity in late stance (Goldberg et al., 2004), although it is unknown which muscles most affect pre-swing knee flexion velocity in stroke survivors.

Previous work has established the importance of pre-swing conditions to achieving adequate swing phase knee flexion. However, actual muscle contributions to propulsion, knee velocity and leg-swing initiation in stroke survivors and their relationship to swing-phase knee flexion has not been established. Moreover, most simulation studies are limited by a low number of participants, and therefore may not be generalizable to the overall stroke population. Thus, the objectives of this study were to: 1) analyze a large number of stroke survivors and determine the underlying causes of propulsion and braking deficits in late-stance, 2) identify primary muscle contributors to knee velocity and leg power in late stance, and 3) determine whether these muscle contributions to knee velocity and leg power predict knee flexion in swing. We hypothesized that 1) braking and propulsion asymmetries

would be caused by both low plantarflexor contributions to propulsion and high vasti contributions to braking, 2) vasti and plantarflexor contributions to propulsion and braking in pre-swing would predict swing phase knee flexion, 3) the rectus femoris would be a major contributor to knee extension in pre-swing in individuals with stiff knee gait, and 4) greater knee flexion would be correlated with greater power delivered to the leg in pre-swing. The outcomes of this work will highlight specific deficits in propulsion and leg swing initiation post-stroke and their implications for swing phase knee flexion, which will provide a basis for developing targeted walking interventions.

METHODS

Data collection

Kinematic, kinetic and electromyography data were collected from 15 stroke survivors (6 female, age: 56.1 ± 13.3 years, at least six months post-stroke) and 5 age-similar control subjects (2 female, age: 53.4 ± 7.3 years) (Table 4.1). Participants provided informed written consent to the Institutional Review Board approved protocol. Participants walked on a split-belt instrumented treadmill (Bertec, Columbus, Ohio) at their self-selected walking speed. Before data collection was initiated, participants practiced treadmill walking to get comfortable with the experimental setup and walked for at least 10 seconds to reach steady-state before each 30-second trial. Kinematic data were collected at 120Hz using a 12-camera motion capture system and 65 active markers (PhaseSpace, San Leandro, CA). Electromyography (EMG) data were collected (Motion Labs, Cortlandt, NY) at 1000 Hz from bilateral electrodes placed on the medial gastrocnemius, soleus, vastus medialis, lateral hamstrings, medial hamstrings, rectus femoris and tibialis anterior. Kinematic and ground reaction force (GRF) data were low-pass filtered at 6 Hz and 15Hz,

respectively. EMG data were high-pass filtered at 40 Hz, demeaned, rectified and low-pass filtered at 4 Hz.

Musculoskeletal models & simulations

A representative paretic leg gait cycle (left gait cycle for control subjects) for each participant was chosen for further analysis using the functional medial distance depth method (Sangeux & Polak, 2015). Using OpenSim 3.3 (Delp et al., 2007), a musculoskeletal model with 23 degrees of freedom and 92 muscle actuators (Delp et al., 1990) was scaled to match the anthropometry of each participant. An inverse kinematics analysis estimated generalized coordinates during the selected gait cycle by minimizing the difference between experimental and model markers (Delp et al., 2007). To reduce dynamic inconsistencies between the experimental GRFs and body segment kinematics, a residual reduction algorithm (RRA) fine-tuned the torso center of mass (COM) position, segment masses and joint kinematics (Delp et al., 2007) until residual forces and tracking errors were within acceptable ranges (Hicks et al., 2015). Computed muscle control (CMC) (Thelen & Anderson, 2006) then estimated muscle excitations that reproduced the experimental motion while minimizing the sum of excitations squared. Muscle excitation timing was constrained to approximately follow normalized EMG signals (Roelker, Kautz, et al., 2019).

Data analysis

Body propulsion and braking were calculated from the integral of the anterior and posterior GRF, respectively. Propulsion and braking asymmetries were defined as the percentage of paretic propulsion (PP) and paretic braking (PB), i.e., the paretic propulsive or braking impulse divided by the sum of paretic and nonparetic propulsive or braking

impulses (e.g., perfectly symmetric PP=0.5). Heel strike and toe-off were identified from GRFs using a threshold of 20 N. Knee flexion velocity at toe-off and peak knee flexion during swing were identified using joint kinematics from RRA. Pre-swing was defined as the double support phase between nonparetic (right) heel strike and paretic (left) toe-off. Individuals were placed into low or typical knee flexion groups depending on whether differences between the paretic and nonparetic knee range of motion was greater or less than 15º, respectively (Akbas et al., 2020; Sulzer et al., 2010).

Muscle contributions to biomechanical subtasks

Induced acceleration and segmental power analyses (Fregly & Zajac, 1996) were used to determine individual muscle contributions to braking, propulsion, knee flexion in pre-swing and leg swing initiation. Muscle contributions were then analyzed in functional groups (Table 4.2). To perform the induced acceleration analyses, a surface rolling constraint was applied to the feet during stance (Hamner et al., 2010) and muscle forces were determined using activations from CMC. Results were compared to experimental GRFs to ensure that the acceleration of the COM tracked the normalized GRFs. Muscle contributions to braking and propulsion were defined as each muscle's contribution to the anteroposterior (AP) acceleration of the body's COM integrated with respect to time over stance, normalized by walking speed. Muscle contributions to knee flexion during pre-swing were determined by integrating each muscle's contribution to knee flexion acceleration over time. To determine muscle contributions to leg swing initiation, a segment power analysis was used to determine the power delivered, absorbed or transferred to the leg by each muscle (Neptune et al., 2004). Muscle power was integrated over time to determine each muscle's mechanical work on the leg during pre-swing and was analyzed with and without normalizing by walking speed. Results were normalized by walking speed

because walking speed is correlated with AP GRFs, knee flexion, and muscle power. However, results for muscle work were also presented without normalization so the reader can interpret absolute muscle work in addition to work relative to walking speed.

Statistical Analyses

To test the hypotheses that greater vasti contributions to braking and lower plantarflexor contributions to propulsion would predict braking and propulsion asymmetries, linear regression models were created with PP and PB as the dependent measures and either soleus, gastrocnemius or vasti contributions to AP COM acceleration over stance as the independent measure. To test the hypothesis that pre-swing braking and propulsion would predict swing-phase knee flexion, linear regression models were created with peak knee flexion as the dependent measure and either total pre-swing AP GRF impulse (normalized by subject mass and walking speed), soleus, gastrocnemius or vasti contributions to AP COM acceleration in pre-swing (normalized by walking speed) as the independent measures. Similarly, to test the hypothesis that individuals with low knee flexion would have less work performed on the paretic leg by paretic muscles, a linear regression was created with peak knee flexion as the dependent measure and net muscle-tendon work performed on the paretic leg as the independent measure. Significance was defined as $\alpha = 0.05$.

Table 4.1: Participant characteristics

Stroke Survivors				
Participant	**Age**	**Mass (kg)**	**Treadmill Self-Selected Walking Speed (m/s)**	**Sex**
1	75	66.6	0.44	M
2	67	76.1	0.55	F
3	58	76.8	0.55	F
4	51	85.9	0.55	M
5	53	112.7	0.35	F
6	63	114.6	0.40	M
7	49	93.5	0.40	M
8	70	85.0	0.30	M
9	70	86.4	0.30	M
10	55	53.7	0.30	F
11	60	75.8	0.45	M
12	35	63.7	0.50	F
13	66	98.5	0.40	M
14	26	77.7	0.30	F
15	43	84.9	0.20	M
Average	**56.1 ± 13.3**	**83.5 ± 16.2**	**0.4 ± 0.1**	

Control Participants				
Participant	**Age**	**Mass (kg)**	**Walking Speed (m/s)**	**Sex**
C1	59	81.3	0.55	M
C2	40	79.7	1.10	M
C3	51	83.6	0.70	F
C4	59	80.1	0.50	M
C5	58	65.3	0.80	F
Average:	**53.4 ± 7.3**	**78.0 ± 6.5**	**0.7 ± 0.2**	

Table 4.2: Muscle analysis groups.

Muscle Group	Muscles
IL	Iliacus, Psoas
AL	Adductor Longus, Adductor Brevis, Pectineus, Quadratus Femoris
AM	Superior, Middle, Inferior Adductor Magnus
SAR	Sartorius
RF	Rectus Femoris
VAS	Vastus Medialis, Vastus Intermedius, Vastus Lateralis
GMEDA	Anterior and Middle Gluteus Medius, Anterior and Middle Gluteus Minimus
GMEDP	Posterior Gluteus Medius, Posterior Gluteus Minimus
TFL	Tensor Fasciae Latae
GMAX	Superior, Middle and Inferior Gluteus Maximus
HAM	Semimembranosus, Semitendinosus, Biceps Femoris Long Head, Gracilis
BFSH	Biceps Femoris Short Head
GAS	Medial Gastrocnemius, Lateral Gastrocnemius
SOL	Soleus, Tibialis Posterior, Flexor Digitorum Longus
TA	Tibialis Anterior, Extensor Digitorum Longus

RESULTS

Muscle contributions to braking and propulsion asymmetry

There was no correlation between propulsion asymmetry and vasti or soleus contributions to propulsion (Figure 4.1A & 1C). However, gastrocnemius contributions to propulsion were positively associated with propulsion asymmetry in stroke survivors (Figure 4.1B) (p=.005, R^2 = 0.47). Vasti, gastrocnemius and soleus contributions to AP GRFs did not predict braking asymmetry (Figure 4.1D-F), although the relationship between vasti contributions to braking and braking asymmetry did approach significance (Figure 4.1D) (p=0.07, R^2 = 0.23).

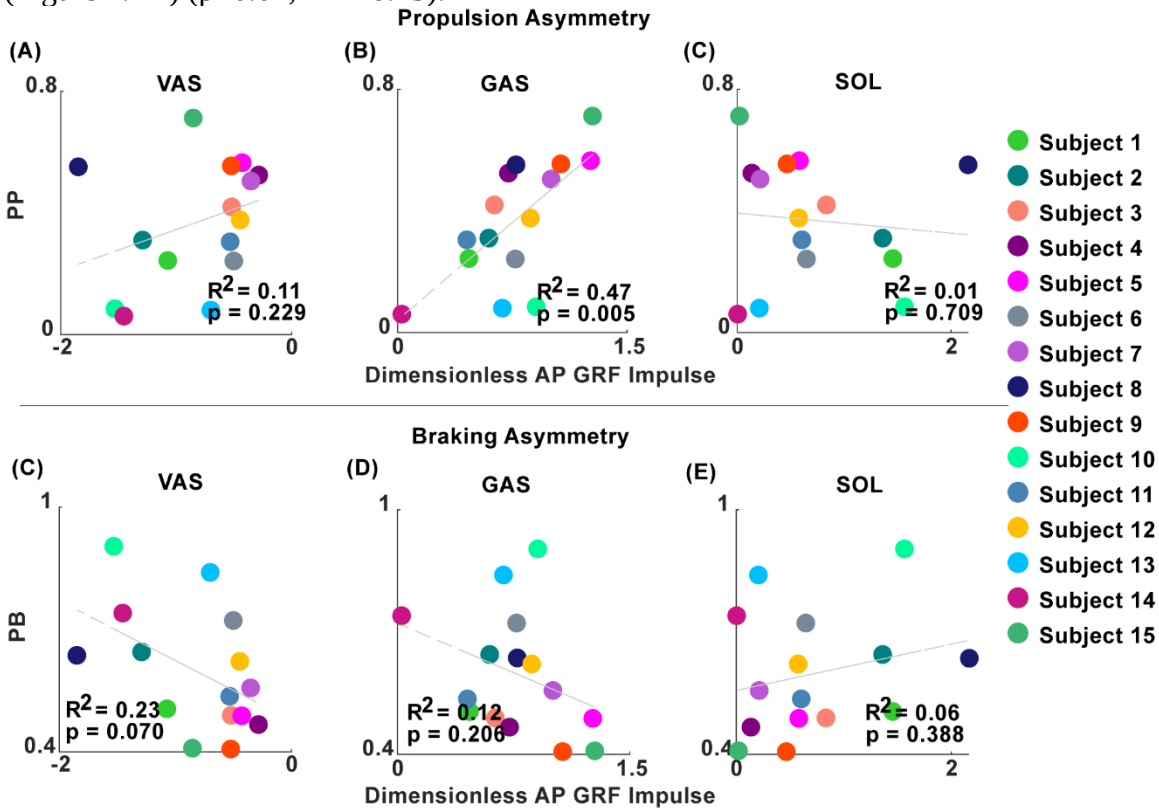

Figure 4.1: Predictors of Propulsion and Braking Asymmetry. Correlation between percentage paretic propulsion (PB) and percentage paretic braking (PB) and muscle contributions to propulsion and braking relative to walking speed in stroke survivors.

Four out of fifteen stroke survivors (27%) had greater plantarflexor contributions to propulsion on their paretic leg compared to their nonparetic leg (Table C1, see Appendix). All four produced more braking with the paretic vasti than the nonparetic vasti. A total of seven out of fifteen stroke survivors (47%) produced more braking with the paretic vasti than the nonparetic vasti. Two examples of muscles contributions to braking and propulsion are provided to illustrate propulsion asymmetries in opposite directions (Figure 4.2). Results for all individuals post-stroke can be found in Appendix C.

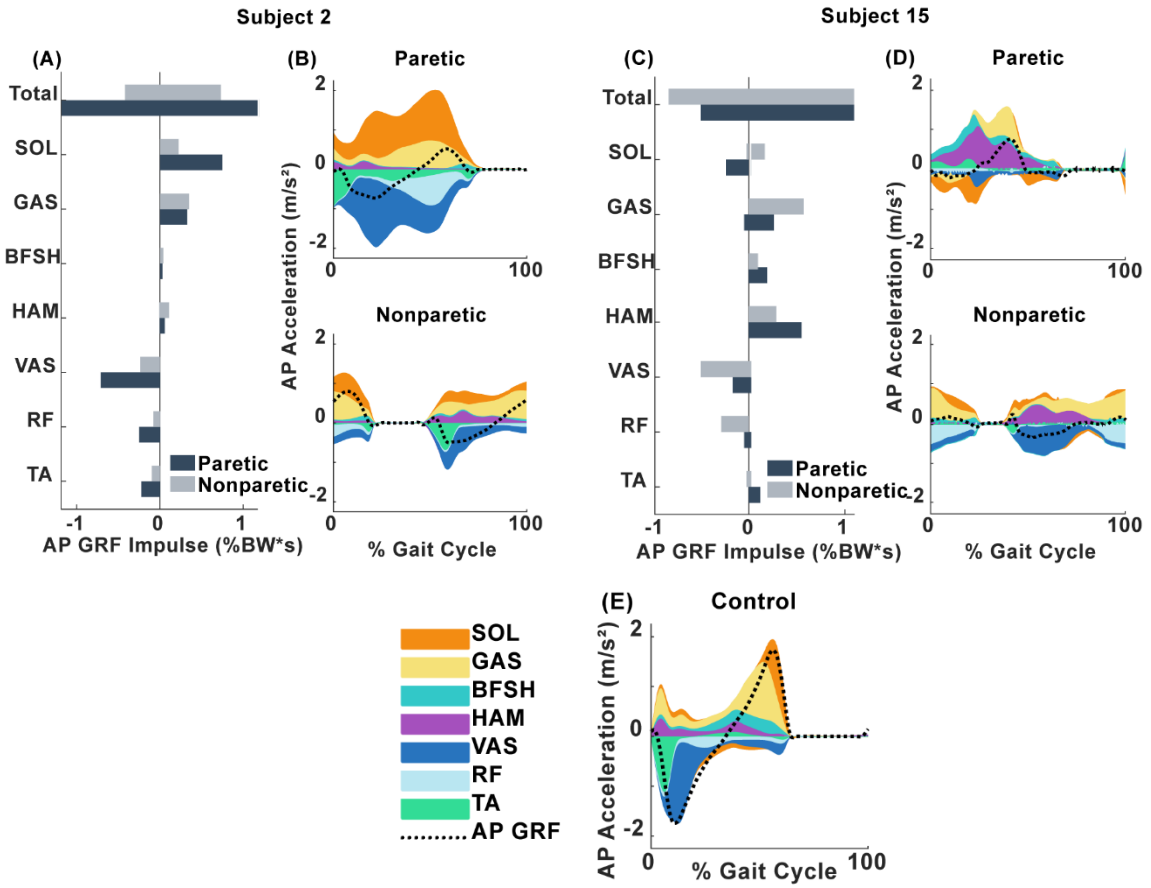

Figure 4.2: Muscle Contributions to Braking and Propulsion. A) Paretic and nonparetic muscle contributions to AP COM acceleration integrated over stance for Subject 2. B) Muscle contributions to AP COM acceleration over the paretic gait cycle, with contributions stacked on top of one another and shown relative to the normalized AP GRF (dotted line) for Subject 2. C) Paretic and nonparetic muscle contributions to AP COM acceleration integrated over stance for Subject 15. D) Muscle contributions to AP COM acceleration over the paretic gait cycle, with contributions stacked on top of one another and shown relative to the normalized AP GRF (dotted line) for Subject 15. E) Muscle contributions to AP COM acceleration over the left gait cycle, with contributions stacked on top of one another and shown relative to the normalized AP GRF (dotted line) for a representative control subject.

Relationship between braking and propulsion and swing phase knee flexion

There was moderate correlation between pre-swing AP GRF impulse and peak swing phase knee flexion (Figure 4.3A). Vasti contributions to braking in pre-swing relative to walking speed were correlated to knee flexion in swing, while soleus and gastrocnemius contributions to propulsion in pre-swing were not (Figure 4.3B-D).

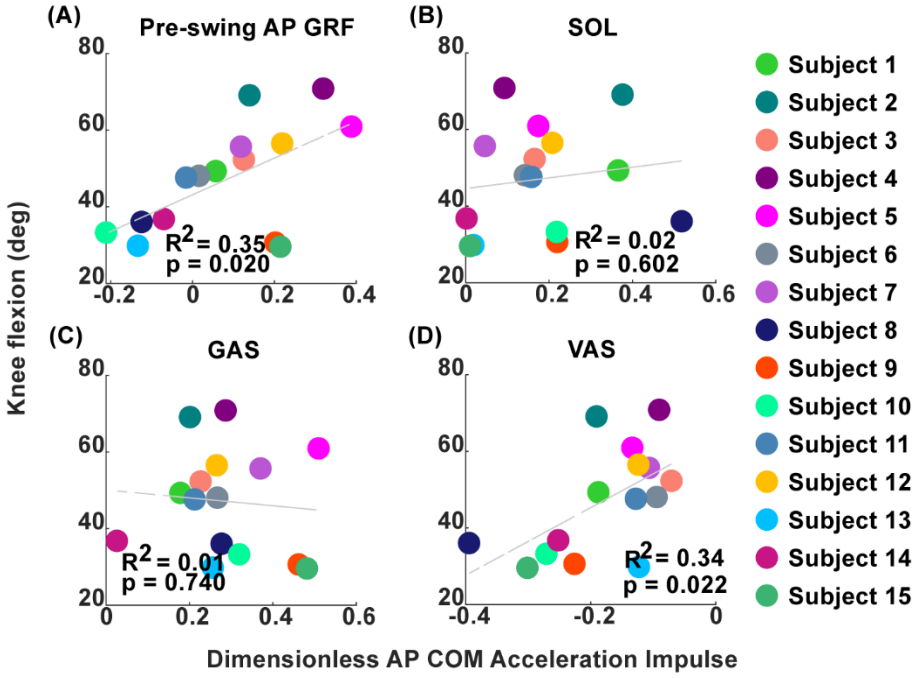

Figure 4.3: Predictors of Peak Knee Flexion. Correlation between peak knee flexion during swing and potential predictors of knee flexion, including A) the impulse of paretic AP GRFs in pre-swing normalized by subject mass, B-D) The AP COM acceleration impulse in pre-swing contributed by the SOL, GAS and VAS groups.

Primary contributors to knee flexion and extension acceleration in pre-swing

The iliopsoas was the greatest contributor to pre-swing knee flexion acceleration (Figure 4.4). On average, individuals with low knee flexion had greater contributions to knee flexion acceleration in pre-swing from the iliopsoas. However, that result was

primarily due to four individuals with very high iliopsoas contributions to knee flexion while the other five participants in the low knee flexion group had similar iliopsoas contributions as the typical knee flexion group (Figure 4.4). The rectus femoris was not a primary contributor to knee extension acceleration in pre-swing for any group. Rather, the vasti had a greater contribution to knee extension in the low knee flexion group compared to the typical knee flexion and control groups.

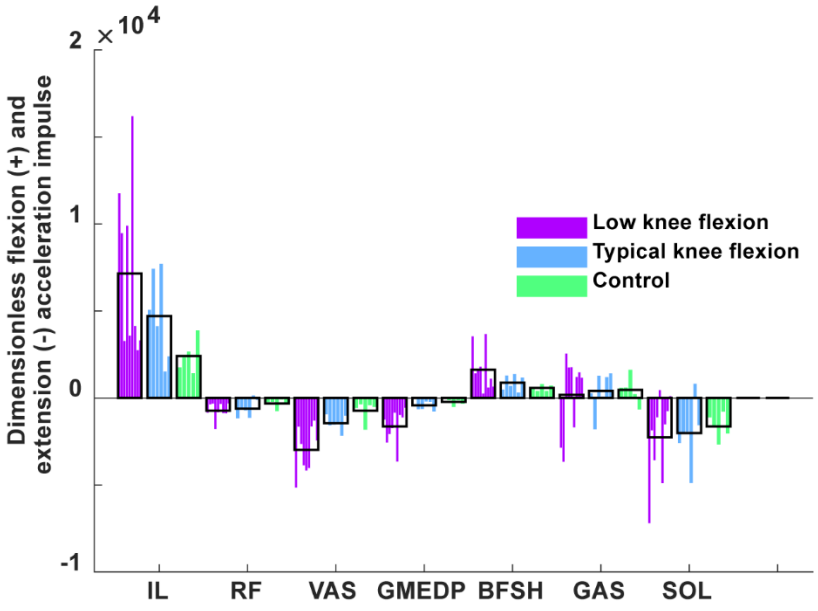

Figure 4.4: Muscle Contributions to Knee Flexion in Pre-Swing. Muscle contributions to knee flexion and extension acceleration integrated over pre-swing and normalized by walking speed. Participants are ordered from least to greatest knee flexion during swing.

Muscle contributions to leg swing initiation

Knee flexion was not predicted by total muscle-tendon work performed on the paretic leg in pre-swing regardless of whether work was normalized by walking speed

(p=0.18, R^2 = 0.13 and p=0.58, R^2 = 0.02 for normalized and not normalized, respectively). The low knee flexion group did have lower total muscle contributions to paretic leg swing on average, but there was high variability between participants in how this was accomplished (Figure 4.5). On average, the low knee flexion group had lower leg swing contributions from the iliopsoas than the typical and control groups regardless of walking speed (Figure 4.5). Both the low and typical knee flexion groups had more power absorption from rectus femoris and vasti on average than the control group relative to their walking speeds (Figure 4.5A). However, there was significant variability in the low knee flexion group, with some participants having very low and some having very high power absorption from the rectus femoris.

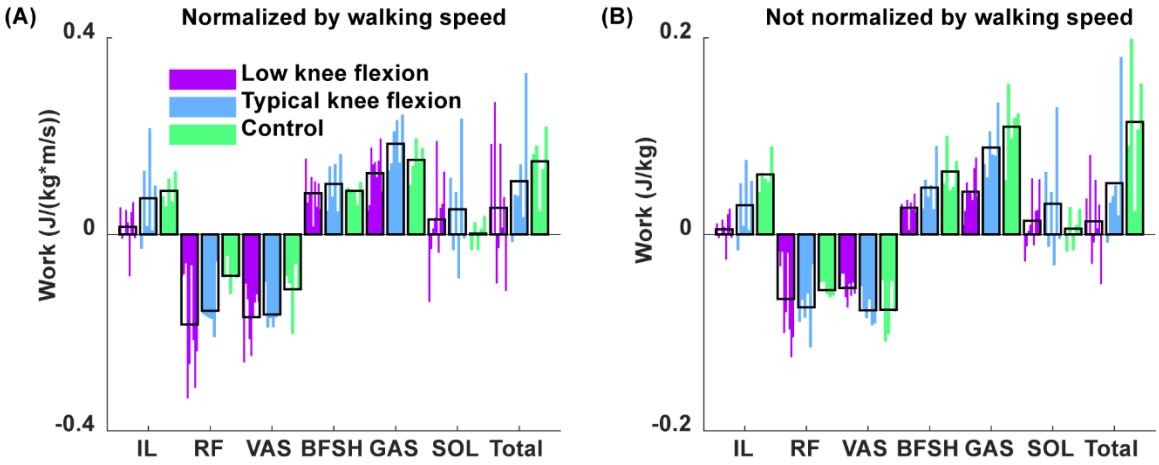

Figure 4.5: Muscle Contributions to Leg Swing Initiation. Work performed on the paretic leg in pre-swing A) normalized by walking speed, and B) not normalized by walking speed. Participants are ordered from least to most knee flexion during swing.

DISCUSSION

Muscle contributions to braking and propulsion asymmetry

The purpose of this study was to investigate impairments in early leg swing that may lead to stiff knee gait in individuals post-stroke. Specifically, we investigated pre-swing muscle contributions to braking, propulsion, knee flexion and leg swing initiation and the underlying relationships between pre-swing muscle function and swing phase knee kinematics in individuals post-stroke. We hypothesized that braking and propulsion asymmetry would correlate with both plantarflexor contributions to propulsion and excessive vasti contributions to braking. These hypotheses were partially supported. Gastrocnemius contributions to propulsion were correlated to propulsion symmetry. However, soleus and vasti contributions to AP GRF impulses were not correlated to braking or propulsion symmetry (Figure 4.1), although there was a strong trend towards an association between vasti and braking. These results are consistent with previous work showing that in individuals with moderate stroke, gastrocnemius and not soleus or vasti activity were correlated with AP GRF impulse (Turns et al., 2007). Moreover, it is well established that the plantarflexor muscles are important rehabilitation targets for improving paretic propulsion and walking speed (Roelker, Bowden, et al., 2019). For example, Participant 15 produced more paretic propulsion than nonparetic propulsion (PP = 0.71) but had lower net plantarflexor contributions to propulsion on the paretic side throughout stance, especially the soleus which did not contribute to propulsion, but instead produced braking in early and late stance (Figures 4.2C & D), likely due to a lack of leg extension (Peterson, Cheng, et al., 2010). Participant 15 compensated for these low plantarflexor contributions to propulsion by relying on the hamstrings for propulsion (Figure 4.2C & D). However, while paretic propulsion was high relative to the nonparetic leg, Participant 15

had low propulsion overall and thus walked slowly at 0.2 m/s (Table 4.1), indicating that compensation from the hamstrings is not an effective way to produce propulsion. These results can be contrasted with a representative healthy participant (Figure E), who produced braking in early stance with the vasti and propulsion in late stance with the plantarflexors as expected. These results demonstrate that while plantarflexor function is an important predictor of propulsion, paretic propulsion can be attained through compensatory mechanisms, such as from the hamstrings, which cannot be identified through force plate measurements of braking and propulsion.

The lack of correlation between vasti braking and propulsion asymmetry does not mean that the vasti do not contribute to propulsion deficits in stroke survivors, as extended braking from the vasti was a factor in propulsion asymmetry for multiple participants. For example, Participant 2 produced less paretic propulsion than nonparetic propulsion (PP = 0.24) due to prolonged braking from the vasti and rectus femoris despite producing more propulsion with the paretic plantarflexors (Figure 4.2A & B). Moreover, we previously showed that individuals with co-activation of the plantarflexors and other muscles such as the vasti had higher paretic braking but not lower propulsion (Brough et al., 2019), suggesting that propulsion asymmetries occurred not because of low plantarflexor contributions to propulsion but also because of excessive braking from other muscles. Thus, the knee extensor muscles can play a significant role in propulsion asymmetries, as they are primary contributors to braking (Liu et al., 2006) and can become overactive post-stroke (Yelnik et al., 1999). Vasti that are active late in stance should be an important target of rehabilitation, as they may affect both propulsion and knee flexion.

Relationship between braking and propulsion and swing phase knee flexion

We hypothesized that vasti and plantarflexor contributions to propulsion and braking in pre-swing would predict swing phase knee flexion. This hypothesis was partially supported. Overall, a lower pre-swing AP GRF impulse and greater vasti contributions to braking were correlated with less knee flexion in swing, but plantarflexor contributions were not (Figure 4.3). Dean et al. (2020) found that knee flexion in stroke survivors was predicted by late braking forces, but not net propulsive forces. These results can be explained by the fact that the vasti are primary contributors to both braking and knee extension and vasti activity is normally minimal in pre-swing for healthy individuals. However, in one sample of stroke survivors with stiff knee gait, 83% had inappropriately late vasti activity (Kerrigan et al., 1991). Other work has suggested that knee flexion deficits may be driven primarily by low ankle push off rather than knee extensors preventing knee flexion (Campanini et al., 2013). However, those conclusions were developed using a kinematic proxy of push off force (peak vertical acceleration of the malleolus marker), a methodological difference that may explain the differences in our studies. Moreover, it was concluded that individuals with lower knee flexion velocity than predicted by the malleolus acceleration model had stiff knee gait due to muscles preventing knee flexion rather than a low push-off force. However, it is likely that excess knee extensor activity could affect vertical malleolus acceleration and therefore these measures are not independent, leading some participants to be classified as having low knee flexion due to low push-off acceleration when knee extensor activity may have also contributed. In summary, our results suggest knee extensor activity in pre-swing predicts reduced knee flexion, while muscle contributions to propulsion in late stance do not.

Primary contributors to knee flexion and extension in pre-swing

We hypothesized that the rectus femoris would be a major contributor to knee extension in pre-swing in individuals with stiff knee gait. This hypothesis was not supported by our results. Rectus femoris contributions to knee extension were minimal in all groups (Figure 4.4), while the stiff knee group had greater knee extension contributions from the vasti. Previous work demonstrated that the while the gluteus maximus, vasti and rectus femoris had the greatest potential to accelerate the knee into extension during pre-swing, the gluteus maximus and rectus femoris produced significantly less force than the vasti in pre-swing, and produced less knee extension than the vasti and soleus, while the iliopsoas produced the most knee flexion (Goldberg et al., 2004). Previous work has also proposed that low knee flexion could be due to weak hip flexor muscles, as hip flexors are key contributors to knee flexion in healthy gait (Piazza & Delp, 1996; Yamaguchi & Zajac, 1990). Modeling studies have identified reduced iliopsoas function during pre-swing in individuals post-stroke with poor walking function (Hall et al., 2011). However, we observed that individuals in the low knee flexion group had greater iliopsoas contributions to knee flexion in pre-swing than healthy controls (Figure 4.4), although this average was dominated by four individuals, with others having lower than average iliopsoas contributions to knee flexion. This result may have occurred in part because the potential of the iliopsoas to flex the knee increases with reduced knee flexion. Interestingly, while Goldberg et al. (2004), Yamaguchi and Zajac. (1990) and Knarr et al. (2013) found that the gastrocnemius contributed to knee flexion in double support, Neptune et al. (2001) found that the gastrocnemius contributed to knee extension in double support. The results of this study suggest that the gastrocnemius may perform both functions (Figure 4.4) depending on the individual's kinematic state.

There is evidence that the rectus femoris does contribute to stiff knee gait in some but not all cases, as rectus femoris Botox injections for stroke survivors improved knee flexion for some individuals but not for those with more severe knee flexion deficits (Stoquart et al., 2008). Thus, there is a need to identify individuals who will benefit from treatments targeting the rectus femoris and others that may experience low knee flexion due to other problems such overactive vasti or weak hip flexors.

Muscle contributions to leg swing initiation

We hypothesized that greater knee flexion would be correlated with greater power delivered to the leg in pre-swing, but this hypothesis was not supported. While on average the low knee flexion group had less power delivered to the paretic leg in pre-swing compared to the typical knee flexion and control groups, there was significant variability between participants (Figure 4.5).

While the rectus femoris was not a primary contributor to knee extension in individuals with low knee flexion, it may still limit leg swing by absorbing power from the leg. Muscles act to either generate, absorb or transfer power between body segments (Fregly & Zajac, 1996; Zajac et al., 2002). In healthy individuals, the rectus femoris lengthens in late stance and absorbs power from the leg and redistributes it to the trunk (Neptune et al., 2004). Compared with healthy controls, a number of participants with low knee flexion had very high power absorption from the rectus femoris in pre-swing relative to their walking speed which would inhibit leg swing initiation (Figure 4.5), and thus limit the trajectory of knee flexion during swing.

The iliopsoas contributed less to leg swing initiation in both stroke groups on average compared healthy controls, but especially the low knee flexion group (Figure 4.5). These results are consistent with previous work showing lower power contributions from

the iliopsoas in pre-swing for stroke survivors compared to healthy controls (Peterson, Hall, et al., 2010). On average the gastrocnemius contributed less to leg swing initiation in the low knee flexion group compared to the typical knee flexion group (Figure 4.5), but contributed more to leg swing initiation in the typical knee flexion group when compared to healthy controls. These results of typical knee flexion group contrast previous work that found lower gastrocnemius contributions to leg swing initiation in stroke survivors (Peterson, Hall, et al., 2010). However, that study only analyzed two representative stroke survivors, while ours shows that substantial variability exists between participants. The gastrocnemius may have contributed more to leg swing initiation in the stroke group with typical knee flexion due to the co-contraction seen in some participants (e.g. Participant 2), where greater plantarflexor output was required to overcome excessive braking from the vasti in late stance.

Limitations

A potential limitation of this study is that stroke survivors often have high levels of muscle co-contraction (Clark et al., 2010; Lamontagne et al., 2000), which may be difficult to replicate in simulations using algorithms such as computed muscle control which minimize the sum of muscle activations squared (Thelen & Anderson, 2006). To address this concern, bilateral EMG was collected and muscle excitations were constrained to stay within bounds determined by experimentally collected EMG. Another limitation is that due to the high variability between individuals, it is difficult to generalize these results to the post-stroke population as a whole. Even with this limitation, few simulation studies of individuals post-stroke include a sample size as large as the present study, which demonstrates the variability between participants and highlights the many different causes of walking deficits. Future work will seek to develop clinical methods for identifying

underlying causes of propulsion and leg swing deficits to inform individualized rehabilitation strategies. Another limitation is that this study focused only on muscle contributions to propulsion, while appropriate leg extension angle at push-off also affects propulsion (Awad et al., 2020; Peterson, Cheng, et al., 2010; Roelker, Bowden, et al., 2019). It is likely that some of these participants experienced propulsion asymmetry in part due to leg positioning and future work should investigate causes of reduced leg extension. A final limitation is the interpretation of results could be affected by some control participants who had below average self-selected walking speeds and asymmetric walking. However, these controls were not used for statistical analyses and served to provide a reference for age-similar individuals without neurological injury rather than perfect walking.

Conclusions

We observed that some participants had paretic propulsion deficits due to low plantarflexor contributions to propulsion and/or excess vasti contributions to braking. Others appeared to produce sufficient or high paretic propulsion, but accomplished that propulsion via compensatory mechanisms such as a reliance on the hamstrings rather than appropriate plantarflexor activity. Greater vasti contributions to braking in pre-swing predicted lower knee flexion. The rectus femoris and iliopsoas did not directly contribute to lower flexion in pre-swing. However, in a number of cases the rectus femoris absorbed more power and the iliopsoas contributed less power from the paretic leg in pre-swing in individuals with low knee flexion. These results highlight the heterogeneity of the post-stroke population and the need to identify the underlying causes of propulsion and knee flexion deficits in each individual in order to improve rehabilitation outcomes.

Chapter 5: Conclusions

The overall goal of this research was to understand the mechanisms used to control balance, foot placement, propulsion and leg swing during walking using both experimental and modeling methods. The results have provided insight into how healthy individuals respond to mediolateral perturbations to foot placement, and the underlying deficits affecting propulsion and leg swing in stroke survivors. Understanding these mechanisms has implications for improving the design of evidence-based interventions for individuals post-stroke and with other types of neurological injury.

In Chapter 2, joint-level responses to mediolateral foot placement perturbations were assessed in young, healthy adults using an experimental framework. Medial perturbations caused the range of frontal plane angular momentum to increase, while lateral perturbations caused angular momentum to decrease. We found that participants responded to medial perturbations with an ankle inversion strategy to shift the center of pressure laterally. Participants also tended to reduce their hip abduction moment, likely to reduce the mediolateral ground reaction force in an effort to modulate angular momentum in the frontal plane. After lateral perturbations, participants responded with an ankle eversion strategy to shift the center of pressure medially but did not demonstrate a clear hip strategy. We had hypothesized that participants would also increase the plantarflexion moment after medial perturbations and decrease it after lateral perturbations. However, on average participants did not use this strategy. These results highlight the complex responses healthy individuals use to recover from foot placement perturbations and can provide a baseline for comparing balance recovery mechanisms for those with neurological deficits and impaired balance control.

In Chapter 3, musculoskeletal modeling and simulation was to investigate individual muscle contributions to the balance responses following medial and lateral foot

placement perturbations. Specifically, we analyzed muscle contributions to mediolateral foot placement in the steps following the perturbation, the mediolateral ground reaction force, trunk power, and the external moment in the frontal plane. After medial perturbations, the net muscle work done on the foot was less medial during the second recovery step and after lateral perturbations the muscle work was more medial during the first recovery step, despite opposite changes in foot placement. After medial perturbations, lateral muscle work done on the foot by the ipsilateral erector spinae and medial work done on the foot by the contralateral erector spinae both decreased, but there were no clear foot placement responses from the ipsilateral and contralateral gluteus medius. After lateral perturbations, work done on the foot by the ipsilateral gluteus medius, erector spinae and internal obliques increased, and medial work done by the contralateral gluteus medius also increased. However, there were no significant changes in muscle contributions to mediolateral ground reaction forces, torso power, or frontal plane angular momentum, although we observed a number of different balance response strategies among participants. These additional strategies included an increase in gluteus medius contributions to the mediolateral ground reaction force after lateral perturbations and an increase in gastrocnemius contribution to the vertical ground reaction force. Individual responses demonstrate strategies that were not obvious from group-averaged data, showing that even young, healthy individuals may use different strategies to recovery from foot placement errors. Moreover, these results highlight the role of the trunk muscles in modulating foot placement and suggest that trunk control has a multifaceted role in maintaining balance.

In Chapter 4, musculoskeletal modeling and simulation were used to investigate individual muscle contributions to propulsion, knee flexion in late stance and leg swing initiation in individuals post-stroke and age-similar participants without neurological

injury. We identified a number of deficits contributing to low or asymmetric paretic propulsion, including low plantarflexor contributions to propulsion and/or excess vasti contributions to braking. We also found that some participants had higher paretic than nonparetic propulsion, but often that propulsion was accomplished via compensation from the hamstrings and resulted in slow walking speeds. Lower knee flexion during swing was predicted by greater vasti contributions to braking in pre-swing. Despite the potential of the rectus femoris to flex the knee in pre-swing (Reinbolt et al., 2008), rectus femoris contributions to knee flexion in pre-swing were low compared to other muscles. However, the rectus femoris absorbed more power and the iliopsoas contributed less power from the paretic leg in pre-swing in a number of individuals with low knee flexion. These results highlight the heterogeneity of the post-stroke population and the need to identify the underlying causes of propulsion and knee flexion deficits in each individual in order to improve rehabilitation outcomes.

Together, these projects investigated specific balance recovery responses and muscle contributions to biomechanical subtasks during walking. The ultimate goal of this research is to provide a foundation for developing more evidence based and individualized therapies that target specific biomechanical deficits in walking and balance control.

Chapter 6: Future Work

This research has provided insight into the joint and muscle-level responses of young, healthy adults to foot placement perturbations. It has also provided insights into muscle contributions to propulsion, knee flexion and leg swing initiation in adults without neurological injury and adults post-stroke. However, there is more work that needs to be done to further our understanding of walking and dynamic balance and to use these results to develop evidence-based interventions. Thus, these results provide a foundation for future studies to expand upon this work.

While Chapters 2 and 3 laid the groundwork for understanding how healthy individuals control foot placement and respond to foot placement perturbations, it is still unknown how individuals post-stroke would respond to these perturbations. Young healthy participants responded to medial and lateral foot placement perturbations using a combination of lateral hip, lateral ankle, ankle plantarflexion and stepping strategies to restore their balance. However, individuals post-stroke have reduced muscle coordination complexity (e.g. Clark et al., 2010) that may impair their ability to use these integrative balance recovery strategies. Future work should investigate the responses of individuals post-stroke and older adults to these perturbations in comparison to young, healthy participants. The results of Chapter 3, which showed that trunk muscles were primary contributors to changes in foot placement control after perturbations, also encourages future study of how trunk and leg muscle coordinate to control dynamic balance, and how that coordination is disrupted post-stroke.

Chapter 3 also highlighted that even among young healthy participants, there was significant variability in the balance recovery responses to foot placement perturbations. The variability and small number of steps analyzed prevented us from reaching conclusions regarding how muscles work to modulate mediolateral ground reaction forces, torso power

and angular momentum. Incorporating more gait cycles into future simulation analyses of this data will allow us to investigate subtypes of responses and work towards determining why some individuals use specific balance recovery strategies. Currently, simulating multiple gait cycles is time intensive and requires manually adjusting parameters to improve residual reduction results. While pipelines were created to fully automate some tasks, such as scaling, inverse kinematics, static optimization and induced acceleration and power analyses, the step of reducing residuals still required manual adjustment of parameters. Future work will further automate the process of reducing residual forces and moments and validating results so that a larger number of gait cycles can be used in analyses.

Chapter 4 highlighted that a number of different factors contribute to propulsion deficits and stiff knee gait in individuals post-stroke. The heterogeneity of the post-stroke population is a factor in the inconclusive results of many clinical trials; a recent review highlighted the need for more standard, quantitative kinetic and kinematic measurements, more stratification of responders and non-responders, and better conceptualization of interventions and outcome measures (Kwakkel et al., 2017). Thus, future work should continue to identify underlying causes of walking performance deficits to provide evidence for interventions targeting specific, individual impairments. Future work should also determine predictors of intervention outcomes, such as initial muscle coordination complexity and propulsion deficits. Chapter 4 also used simulation methods to identify the underlying causes of body propulsion and leg swing deficits, but these time intensive simulation methods currently have minimal potential for clinical relevance. Thus, future work will seek to improve clinical measurements for identifying these underlying deficits and discerning between improved paretic function and compensatory changes.

Finally, the analysis in Chapter 4 was limited in that it only focused on muscle contributions to propulsion, while leg extension is also important to generating propulsion (Awad et al., 2020; Peterson, Cheng, et al., 2010; Roelker, Bowden, et al., 2019). Thus, future work should also investigate the underlying causes of leg extension deficits. Moreover, Chapter 4 focused primarily on the pre-swing phase of walking so future work should extend the analyses of stiff knee gait to the swing phase and investigate other biomechanical measures to quantify stiff knee gait.

Appendices

APPENDIX A: SUPPLEMENTAL MATERIAL FOR CHAPTER 2

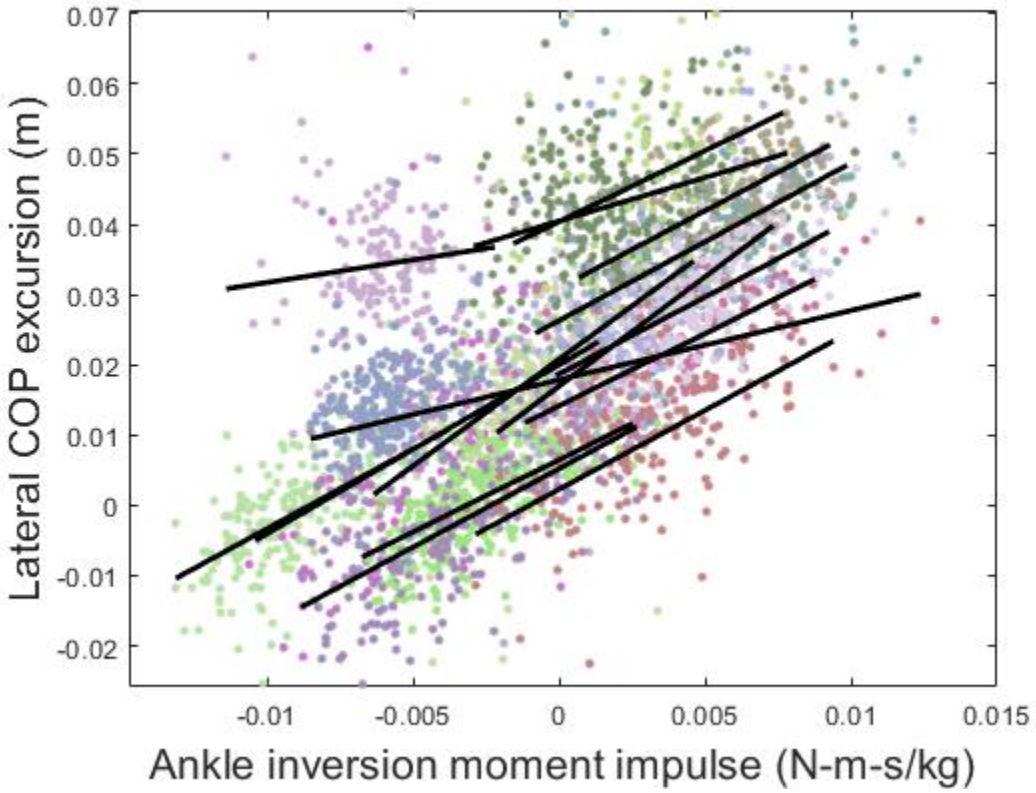

Figure A1: Results of the linear mixed effects model analyzing the correlation between ankle inversion impulse and lateral COP excursion. Each dot represents one perturbed step and each color shows a different subject. The black lines show individual regression models for each subject.

Table A1: Results of dependent measures ± 1SD. '*' indicates a significant difference between medially or laterally perturbed steps and unperturbed steps.

		Unperturbed	Medially Perturbed		Laterally perturbed	
H_R (Dimensionless)	stance	0.048 ± 0.009	0.074 ± 0.013	*p<.001	0.045 ± 0.010	*p<.001
COP Excursion (cm)	stance	0.65 ± 0.52	2.58 ± 1.10	*p<.001	-1.62 ± 0.77	*p<.001
ML GRF impulse (% BW-s)	stance	3.30 ± 0.68	2.71 ± 0.68	*p<.001	4.21 ± 0.83	*p<.001
Vertical GRF impulse (% BW-s)	stance	50.75 ± 1.89	51.32 ± 3.72		50.52 ± 2.94	
Ankle inversion moment impulse ($N\text{-}m\text{-}s/kg \times 10^2$)	stance	-0.08 ± 5.71	3.80 ± 6.13	*p<.001	-3.30 ± 4.81	*p<.001
	phase 1	0.86 ± 0.93	1.46 ± 0.91	*p<.001	0.48 ± 0.99	*p<.001
	phase 2	0.94 ± 2.14	1.78 ± 2.31	*p<.001	-0.34 ± 1.92	*p<.001
	phase 3	-0.89 ± 2.20	0.90 ± 2.57	*p<.001	-2.11 ± 1.66	*p<.001
	phase 4	-1.01 ± 1.00	-0.25 ± 1.04	*p<.001	-1.47 ± 0.95	*p<.001
Hip abduction moment impulse ($N\text{-}m\text{-}s/kg \times 10^2$)	stance	43.03 ± 10.69	36.51 ± 8.71	*p<.001	39.25 ± 9.00	*p<.001
	phase 1	5.15 ± 1.84	5.13 ± 2.27		3.95 ± 1.59	*p<.001
	phase 2	18.05 ± 4.40	15.13 ± 3.34	*p<.001	17.26 ± 3.77	*p<.05
	phase 3	15.91 ± 3.68	13.85 ± 3.07	*p<.001	14.10 ± 3.02	*p<.001
	phase 4	6.11 ± 2.38	4.38 ± 2.91	*p<.001	5.32 ± 2.01	*p<.01
Ankle plantarflexion moment impulse ($N\text{-}m\text{-}s/kg \times 10^2$)	stance	43.92 ± 4.99	40.41 ± 4.47	*p<.001	42.27 ± 5.72	*p<0.05
	phase 1	-1.54 ± 0.61	-0.81 ± 3.32		-0.98 ± 0.99	*p=.001
	phase 2	10.25 ± 2.46	9.69 ± 2.15		11.64 ± 1.92	*p<.001
	phase 3	23.58 ± 2.92	22.37 ± 4.80		21.20 ± 1.66	*p<.001
	phase 4	14.08 ± 3.02	11.31 ± 2.82	*p<.001	12.92 ± 0.95	*p<.05

APPENDIX B: SUPPLEMENTAL MATERIAL FOR CHAPTER 3

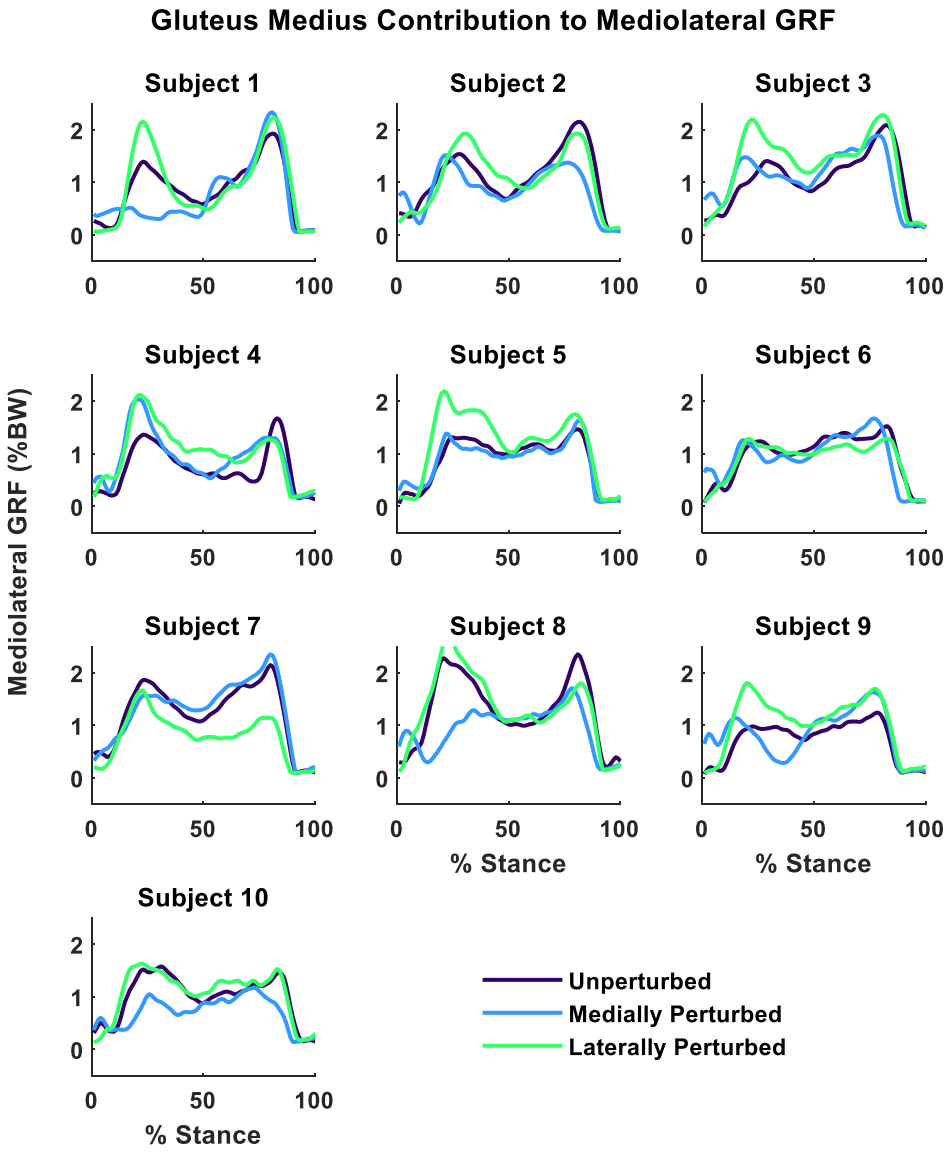

Figure B1: Contributions from the gluteus medius muscle group to mediolateral ground reaction forces during the stance phase immediately following the foot placement perturbations for each participant.

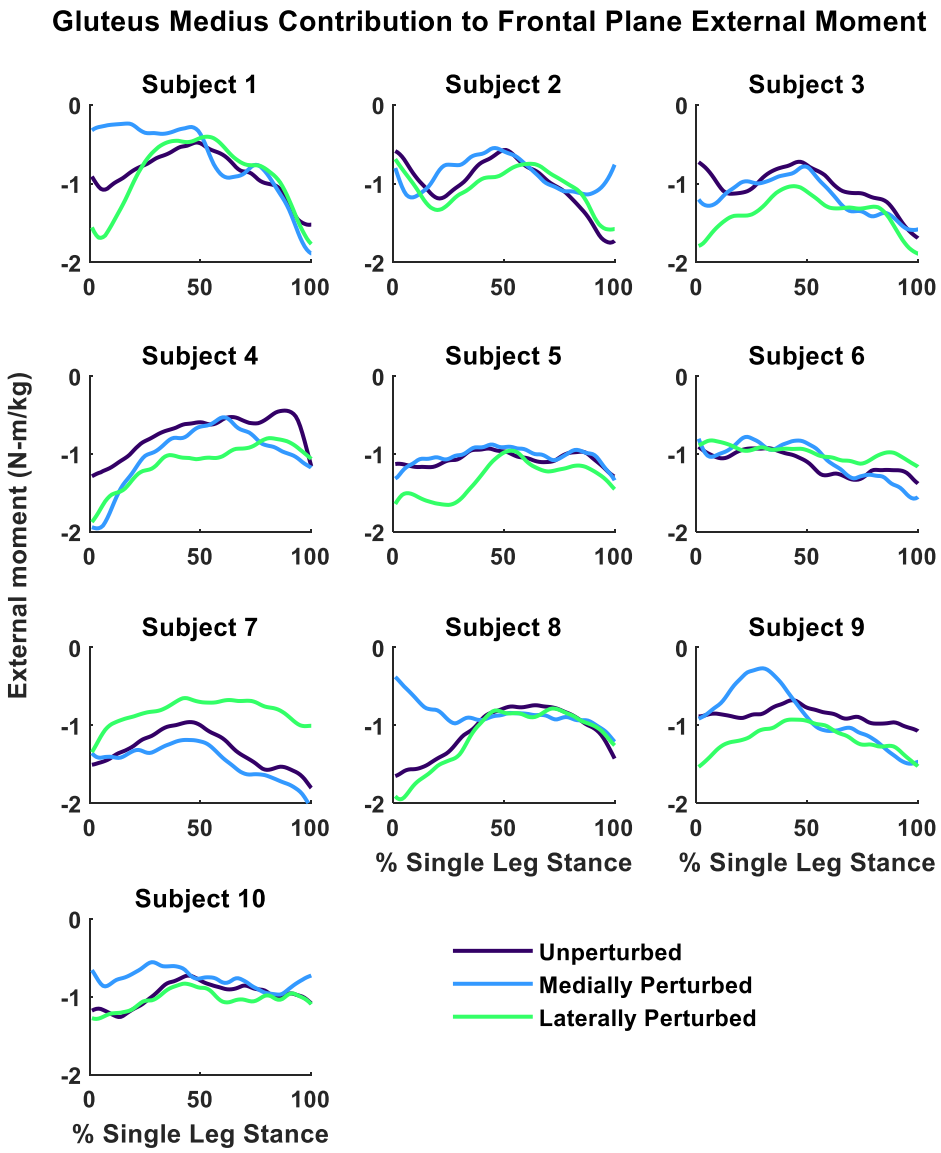

Figure B2: Contributions from the gluteus medius muscle group to the frontal plane external moment during the single leg stance phase immediately following the foot placement perturbations for each participant.

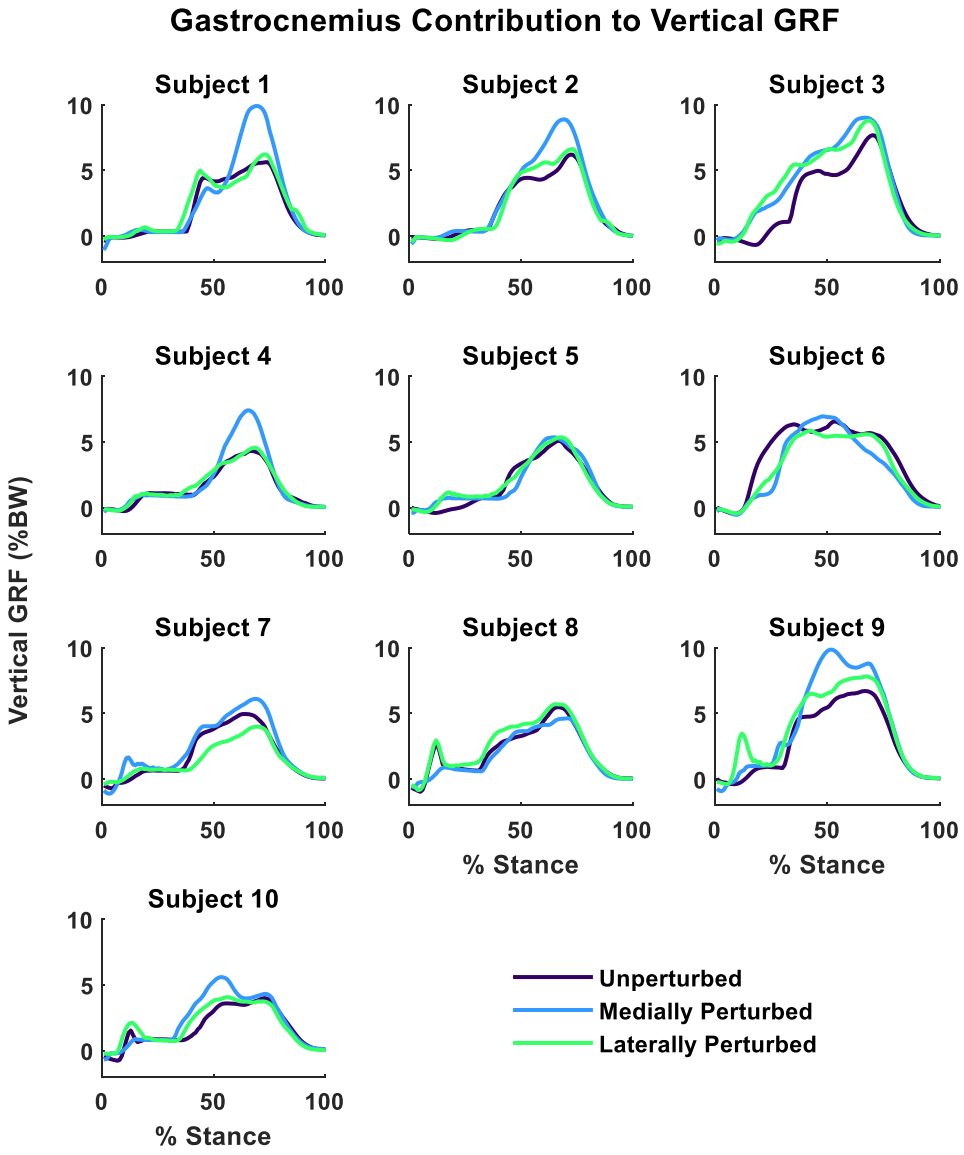

Figure B3: Contributions from the gastrocnemius muscle group to vertical ground reaction forces during the stance phase immediately following the foot placement perturbations for each participant.

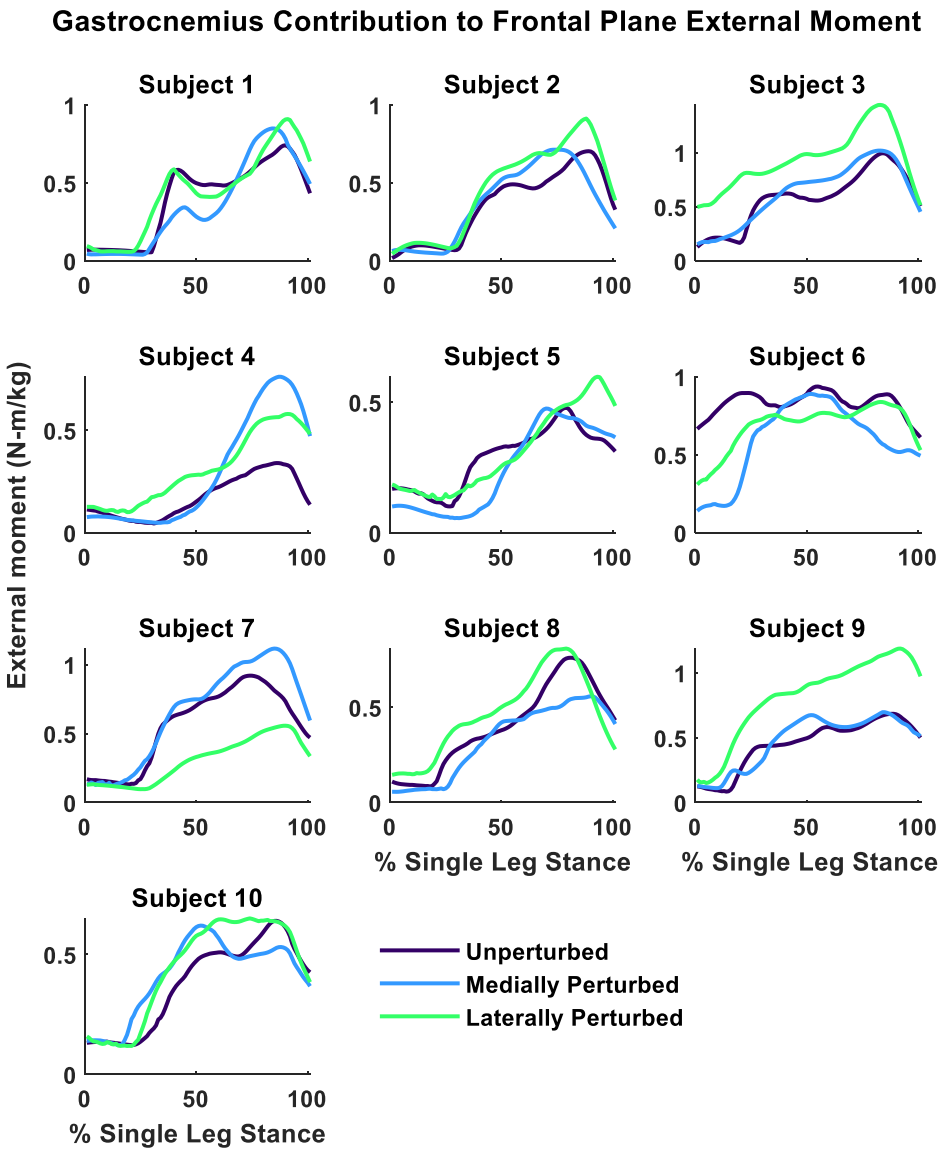

Figure B4: Contributions from the gluteus medius muscle group to the frontal plane external moment during the single leg stance phase immediately following the foot placement perturbations for each participant.

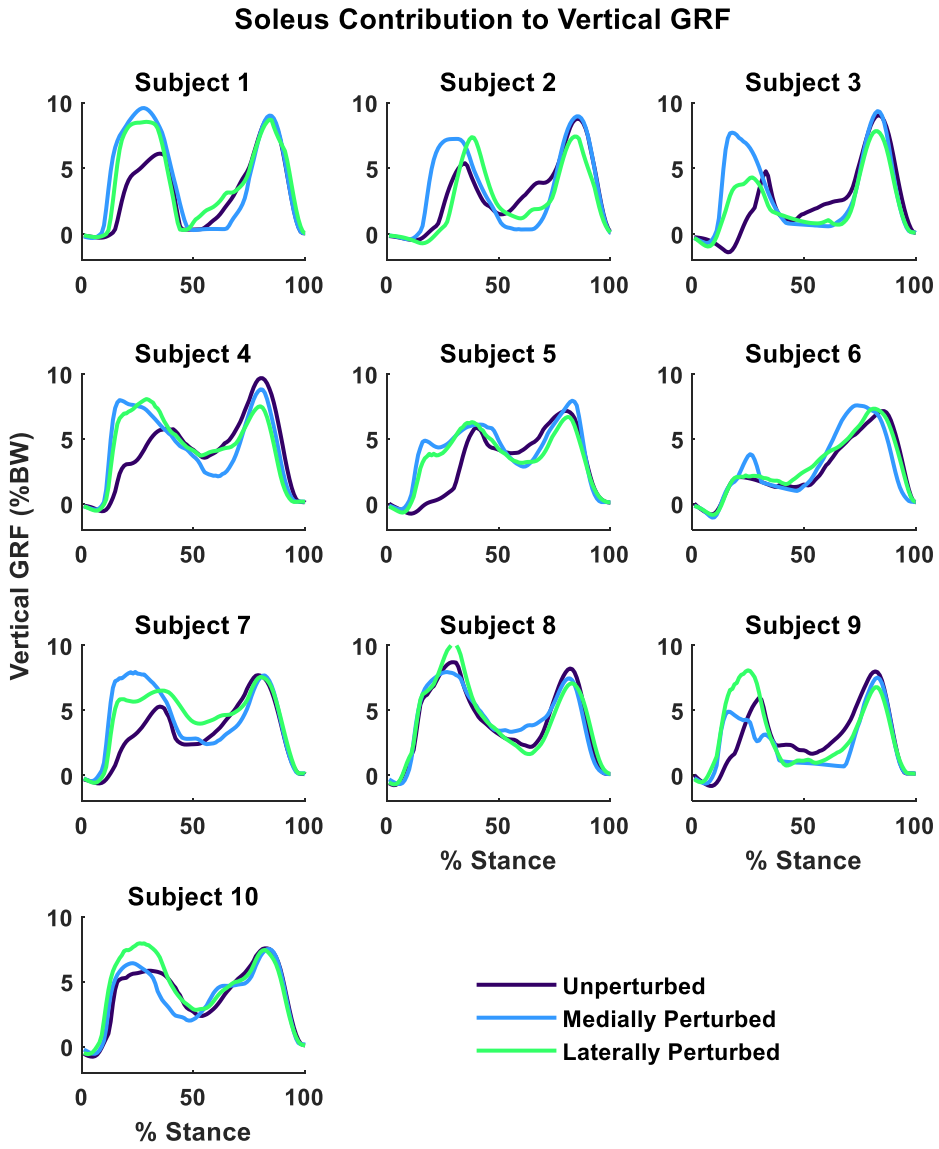

Figure B5: Contributions from the soleus muscle to the vertical ground reaction forces during the stance phase immediately following the foot placement perturbations for each participant.

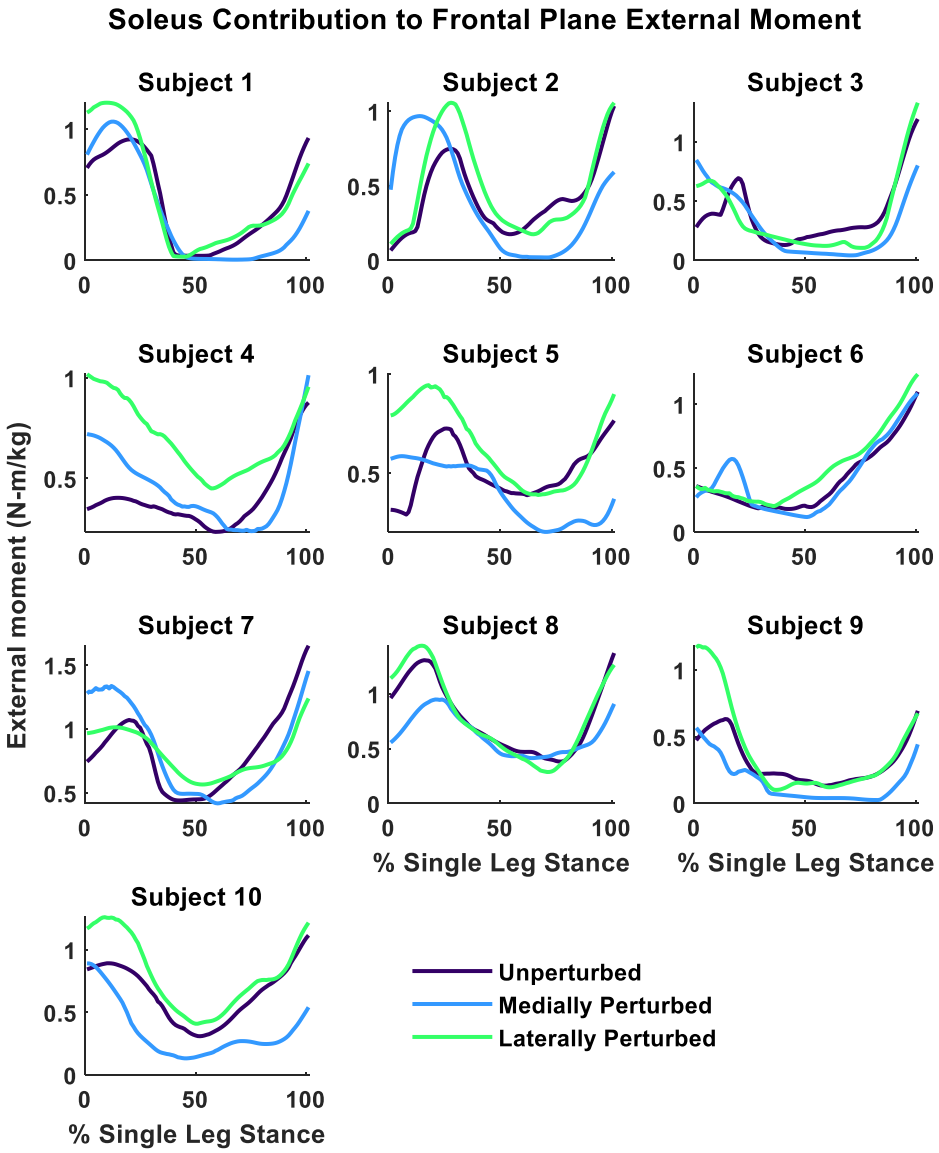

Figure B6: Contributions from the soleus muscle to the frontal plane external moment during the single leg stance phase immediately following the foot placement perturbations for each participant.

Table B1: Muscle mechanical work done on medial and lateral foot placement relative to the pelvis during swing in J/(kg*m/s) x 10^{-3}. 'Contra' indicates the muscle is on the stance leg side. Significant differences between perturbed and unperturbed steps are bolded.

		First recovery step				Second recovery Step			
Muscle	**Unperturbed**	**Medially Perturbed**	***p-value***	**Laterally Perturbed**	***p-value***	**Medially Perturbed**	***p-value***	**Laterally Perturbed**	***p-value***
Contra. GMED	-6.5 ± 2.3	-6.6 ± 3.9	0.955	-10.4 ± 5.5	**0.018**	-5.7 ± 2.8	0.280	-7.9 ± 3.2	0.075
Contra. ERSPIN	-3.0 ± 1.2	-1.6 ± 1.1	**0.003**	-3.0 ± 1.4	0.942	-2.1 ± 0.8	**0.009**	-3.5 ± 1.7	0.253
ADD	-2.2 ± 0.8	-3.1 ± 1.6	0.120	-3.0 ± 0.9	**0.025**	-2.1 ± 1.0	0.689	-3.4 ± 1.2	**0.000**
HAM	-1.5 ± 0.6	-1.1 ± 0.7	0.105	-2.6 ± 1.5	0.033	-1.6 ± 0.7	0.557	-2.5 ± 1.3	**0.017**
IL	-1.4 ± 0.5	-1.8 ± 1.0	0.176	-2.7 ± 0.2	**0.000**	-1.0 ± 0.5	0.034	-1.6 ± 1.2	0.609
Contra. INTOB	-1.2 ± 0.6	-1.0 ± 0.5	0.458	-1.8 ± 1.4	0.105	-0.9 ± 0.3	0.080	-1.2 ± 0.5	0.730
SOL	-0.8 ± 0.6	-1.5 ± 1.2	0.129	-0.3 ± 0.6	0.079	-0.8 ± 0.9	0.848	-1.9 ± 1.7	**0.018**
RF	0.7 ± 0.3	0.6 ± 0.5	0.738	1.0 ± 0.6	0.164	0.6 ± 0.4	0.434	1.1 ± 0.4	**0.000**
Contra. GAS	1.0 ± 1.1	1.3 ± 1.5	0.430	2.1 ± 2.0	**0.011**	1.5 ± 1.3	0.321	0.5 ± 1.2	0.382
Contra. IL	1.6 ± 1.2	-0.4 ± 1.1	**0.000**	2.6 ± 2.8	0.125	1.3 ± 0.9	0.586	1.7 ± 1.4	0.669
TA	1.2 ± 0.9	2.2 ± 1.5	0.119	0.4 ± 0.9	0.084	1.2 ± 1.3	0.993	2.7 ± 2.0	**0.009**
INTOB	2.5 ± 0.9	2.9 ± 1.4	0.355	3.9 ± 0.7	**0.001**	1.9 ± 1.3	0.056	5.1 ± 3.7	**0.025**
ERSPIN	4.5 ± 1.2	2.6 ± 1.7	**0.010**	7.0 ± 2.3	**0.001**	2.8 ± 1.0	**0.000**	5.6 ± 2.3	0.028
GMED	3.3 ± 1.4	4.1 ± 1.7	0.288	6.4 ± 2.5	**0.004**	3.0 ± 1.7	0.156	7.4 ± 2.9	**0.000**

APPENDIX C: SUPPLEMENTAL MATERIAL FOR CHAPTER 4

The following figures show the different ways the muscles contribute to braking and propulsion in individuals post-stroke. We identified a number of sources of propulsion deficits, including:

1. **High propulsion, high braking:** Sufficient paretic plantarflexor output but excess braking from the vasti or rectus femoris throughout mid and late stance.
2. **Plantarflexor braking:** The paretic plantarflexors contribute primarily to braking instead of propulsion.
3. **Compensations:** The primary paretic propulsion generators are not the plantarflexors.
4. **Low plantarflexor output:** Plantarflexor contributions to propulsion are low, but coordination and timing are not significantly impaired.

Individuals post-stroke also spend less time in paretic stance than nonparetic stance (Olney & Richards, 1996), which reduced the amount of time available for paretic muscles to generate propulsion. Below, many individuals demonstrate atypical muscle coordination on the nonparetic leg which may be due to increased co-activation of nonparetic muscles as well as paretic muscles (e.g. Clark et al., 2010), or due to nonparetic compensation or atypical gait patterns.

See Figure 4.2 for detailed information about how to interpret the following figures.

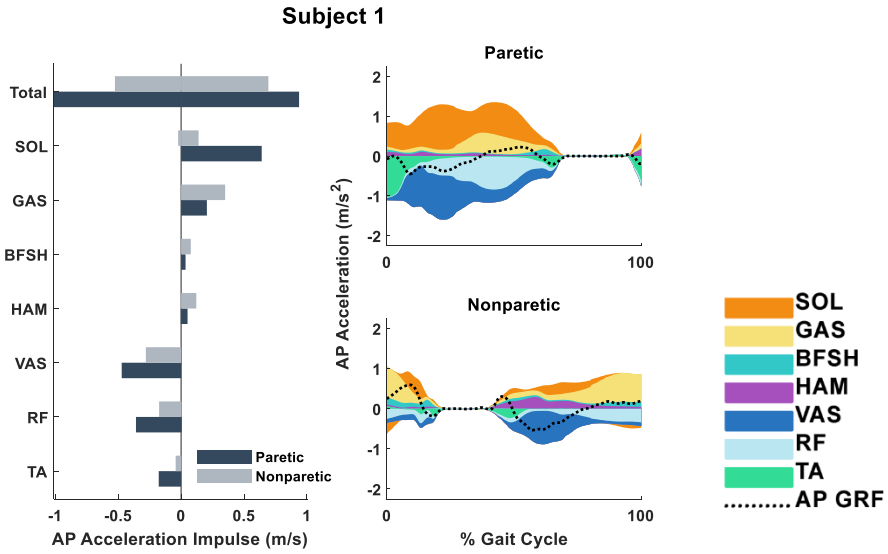

Figure C1: Participant 1 produces both high paretic propulsion and excess paretic braking, as the paretic plantarflexors produce more propulsion than the nonparetic plantarflexors, but the vasti and rectus femoris produce braking throughout stance instead of only during early stance.

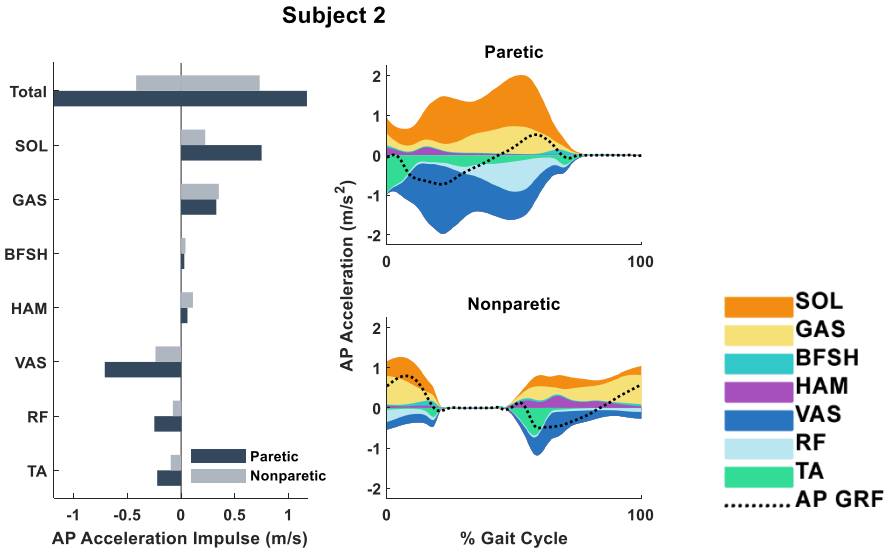

Figure C2: Similar to Participant 1, Participant 2 also generates high paretic propulsion but also high paretic braking due to excess vasti and rectus femoris contributions to braking throughout stance.

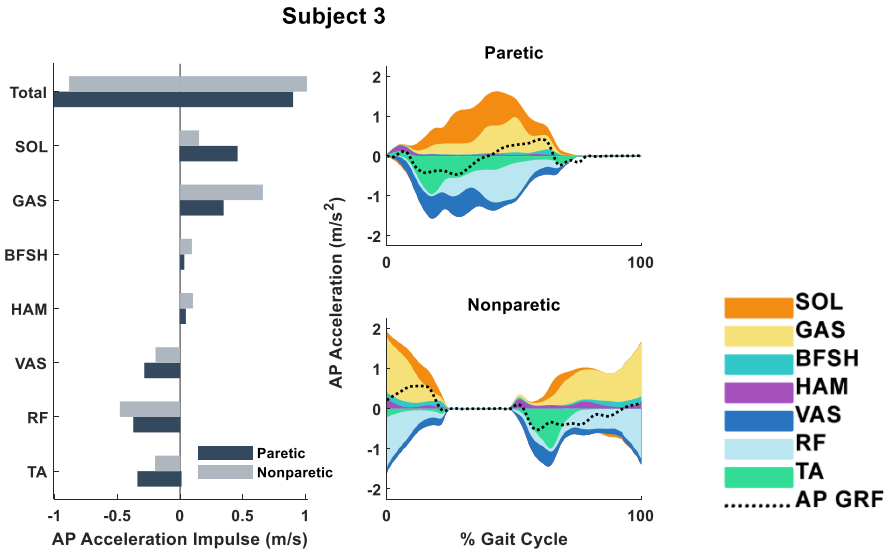

Figure C3: Participant 3 generates excess braking from the rectus femoris throughout stance on both the paretic and nonparetic legs.

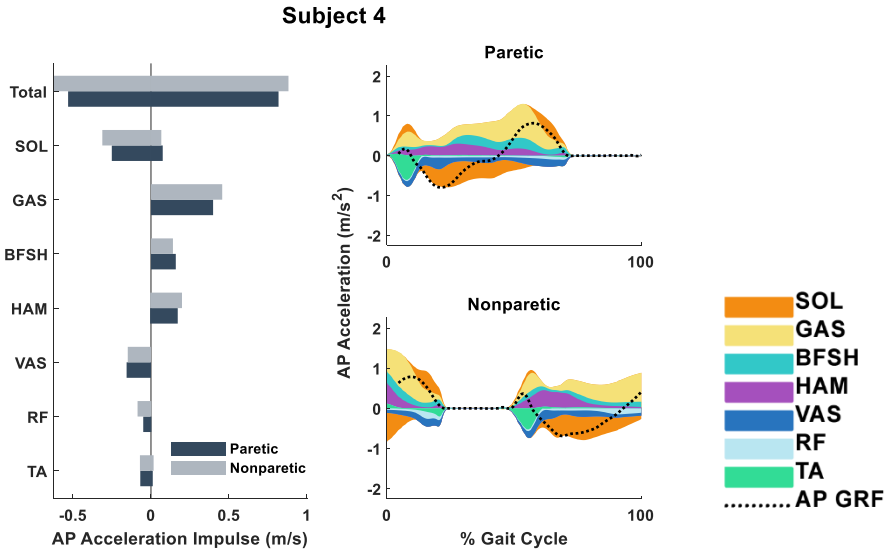

Figure C4: Participant 4 has symmetric muscle activity on the paretic and nonparetic sides, but interestingly, the soleus is a primary contributor to braking and contributes very little to propulsion on both legs.

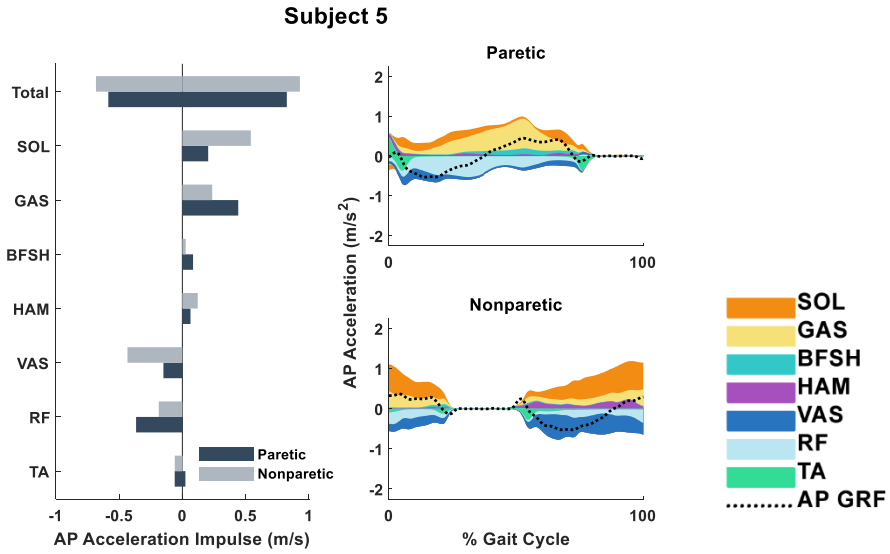

Figure C5: Participant 5 has low plantarflexor contributions to propulsion.

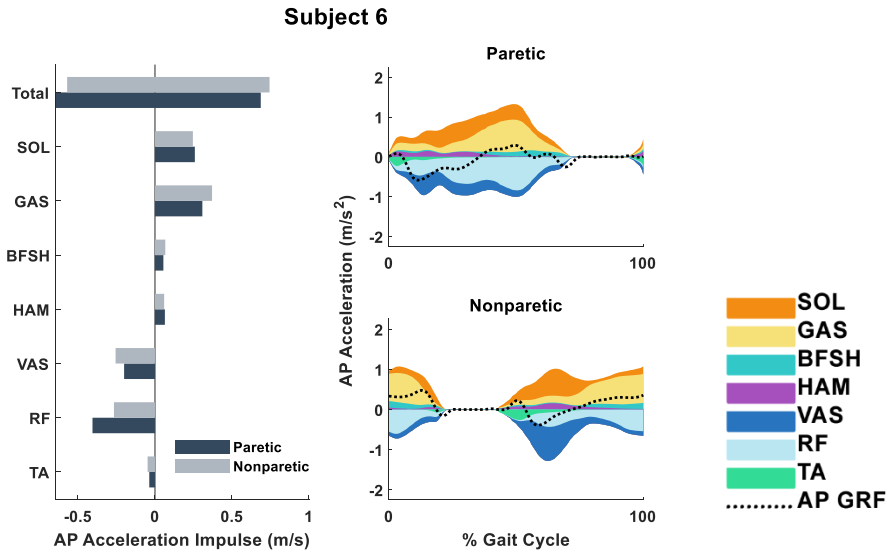

Figure C6: Participant 6 generates excess braking from the rectus femoris on the paretic leg.

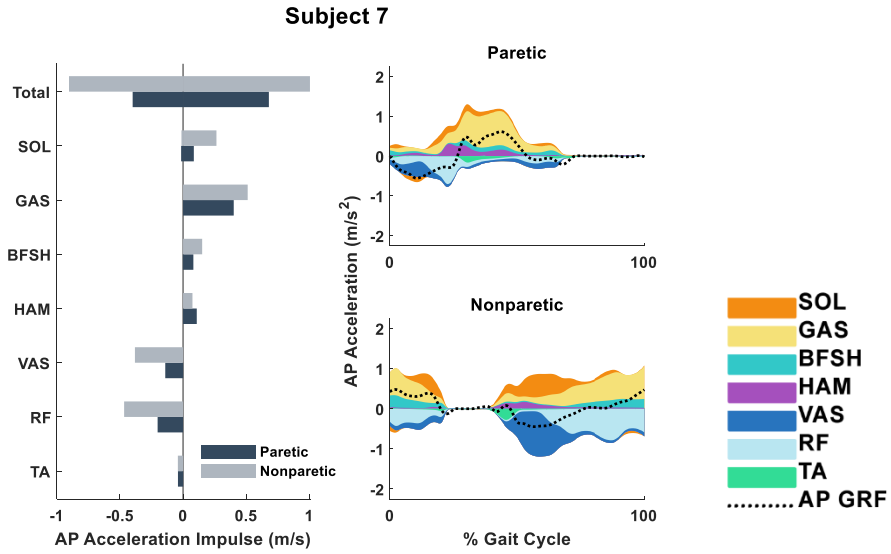

Figure C7: Participant 7 has low plantarflexor contributions to propulsion on the paretic side, especially from the soleus.

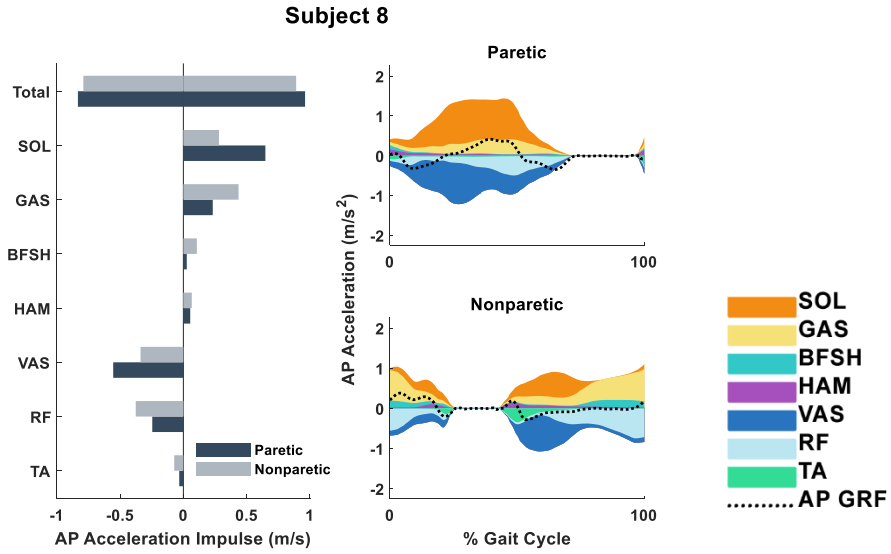

Figure C8: Participant 8 generates braking from the vasti and rectus femoris throughout stance.

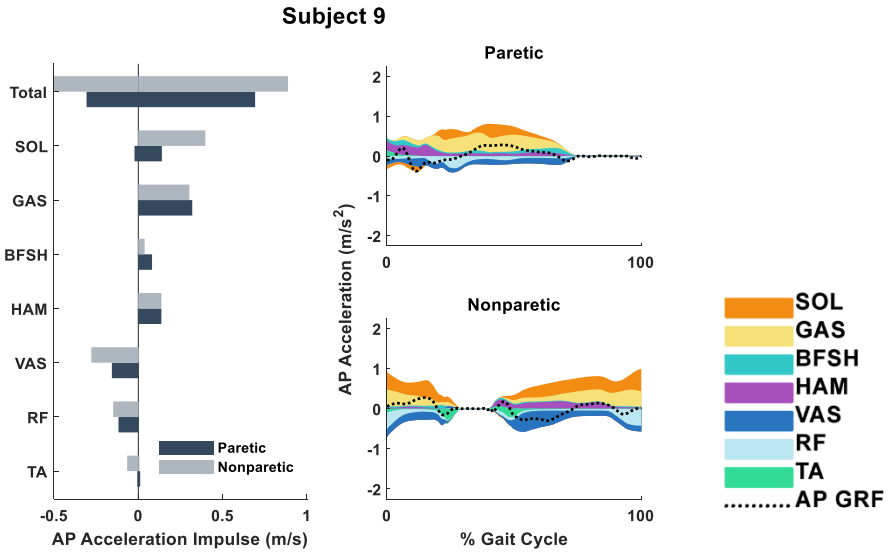

Figure C9: Participant 9 has very low plantarflexor contributions to propulsion on the paretic side.

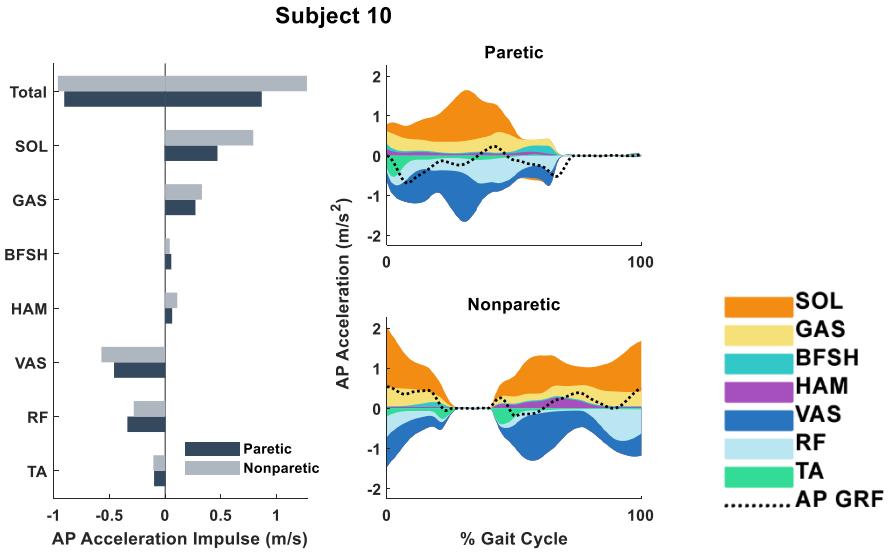

Figure C10: Participant 10 produces less paretic propulsion from the soleus and produces braking throughout stance on both legs.

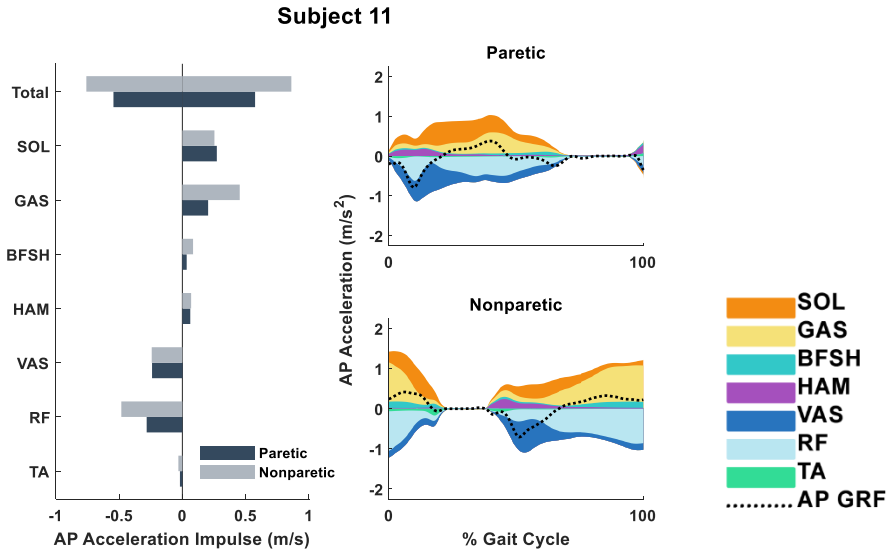

Figure C11: Participant 11 generates lower propulsion on the paretic side due to lower contributions from the gastrocnemius.

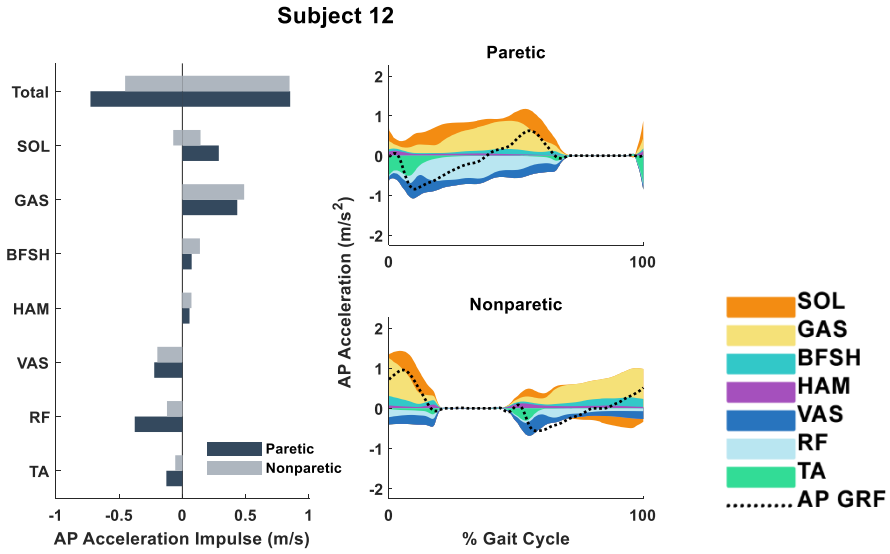

Figure C12: Participant 12 generates excess braking from the paretic rectus femoris, but generates more propulsion from the paretic soleus than nonparetic soleus.

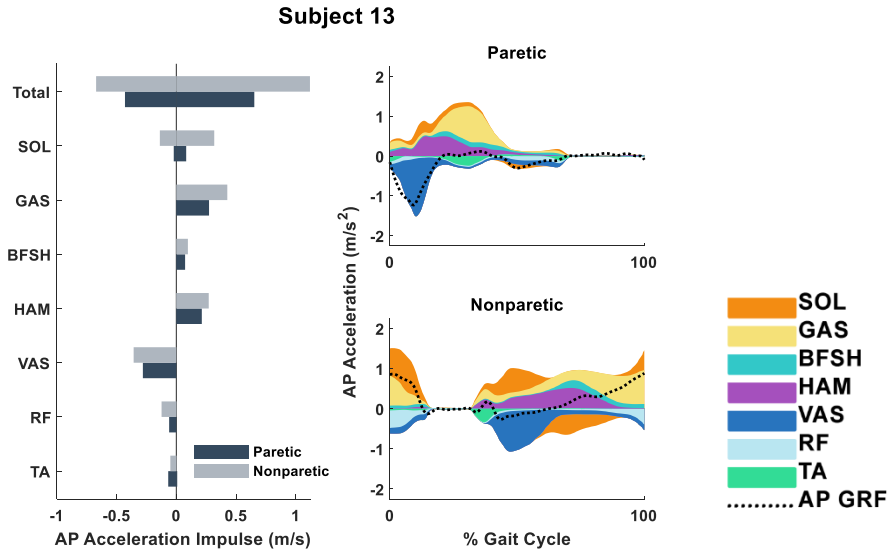

Figure C13: Participant 13 generates low propulsion from the paretic plantarflexors but does not produce excess paretic braking through mid and late stance like many of the participants.

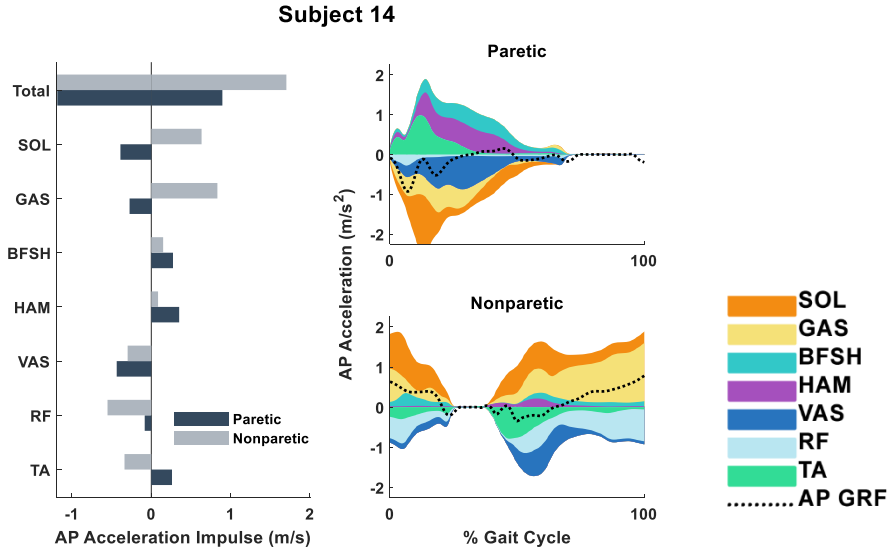

Figure C14: Participant 14 uses compensatory mechanisms to achieve almost all paretic propulsion from the hamstrings and tibialis anterior rather than from the plantarflexors, which primarily produce braking.

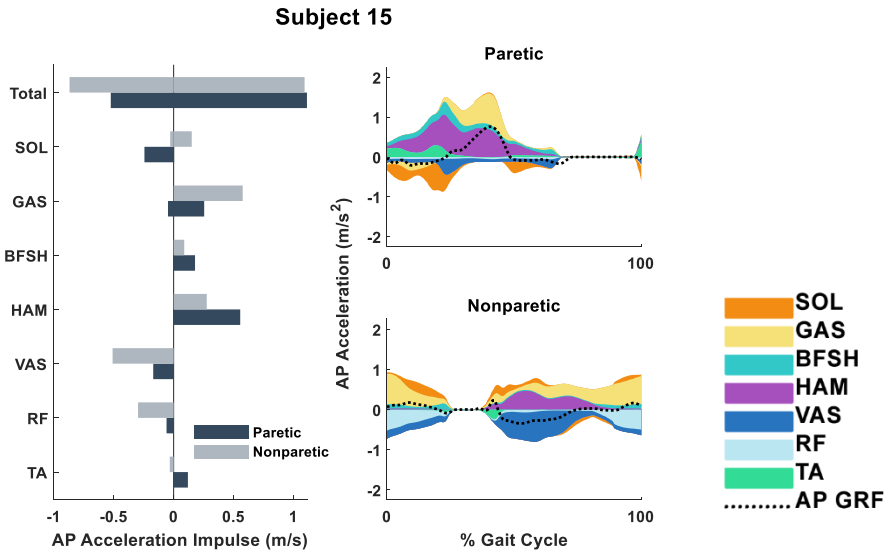

Figure C15: Participant 15 uses compensation from the hamstrings to generate paretic propulsion while the paretic soleus produces primarily braking.

Table C1: Propulsion and braking symmetry, muscle contributions to propulsion and braking, and knee kinematics for all participants in Chapter 4. Values for participants in the low knee flexion group are bolded.

			Paretic AP COM acceleration impulse (m/s)			Nonparetic AP COM Acceleration impulse (m/s)			Knee flexion velocity at toe-off (deg/s)	Peak knee flexion (deg)	ROM difference (deg)
Participant	**PP**	**PB**	**SOL**	**GAS**	**VAS**	**SOL**	**GAS**	**VAS**			
1	**0.24**	**0.51**	**0.640**	**0.205**	**-0.471**	**0.116**	**0.350**	**-0.279**	**122**	**49**	**35**
2	0.31	0.65	0.749	0.328	-0.710	0.225	0.351	-0.238	178	69	12
3	0.42	0.49	0.454	0.348	-0.285	0.142	0.661	-0.196	252	52	12
4	0.52	0.47	-0.176	0.398	-0.156	-0.246	0.457	-0.149	247	71	9
5	0.57	0.49	0.200	0.440	-0.145	0.540	0.235	-0.434	200	61	-9
6	**0.24**	**0.72**	**0.259**	**0.308**	**-0.200**	**0.246**	**0.371**	**-0.255**	**93**	**48**	**22**
7	0.50	0.56	0.070	0.401	-0.140	0.252	0.512	-0.380	161	56	1
8	**0.55**	**0.64**	**0.648**	**0.232**	**-0.554**	**0.280**	**0.436**	**-0.338**	**-8**	**36**	**42**
9	**0.55**	**0.41**	**0.117**	**0.319**	**-0.156**	**0.395**	**0.302**	**-0.274**	**85**	**31**	**31**
10	**0.08**	**0.90**	**0.464**	**0.272**	**-0.459**	**0.791**	**0.330**	**-0.573**	**86**	**33**	**21**
11	**0.31**	**0.54**	**0.272**	**0.203**	**-0.237**	**0.253**	**0.453**	**-0.241**	**116**	**48**	**20**
12	0.38	0.62	0.288	0.434	-0.222	0.071	0.488	-0.198	185	57	10
13	**0.08**	**0.84**	**0.061**	**0.274**	**-0.279**	**0.180**	**0.427**	**-0.357**	**120**	**30**	**24**
14	**0.06**	**0.74**	**-0.388**	**-0.266**	**-0.435**	**0.634**	**0.832**	**-0.297**	**158**	**37**	**27**
15	**0.71**	**0.41**	**-0.239**	**0.208**	**-0.170**	**0.122**	**0.576**	**-0.509**	**34**	**30**	**21**
Average	0.37	0.60	0.228	0.274	-0.308	0.267	0.452	-0.315	135	47	18
SD	0.19	0.15	0.322	0.164	0.170	0.241	0.146	0.115	70	13	13
C1	0.55	0.56	-0.077	0.284	-0.065	0.005	0.347	-0.312	253	56	5
C2	0.53	0.52	0.079	0.300	-0.260	0.262	0.376	-0.293	338	68	3
C3	0.43	0.55	-0.067	0.251	-0.220	0.417	0.208	-0.462	363	77	-1
C4	0.72	0.33	0.118	0.322	-0.225	0.158	0.483	-0.337	259	72	-9
C5	0.56	0.47	0.221	0.381	-0.295	0.279	0.384	-0.379	335	72	-7
Average	0.56	0.48	0.055	0.307	-0.213	0.224	0.360	-0.357	310	69	-2
SD	0.09	0.08	0.114	0.043	0.079	0.137	0.088	0.060	45	7	6

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Vita

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