Muscle contributions to support during gait in an individual with post-stroke hemiparesis

J.S. Higginson[a,b,c,*], F.E. Zajac[a,b,d], R.R. Neptune[e], S.A. Kautz[f,g,h], S.L. Delp[a,b,d,i]

[a]*VA Palo Alto Rehab. R&D Ctr., Palo Alto, CA, USA*
[b]*Dept. of Mech. Eng., Stanford University, Stanford, CA, USA*
[c]*Dept. of Mech. Eng., University of Delaware, Newark, DE, USA*
[d]*Dept. of Orthopedic Surgery, Stanford University, Stanford, CA, USA*
[e]*Dept. of Mech. Eng., University of Texas, Austin, TX, USA*
[f]*Brain Rehab. Research Ctr., Malcom Randall VA Medical Ctr., Gainesville, FL, USA*
[g]*Dept. of Physical Therapy, University of Florida, Gainesville, FL, USA*
[h]*Brooks Ctr. Rehab. Studies., University of Florida, Gainesville, FL, USA*
[i]*Dept. of Bioengineering, Stanford University, Stanford, CA, USA*

Abstract

Walking requires coordination of muscles to support the body during single stance. Impaired ability to coordinate muscles following stroke frequently compromises walking performance and results in extremely low walking speeds. Slow gait in post-stroke hemiparesis is further complicated by asymmetries in lower limb muscle excitations. The objectives of the current study were: (1) to compare the muscle coordination patterns of an individual with flexed stance limb posture secondary to post-stroke hemiparesis with that of healthy adults walking very slowly, and (2) to identify how paretic and non-paretic muscles provide support of the body center of mass in this individual. Simulations were generated based on the kinematics and kinetics of a stroke survivor walking at his self-selected speed (0.3 m/s) and of three speed-matched, healthy older individuals. For each simulation, muscle forces were perturbed to determine the muscles contributing most to body weight support (i.e., height of the center of mass during midstance). Differences in muscle excitations and midstance body configuration caused paretic and non-paretic ankle plantarflexors to contribute less to midstance support than in healthy slow gait. Excitation of paretic ankle dorsiflexors and knee flexors during stance opposed support and necessitated compensation by knee and hip extensors. During gait for an individual with post-stroke hemiparesis, adequate body weight support is provided via reorganized muscle coordination patterns of the paretic and non-paretic lower limbs relative to healthy slow gait.
Published by Elsevier Ltd.

1. Introduction

Stroke is the leading cause of long-term adult disability in the US (NINDS, 2004). Because stroke impairs muscle coordination, survivors often have difficulty walking independently, which may be associated with limited ability to return to work, participate in the community, or perform other activities of daily living. Body weight supported treadmill training and functional electrical stimulation have demonstrated promising results for gait rehabilitation, but evidence-based practice is limited because the optimal timing, intensity and patient characteristics suitable for a given therapeutic protocol are unknown (Barbeau and Fung, 2001; Richards et al., 1999). An improved

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*Corresponding author. University of Delaware, Department of Mechanical Engineering, 201A Spencer Laboratory, Newark, DE 19716, USA. Tel.: +302 831 6622; fax: +302 831 3619.

E-mail address: higginson@me.udel.edu (J.S. Higginson).

understanding of the relationship between underlying coordination deficits and observed gait deviations should lead to better rehabilitation outcomes.

Gait deviations vary with stroke severity, location of infarct, time since stroke, type of rehabilitation received and other individual differences (Olney and Richards, 1996; Richards and Olney, 1996). However, several features of post-stroke hemiparetic gait are commonly observed, including slow speed, asymmetric joint kinematics and kinetics, prolonged stance duration on the non-paretic side, and increased double support time relative to neurologically healthy individuals walking at self-selected speeds (Olney and Richards, 1996). The impaired ability to coordinate muscle activity following stroke may limit gait speed, which ranges from about 0.1 m/s to greater than 1.0 m/s (Olney and Richards, 1996; Turnbull et al., 1995). Comparison of neurologically healthy and post-stroke gait at matched speeds has been performed to discriminate gait deviations independent of speed (Wagenaar and Beek, 1992; Chen et al., 2005). Some characteristics of post-stroke hemiparetic gait, such as a single plateau for the vertical ground reaction force, may be consistent with slow gait (Morita et al., 1995; Andriacchi et al., 1977). By studying non-speed-related gait deviations, we hope to identify the primary impairments in post-stroke hemiparetic gait.

Mulroy et al. (2003) identified gait speed, knee flexion at the end of paretic midstance, and pre-swing knee flexion as key determinants in a classification scheme for gait patterns in chronic stroke. Of the four gait patterns identified, the chronic "flexed" group exhibited very slow gait speed (27% of healthy age-matched adults) and excessive paretic stance phase knee flexion and ankle dorsiflexion (approximately 18° and 16°, respectively). These individuals also had severe ankle plantarflexor and hip extensor weakness, and required prolonged quadriceps activity to support the body in midstance.

Abbreviated paretic single stance duration may reflect the impaired ability of muscles to provide support. Similarly, prolonged non-paretic stance duration may require the reorganization of muscle coordination patterns to promote stability. Impaired force generation by the ankle plantarflexors following stroke will necessitate a change in the coordination patterns of proximal and contralateral muscle groups to provide support of the center of mass (COM). In stroke survivors who exhibit "flexed" characteristics and increased co-contraction, it is unclear how the roles of individual muscles differ from those of neurologically healthy individuals walking slowly.

Simulation studies have quantified the effect of individual muscles on segmental power (Neptune et al., 2001), joint and segment accelerations (Zajac and Gordon, 1989; Neptune et al., 2001; Jonkers et al., 2003; Anderson et al., 2004; Higginson et al., in press-b;

Goldberg et al., 2004; Higginson, 2005) and ground reaction forces (Anderson and Pandy, 2003; Neptune et al., 2004). The ankle plantarflexor muscles have been shown to contribute significantly to support in mid- and late stance at self-selected walking speed (Neptune et al., 2001; Anderson and Pandy, 2003). At slower speeds, contributions of the ankle plantarflexors are supplemented by uniarticular quadriceps to counteract stance phase dorsiflexor activity, which opposes COM support (Higginson, 2005).

In the current study, the gait patterns of one individual with post-stroke hemiparesis who exhibited characteristics similar to subjects classified as "flexed" by Mulroy et al. (2003) and three speed-matched healthy older adults were simulated. The objectives were: (1) to compare the muscle coordination patterns of an individual with a flexed stance limb posture secondary to post-stroke hemiparesis with that of healthy adults walking very slowly, and (2) to identify how paretic and non-paretic muscles provide support of the body center of mass in this individual. In this case study, we developed the first muscle-actuated forward dynamic simulation of a full cycle of hemiparetic gait. Our long term goal is to relate altered muscle coordination patterns to specific types of abnormal hemiparetic gait deviations (e.g. "flexed"), which will provide a scientific rationale for the application of targeted therapeutic interventions.

2. Methods

2.1. Modeling and simulation framework

A two-dimensional musculoskeletal model of the pelvis and lower extremities based on the anthropometry of an adult male (75 kg; 178 cm) was developed using SIMM (Delp and Loan, 1995, 2000). Equations of motion for the rigid body dynamics were generated using SD/FAST (PTC). The model (Delp et al., 1990) was actuated by 15 Hill-type muscle-tendon units per leg: soleus (SOL), medial and lateral gastrocnemius (GAS), semitendinosus and biceps femoris long head (HAMS), biceps femoris short head (BFSH), tibialis anterior (TA), quadriceps (three vasti compartments, VAS), rectus femoris (RF), gluteus maximus (GMAX), adductor magnus (AM) and the uniarticular hip flexors (iliacus and psoas, IP). The input to the model was a muscle excitation pattern. Each muscle excitation was constrained to one burst of activity per gait cycle, except RF, where two bursts of activity were permitted, and SOL and GAS, where three levels of excitation were permitted within their bursts. To produce each simulation, we optimized 37 muscle excitation control variables for healthy slow gait (bilateral symmetry assumed) and 74 for hemiparetic gait (bilateral asymmetry

assumed), in addition to the 9 generalized kinematic velocities at foot strike needed to represent the initial state (Neptune et al., 2001).

2.2. Experimental data

All procedures were approved by the Stanford University Human Subjects Committee and informed consent was obtained from each subject. Three-dimensional kinematic data were collected on three healthy older adults (mean age 66 years, 62 kg, 164 cm) walking at an extremely slow speed (0.3 m/s) on a treadmill (Chen, 2003; Chen et al., 2005). Vertical ground reaction forces were estimated from Pedar insole pressure sensors (Novel Electronics, Inc.). Anterior/posterior ground reaction forces and EMG data were not available for these subjects.

Three-dimensional marker positions (Qualisys, Inc.), bilateral ground reaction forces (Advanced Mechanical Technology, Inc.; Bertec Corp.), and EMG data during three overground walking trials were collected for more than 30 adults with hemiparesis secondary to stroke as part of a separate study. The data from one subject (67 years, 78 kg, 178 cm, 28 months since stroke onset), who exhibited slow gait speed and minimal frontal plane excursion, was selected as a case study. Motion capture features in SIMM were used to scale and animate the marker data with a three-dimensional model of the musculoskeletal system. For application with the previously described two-dimensional model of the pelvis and lower extremities, the resultant joint kinematics were projected onto the sagittal plane. EMG data were recorded from 8 muscles in each leg (SOL, GAS, TA, vastus lateralis, HAMS, RF, and gluteus medius). Raw EMG data were rectified, low-pass filtered with a cut-off frequency of 50 Hz, normalized to peak magnitude, and averaged over three trials.

2.3. Optimization

A simulated annealing optimization algorithm was implemented to find the feasible set of muscle excitations and kinematic initial conditions for the simulations (i.e., the 46 independent controls for the healthy slow gait simulation; the 83 controls for the post-stroke hemiparetic gait simulation) (Neptune and Hull, 1998; Neptune et al., 2001). A parallel implementation of simulated annealing was used to reduce time to convergence (Higginson et al., in press-a). An objective function for the simulated annealing algorithm was selected to minimize the difference between simulated and experimental joint kinematics, vertical ground reaction force, and position of the COM. Two muscle-actuated forward dynamic simulations were produced which replicated the experimental healthy slow and post-stroke hemiparietic gait data.

2.4. Analysis

The feasible muscle excitations of the simulations were subjectively compared with EMG patterns in the literature and those recorded in this study to validate the simulation solutions for healthy slow and post-stroke hemiparetic gait, respectively. Timing and magnitude of the muscle excitations for the slow and post-stroke gait simulations were compared. In the simulations of slow and post-stroke gait, we perturbed individual muscle forces to determine how individual muscles contribute to body support given the current muscle excitation level and body configuration. Individual muscle forces were eliminated for 0.06 s at the beginning and midpoint of midstance with all other muscle forces maintained and the averaged effect on the height of the COM, and hip, knee and ankle extension was quantified.

3. Results

The experimental kinematics and vertical ground reaction force for slow gait in neurologically healthy individuals were generally reproduced within two standard deviations by the simulation, except for the stance knee, which was slightly more extended during mid- and late-stance to enable contralateral foot clearance due to the absence of pelvic obliquity and hip abduction in the two-dimensional simulation (Higginson, 2005). Stance duration was 75% of the gait cycle for slow gait (0.3 m/s), with midstance between 25% and 50%. Muscle excitations agreed with EMG data available in the literature (den Otter et al., 2004).

For the simulation of stroke gait, paretic and non-paretic vertical ground reaction forces and joint kinematics were tracked throughout the gait cycle (Fig. 1: thick black lines within or near $\pm$2SD window). A discrepancy in non-paretic knee angle was observed in mid- to late stance (see Discussion). Experimental spatio-temporal parameters were reproduced by the simulation with non-paretic stance lasting 72% of the gait cycle, and abbreviated stance phase for the paretic limb (64% of the gait cycle).

The feasible muscle excitation pattern in the simulation of post-stroke gait was similar to the EMG pattern. Notable exceptions were the non-paretic TA excitation and bilateral RF excitations during stance. In addition, non-paretic VAS, GAS and SOL and paretic GAS excitations terminated sooner in late stance than the EMG.

The feasible excitations of the non-paretic SOL, GAS, BFSH, and IP and the paretic GAS, VAS, and HAMS in the post-stroke gait simulation compared reasonably well with the predicted muscle excitations of the healthy slow gait simulation (Fig. 2). TA and VAS were excited less and HAMS longer in non-paretic stance compared
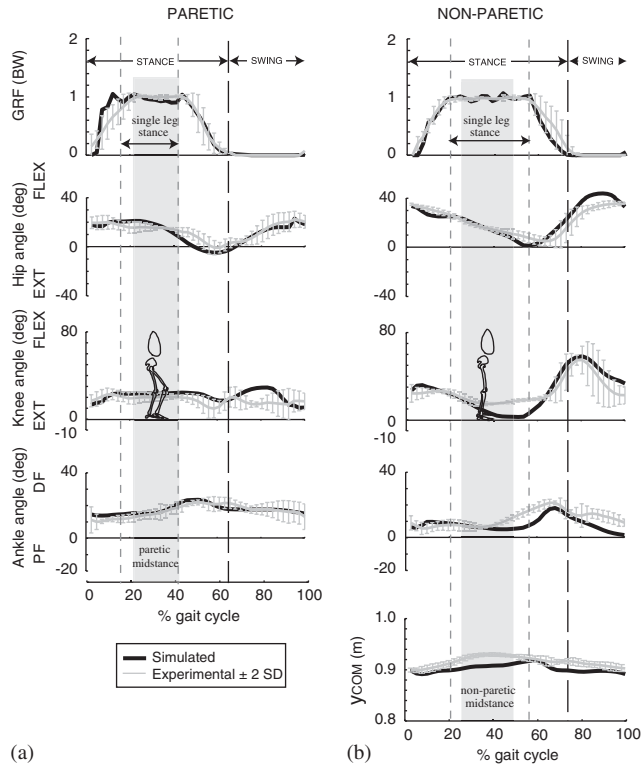
Fig. 1. Simulated (black line) and experimental data (gray line with error bars at ±2 SD) for post-stroke hemiparetic gait (0.3 m/s, $n = 1$). Note that kinematic and kinetic variability is low because the three trials are from a single hemiparetic subject. The vertical ground reaction force and hip, knee and ankle kinematics were reproduced reasonably well for the (a) paretic and (b) non-paretic limbs over the gait cycle, as well as the vertical position of the COM (y_{COM}). 0% of the gait cycle corresponds to initial contact for the paretic or non-paretic limb respectively (y_{COM} shown with respect to the non-paretic gait cycle). The shaded region and long dashed line indicate midstance and toe-off, respectively.

to slow gait. SOL was excited very early in paretic stance, TA more, BFSH longer, and IP not at all compared to healthy slow gait. GMAX in the non-paretic and paretic legs and in the slow gait simulation were excited in stance but at a low level. Post-stroke RF in both legs was excited more in stance and swing.

Non-paretic SOL, GAS and VAS contributed significantly to midstance COM support but their contributions were lower compared to healthy slow gait (Fig. 3: compare black and gray bars). Non-paretic GMAX and HAMS made minor contributions to support. Non-paretic TA opposed support much less than in slow gait.

Paretic SOL and GAS contributed even less to support (Fig. 3: white bars compared to black and gray bars). The VAS, RF and GMAX on the paretic leg contributed significantly more compared to the non-paretic leg or to their contributions in healthy slow gait. Paretic TA opposed support substantially like in slow gait, and paretic BFSH opposed COM support even

more, though no such contribution was seen in either the non-paretic leg or in slow gait.

4. Discussion

We have developed the first muscle-actuated forward dynamic simulation of a whole cycle of post-stroke hemiparetic gait. Despite observed differences in muscle excitations relative to slow gait of neurologically healthy older adults, the paretic and non-paretic ankle plantar-flexors and knee extensors still contributed to midstance support of the COM for this stroke survivor. However, paretic muscles exhibited increased co-contraction and required supplemental contributions to body weight support.

The simulations of slow and post-stroke hemiparetic gait presented here were constrained to the sagittal plane. Motion in the frontal plane is known to occur at slow speeds (Murray et al., 1984) or when gait is severely impaired by stroke (Kim and Eng, 2004). Muscles which predominantly generate moments in the frontal plane (e.g. gluteus medius) may contribute to support of the center of mass (Anderson and Pandy, 2003; MacKinnon and Winter, 1993), but their effects will not be captured by a two-dimensional model. Nevertheless, at slow speeds, mechanical energy of the COM moving in the frontal plane is negligible compared to sagittal plane movement (Tesio et al., 1998). Furthermore, inspection of the experimental data used to create the simulation of post-stroke hemiparetic gait revealed that mechanical energy expended in the sagittal plane exceeded that in the frontal plane by about a factor of three. Thus, we assert that our two-dimensional model is sufficient for a preliminary study of muscle contributions to COM support for this subject.

Because a two-dimensional model was used, only sagittal plane kinematics were included for tracking; consequently, irregularities were introduced by the simulation to ensure adequate clearance of the swing leg. For example, we found increased non-paretic knee extension in mid- and late-stance, which enabled paretic swing leg clearance (Fig. 1a: simulated knee angle outside error bars at 30–60% gait cycle). The lack of articulation between the pelvis and trunk segments in our model prevented better replication of the absolute position of the COM. Despite an extremely slow, unnatural velocity for the healthy and impaired subjects, intersubject and intrasubject variability was low. Although we were able to reproduce experimental kinematics and vertical ground reaction force with our two-dimensional model, poor tracking of anterior/posterior ground reaction forces reflects model inadequacies and limits our confidence in the results during double support; thus only muscle contributions to midstance support were analyzed.
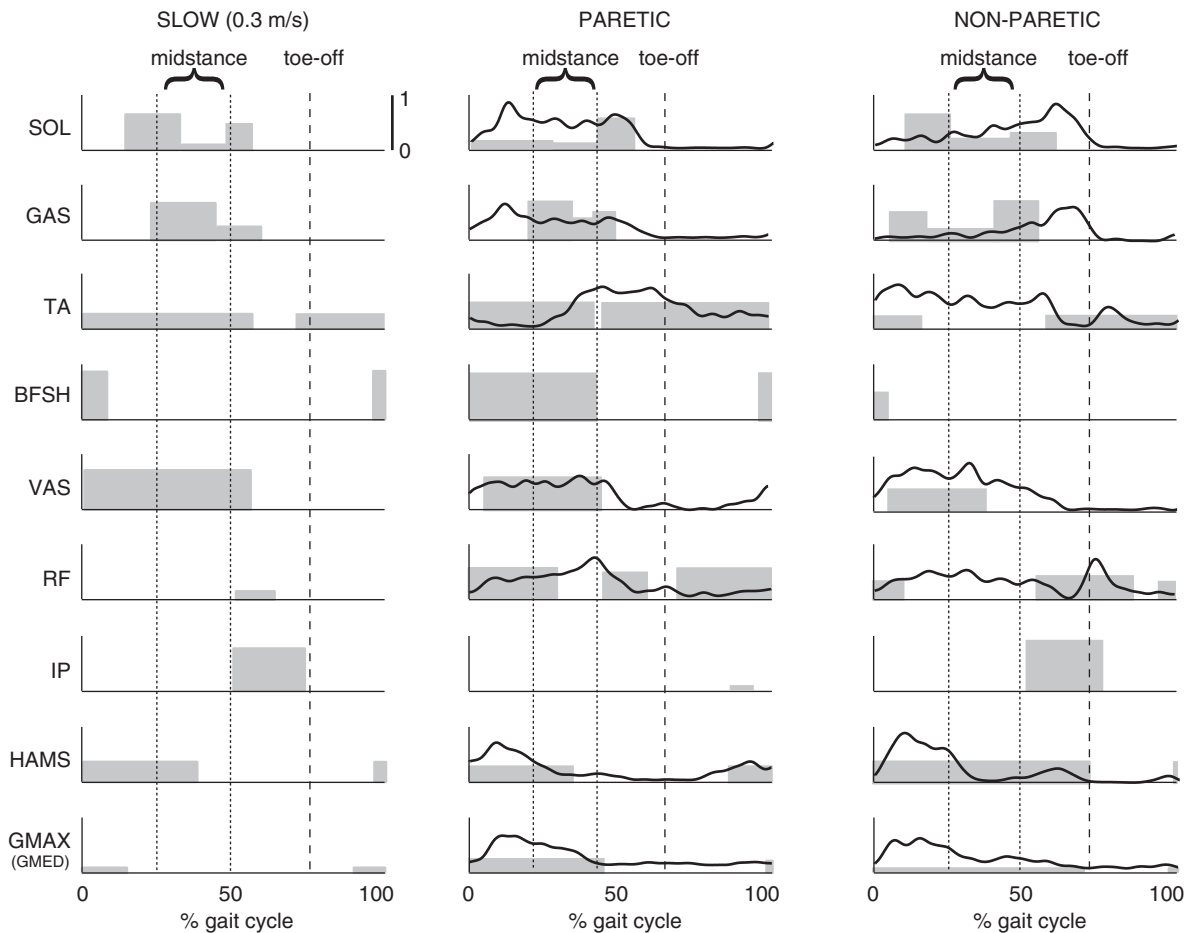
Fig. 2. Feasible muscle excitation patterns over the gait cycle for healthy slow gait and hemiparetic gait and smoothed, rectified EMG data for hemiparetic gait. Excitation of non-paretic SOL, GAS and VAS (but lower in magnitude) and paretic VAS and GAS compared reasonably well with their counterpart excitations in slow gait. Paretic SOL was excited much sooner at foot strike, TA excitation was virtually continuous, and BFSH was excited much in stance also. Excitation magnitude was scaled from 0 to 1, and EMG was normalized to peak amplitude, which was set to 1. Note that EMG from gluteus medius (not GMAX) is shown here, but timing of these muscle groups is similar (Perry, 1992).

Characteristics of post-stroke hemiparetic gait vary widely between individuals, encompassing a range of speeds, muscle impairments, and joint kinematics and kinetics. To build the first muscle-actuated simulation of post-stroke hemiparetic gait, we selected one subject who walked without an assistive device, had no significant frontal plane movement, and exhibited extremely slow (0.3 m/s), asymmetrical gait. This walking speed was on the low end of the typical range following stroke (Olney and Richards, 1996). His stride length (0.5 m) and cycle time (1.67 s) were comparable to values observed in the slowest walking stroke survivors (Turnbull et al., 1995). Paretic and non-paretic stance durations (64% and 72%, respectively) compared favorably to values reported elsewhere (62% and 71%, respectively) (Kim and Eng, 2003). With slow walking speed and excessive knee flexion in paretic midstance, this subject exhibited characteristics similar to those subjects classified as "flexed" by Mulroy et al. (2003). Thus, he provided an ideal case study for investigating

COM support, as he was representative of a class of subjects that have difficulty generating body weight support.

The musculoskeletal properties included in the current model are based on data from healthy adult males (Delp et al., 1990), despite evidence that subjects with "flexed" characteristics exhibit significantly reduced lower extremity strength (Mulroy et al., 2003). Although disuse atrophy may occur following stroke, weakness is predominantly explained by the impaired ability of the central nervous system to activate muscles adequately (Landau and Sahrmann, 2002), and may be greater during walking than during isometric testing (Maegele et al., 2002). Furthermore, reductions in peak isometric force could be compensated for with increased excitation, provided that these levels are sub-maximal. In our simulation, peak excitations were approximately 60% for SOL, VAS and RF, and 70% for GAS. This means that as long as the actual peak isometric force of these muscles was not reduced by more than 40% or
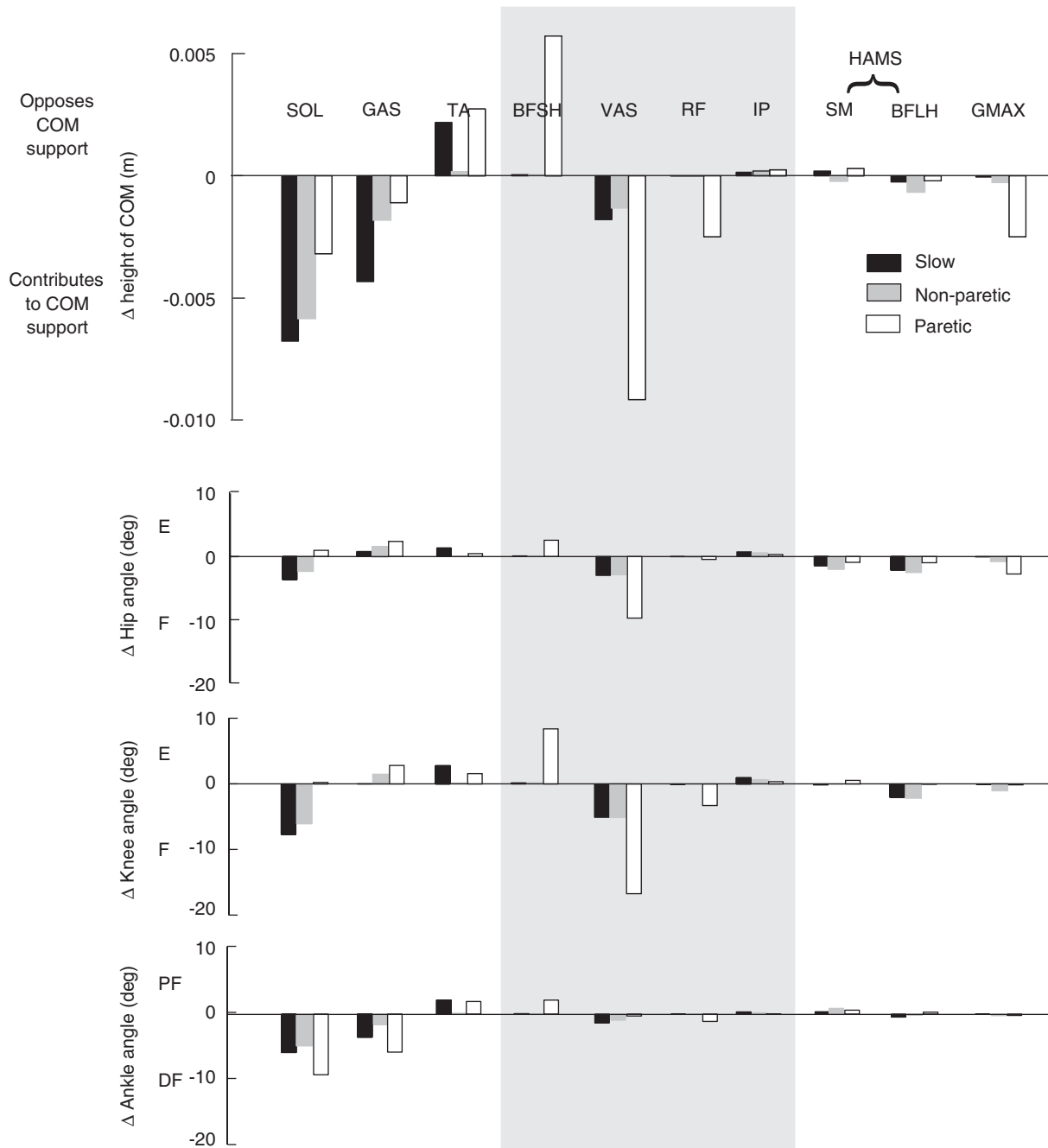
Fig. 3. Contribution of muscles to midstance support of COM. A negative change of the height of the COM when muscle force is set to zero suggests that a muscle contributes to support (i.e. maintains upright posture). Similarly, hip flexion, knee flexion or ankle dorsiflexion with zero muscle force indicates a muscle contributes to joint extension. Non-paretic SOL, GAS and VAS contributed to midstance COM support (gray bars) but their contributions were lower compared to healthy slow gait (black bars). Paretic SOL and GAS contributed even less to support, TA and BFSH opposed support significantly, and VAS, RF and GMAX compensated (white bars).

30%, respectively, then a solution could be found without exceeding the maximal level of excitation. The only muscle excitation which approached maximal values was for BFSH, which is unlikely to experience significant atrophy. We believe that inappropriate timing and level of muscle activation are responsible for the observed asymmetries in kinematics and kinetics.

Our simulation reveals differences in both magnitude and timing of muscle excitation patterns compared to slow, healthy gait.

Increased muscle stiffness, particularly of the ankle plantarflexors, is known to occur due to active and passive mechanisms (Dietz et al., 1981; Berger et al., 1984). A potential implication of increased stiffness is

the reduced need for active recruitment of muscles when plantarflexion is desired or the increased recruitment of antagonistic muscles when dorsiflexion is desired (Lamontagne et al., 2000a). Note that if the passive muscle force output were underestimated in our simulation, then the plantarflexor excitation level would be further reduced, but the contribution of net muscle force to COM support would remain unchanged.

4.1. Muscle coordination patterns

Differences in timing and magnitude of feasible muscle excitations were observed in the unimpaired slow gait and the post-stroke hemiparetic gait simulations. Importantly, premature SOL excitation on the paretic side was found in agreement with previous observations (Knutsson and Richards, 1979) and may prevent the knee from flexing further in response to early stance phase loading. The peak excitation of SOL and GAS in paretic and non-paretic stance was comparable to the peak excitation in slow gait.

Depending on time since stroke and gait speed of subjects tested, reports about post-stroke co-activation in the literature are varied. Shiavi et al. (1987) reported abnormal co-activation during paretic and non-paretic stance. Lamontagne and colleagues (2000b, 2002) hypothesized that excessive co-activation of non-paretic ankle muscles in double support for individuals less than six months post-stroke may be an adaptation to help maintain postural stability during gait. They also observed reduced co-contraction on the paretic side of the most severely affected stroke survivors relative to healthy controls. Conversely, others have categorized stroke survivors who exhibit abnormal co-activation of several paretic muscles in stance (Knutsson and Richards, 1979), and found that subjects who exhibited abnormal stance phase co-contraction of several muscles, including the quadriceps and hamstrings, also flexed the knee less than normal in swing. Similarly, Carlsoo et al. (1974) noted paretic stance phase TA co-activation with ankle plantarflexors. In our simulation based on a single stroke survivor, we found more stance phase co-activation by paretic muscles relative to healthy adults walking very slowly. Our simulated paretic dorsiflexor excitation was essentially continuous with elevated magnitude relative to slow and non-paretic muscles, and may reflect an adaptation occurring in chronic stroke to promote stability given a "flexed" limb posture.

Mulroy et al. (2003) found that patients classified as "flexed" exhibited excessive stance phase flexion of all joints coupled with prolonged quadriceps activity when hip extensor strength was reduced relative to normal. In our simulation, we observed stance phase co-contraction of paretic BFSH and VAS which effectively enhances joint stability while providing COM support despite the flexed knee posture. Although we did not record EMG from BFSH, other investigators have reported co-activation of knee muscles in paretic stance (Knutsson and Richards, 1979).

4.2. Midstance support

Previous simulations of normal self-selected speed gait have confirmed clinical intuition that the ankle plantarflexors contribute significantly to support during stance (Neptune et al., 2001; Anderson and Pandy, 2003). The current results for a single stroke survivor representative of subjects classified as "flexed" by Mulroy et al. (2003) suggest that although the plantarflexors are still important in healthy slow and post-stroke hemiparetic gait, supplemental support is provided by other muscles (e.g. VAS). Because co-contraction is occurring at the paretic knee and ankle, additional muscles (e.g. RF and GMAX) are recruited during paretic stance to maintain upright posture.

A muscle can accelerate joints that it does not span (Zajac and Gordon, 1989). Some muscles extended the hip while contributing to body weight support (and upright posture) (see Fig. 3: slow and non-paretic SOL; slow, non-paretic and paretic VAS; paretic GMAX). Muscles causing midstance knee extension also contributed to body weight support (e.g. slow and non-paretic SOL; slow, non-paretic and paretic VAS; paretic RF). The COM was also supported when muscles induced ankle plantarflexion (e.g. slow, non-paretic and paretic SOL and GAS). Thus, when addressing muscle impairments, the implications for whole body movement should be investigated. For example, in slow and non-paretic midstance, SOL induced hip, knee and ankle extension and therefore promoted upright posture; in paretic midstance, however, SOL had an opposite or negligible effect at the hip and knee, respectively, and its overall contribution to COM support was reduced. Similarly, although GAS promoted ankle plantarflexion in all cases, it flexed the knee and hip more in paretic midstance, and therefore contributed less to COM support. Such cause and effect relationships arise due to the current body configuration and do not depend on the level of muscle excitations.

Co-contraction has been suggested to enhance joint stability (Baratta et al., 1988) and may be beneficial when learning new tasks or in the presence of uncertainty (Lewek et al., 2004; De Visser et al., 2000). Midstance excitation of paretic BFSH and TA stabilized the knee and ankle, respectively, and opposed knee extension induced by VAS. Relaxation of antagonistic BFSH, if possible, would enhance paretic knee extension and ankle plantarflexion, and would reduce the need for supplemental support by proximal muscles, such as GMAX. Antagonistic activity of dorsiflexors during stance counteracts plantarflexor action (Lamontagne et al.,

2000b, 2002) and results from this case study suggest that compensation by proximal muscles is required to enable the maintenance of body weight support normally provided by the plantarflexors. Although co-contraction promotes joint stability, excess muscular activity may be counterproductive for the stroke survivor who can fatigue rapidly.

This case study has shown that the contributions of individual muscles to support in midstance are altered for an individual with post-stroke hemiparesis compared to neurologically healthy older adults walking slowly. Future work will apply three-dimensional simulations to improve our understanding of muscle coordination in post-stroke hemiparetic gait and promote the development of a scientific rationale for therapeutic intervention.

Acknowledgements

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