REDUCED PLANTARFLEXOR CONTRIBUTIONS TO SUPPORT IN POST-STROKE HEMIPARETIC GAIT

[1,2,4]JS Higginson, [1,2]FE Zajac, [5]RR Neptune, [6,7]SA Kautz, [1,2,3]SL Delp
[1]VA Palo Alto Rehab. R&D Ctr., [2]Depts. of Mech. Eng. and [3]Bioeng., Stanford University,
[4]Dept. of Mech. Eng., Univ. of Delaware, [5]Dept. of Mech. Eng., Univ. of Texas at Austin,
[6]VA Brain Rehab. Research Ctr., [7]Dept. of Phys. Ther., Univ. of Florida; email: higginson@me.udel.edu

INTRODUCTION

Muscle-actuated forward dynamic simulations of self-selected and slow walking speeds have shown that the ankle plantarflexors are supplemented by the uniarticular knee extensors to provide body weight support in midstance [1,2]. Patients with severe ankle plantarflexor weakness show reduced walking speed and compensatory strategies related to the strength of hip and knee extensors [3]. This case study presents the first muscle-actuated forward dynamic simulation of post-stroke hemiparetic gait over the entire gait cycle and demonstrates one way that post-stroke muscle contributions to support differ from those of neurologically healthy older adults.

METHODS

Two forward dynamic simulations of slow gait (0.3 m/s) were developed based on the walking patterns of healthy older adults [4] and an individual with post-stroke hemiparesis who walked with his paretic knee abnormally flexed during midstance. A 2D musculoskeletal model consisting of a trunk, pelvis and two legs was developed using SIMM [5] and actuated by 15 Hill-type muscle-tendon units per leg, including the soleus (SOL), gastrocnemius (GAS), vasti (VAS), tibialis anterior (TA), gluteus maximus (GMAX) and biceps femoris short head (BFSH). Muscle excitation patterns (i.e., onset, offset, magnitude) were assumed symmetric for healthy slow gait, and asymmetric for post-stroke gait. Dynamic optimization was used to find the appropriate muscle excitation patterns that best emulated the experimental kinematics and kinetics during slow and post-stroke hemiparetic gait. To assess the contribution of each muscle to support of the center of mass (COM), individual muscle forces were reduced to zero during midstance (i.e., middle third of the total stance duration) while all other muscle forces remained unchanged. The effect of each muscle force perturbation on vertical position of the COM was quantified 0.06 seconds later.

RESULTS AND DISCUSSION

Experimental kinematics and vertical ground reaction forces were reproduced by the simulations of slow and post-stroke hemiparetic gait. Muscle excitations agreed reasonably well with EMG [6]. Compared to healthy slow gait, peak excitation of the paretic and non-paretic plantarflexors were unaltered, paretic TA and BFSH excitations prolonged and increased, and paretic and non-paretic VAS excitations shortened.

In midstance, on the non-paretic side, SOL, GAS and VAS contributed less to support than in speed-matched healthy older adults (Figure 1) due to reduced contributions to extension of the ankle, knee and hip by SOL and GAS. The non-paretic TA no longer opposed COM support, thus less effort was required by the plantarflexors.

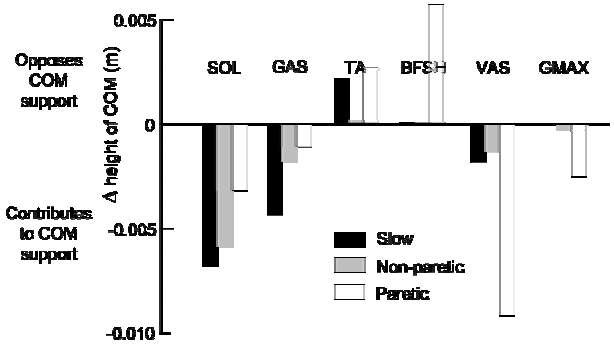

Figure 1: Contributions of key muscles to midstance support of COM in slow and post-stroke hemiparetic gait.

On the paretic side, the plantarflexor contributions were reduced even further (Figure 1), with compensation to COM support provided by VAS, GMAX and other muscles not shown. Although paretic TA and BFSH midstance activity may enhance ankle and knee stability, respectively, these muscle actions opposed COM support. Because the model used in the analysis was 2D, the hip abductor contributions to COM support have been neglected and may be significant [7].

CONCLUSIONS

These simulations represent the first muscle-actuated forward dynamic simulations of healthy slow and post-stroke hemiparetic gait, characterized by a flexed paretic stance knee posture. Despite reorganization of paretic and non-paretic muscle coordination patterns with respect to that of speed-matched healthy older adults, adequate body weight support was provided by altered muscle contributions.

REFERENCES

1. Neptune et al., *J Biomech*, 34(11): 1387-98, 2001.
2. Higginson et al., submitted. *J Biomech*.
3. Mulroy et al., *Gait & Posture,* 18: 114-125, 2003.
4. Chen et al., in press. *Gait & Posture*.
5. Delp and Loan, *Comput Biol Med,* 25: 21-34, 1995.
6. den Otter et al., *Gait & Posture*, 19(3): 270-8, 2004.
7. Anderson and Pandy, *Gait & Posture,* 17: 159-169, 2003.

ACKNOWLEDGEMENTS

We thank G. Chen and D. Kothari for providing the slow gait data [4], L. Worthen and M. Kim for collecting the post-stroke gait data, and the supercomputing resources at Stanford University. Funding was provided by the Rehabilitation R&D Service of the U.S. Department of Veterans Affairs and NIH GM 63495. This work is based on Jill Higginson's Ph.D. thesis (2005).