ISB 2013
BRAZIL
XXIV CONGRESS OF THE INTERNATIONAL SOCIETY OF BIOMECHANICS
XV BRAZILIAN CONGRESS OF BIOMECHANICS

# A FORWARD SIMULATION FRAMEWORK TO PREDICT THE EFFECT OF INCREASED LENGTH AND VELOCITY FEEDBACK DURING GAIT

[1]Karen Jansen, [2]Friedl De Groote, [2]Wouter Aerts, [1]Jacques Duysens, [2]Jos Vander Sloten, [2]Joris De Schutter and [1]Ilse Jonkers
[1] Department of Kinesiology, KU Leuven, Leuven, Belgium
[2]Department of Mechanical Engineering, KU Leuven, Leuven, Belgium; email: friedl.degroote@mech.kuleuven.be

## INTRODUCTION

It has been suggested that a higher reflex excitability is at the origin of gait deficits in a subgroup of hemiparetic subjects [1]. The aim of this study is to develop a simulation framework to predict the effect of increased length and velocity feedback on gait kinematics. To this aim, we extended the musculoskeletal model with a model of length and velocity feedback and a ground contact model. The outcome of the predictive simulations was compared to reported gait deviations of hemiparetic subjects.

## METHODS

Simulations were performed in OpenSim [2]. In a first step control muscle excitations were determined based on experimental gait data (marker trajectories and ground reaction forces) of one control subject walking at 4km/h on an instrumented treadmill. Simulations were based on a musculoskeletal model [3]. The skeletal system is modeled by 12 bodies and 27 degrees of freedom. The leg and trunk joints are actuated by 92 Hill-type muscle-tendon units [4] and the arms are driven by torque actuators. This musculoskeletal model was scaled to the subject's anthropometry using a static trial. Kalman smoothing for inverse kinematics [5], residual reduction algorithm, and computed muscle control [6] were consequently applied to calculate the muscle excitations underlying the measured motion. Corresponding reference states were determined by a forward simulation using the control excitations as inputs.

To predict the effect of higher reflex excitability the musculoskeletal model was then extended with (1) a model of length and velocity feedback for soleus, gastrocnemii, vasti, and rectus femoris; and (2) a contact model to calculate the reaction forces between the feet and the ground, which will differ from the experimental ground reaction forces if the reflex excitability changes.

Muscle spindles give sensory feedback about the length and velocity of the muscles. The feedback model proposed by [7] is adapted to reflect that length feedback only occurs while the muscle fiber is lengthening. The muscle excitation resulting from length feedback $u_l$ is modeled by:

$$\tau_l \frac{du_l}{dt} = \begin{cases} -u_l + k_l l_m^n, & v_m > 0 \\ -u_l, & v_m \leq 0 \end{cases},$$

with $\tau_l$ the latency of the length feedback, $k_l$ the gain of the length feedback, $l_m^n$ normalized muscle fiber length, and $v_m$ muscle fiber velocity. The muscle excitation resulting from velocity feedback $u_v$ is modeled by:

$$\tau_v \frac{du_v}{dt} = -u_v + k_v \max(v_m^n, 0),$$

with $\tau_l$ the latency of the velocity feedback, $k_v$ the gain of the velocity feedback, and $v_m^n$ normalized muscle fiber velocity. Gains vary as a function of the gait cycle. The gain modulation pattern is described based on H-reflex and stretch reflex measurements in both control and stroke subjects performed in our gait lab or reported in literature [8-10]. The total muscle excitation is modeled by:

$$u = u_b + u_l + u_v,$$

where the base excitation $u_b$ consists of the contributions of the central nervous system and unmodeled feedback pathways. Values for $\tau_l$ and $\tau_v$ are based on literature. It was assumed that part of the control excitations was due to length and velocity feedback. Reference length and velocity gains were determined so that the excitation resulting from length and velocity feedback never exceeds the control excitation, and the contribution of length and velocity feedback has the same order of magnitude. Hence, the control base excitations (to be used in step 2) were calculated by subtracting excitations resulting from length and velocity feedback from the control excitations (calculated in step 1).

An elastic foundation model [11] describes the contact between the foot and the ground. The contact geometry is described by a sphere located at the heel and two spheres located at the level of the metatarsal arch. The positions of the spheres were optimized. The optimization criterion was the sum of the squared corrector forces and moments that were needed to drive the simulated gait motion towards the experimentally measured gait motion. Muscle excitations input to the forward analysis to simulate the motion were calculated based on the experimentally measured kinematics and ground reaction forces. Hence, corrector forces and moments are close to zero when the contact force predicted by the elastic foundation model equals the measured ground reaction forces.

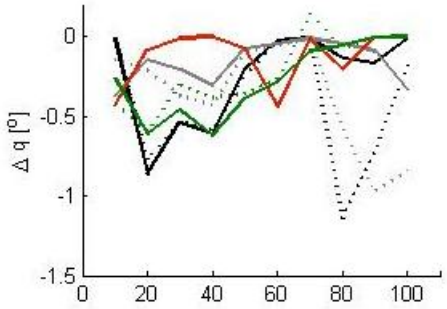
In a second step, a forward simulation was used to predict the effect of increased length and velocity gains. The forward simulation was repeated with gains that equaled 1, 1.5, 3, and 6 times the reference gains for soleus, gastrocnemii, rectus femoris, and vasti respectively. To limit

integration errors, we divided the gait cycle in ten intervals with an overlap of 10%. The initial states for each interval were the reference states at the corresponding time instant. Inputs to this forward simulation were the control base excitations. Each simulation was performed using both control and stroke gain modulation patterns. The mean difference between reference and predicted joint angles was calculated for each of the ten intervals.

## RESULTS AND DISCUSSION

Higher feedback gains for soleus resulted in increased plantar flexion, knee extension, and hip flexion during stance. Using stroke instead of control gain modulation patterns, plantar flexion also increased during swing. Higher feedback gains for gastrocnemii resulted in increased plantar flexion, knee flexion, and hip extension during stance and terminal swing. Using stroke gain modulation patterns, the increase in terminal swing was more pronounced. Higher feedback gains for vasti resulted in increased plantar flexion and knee extension in initial stance and during swing and increased hip flexion at terminal stance and during swing. Higher feedback gains for rectus femoris resulted in increased plantar flexion during stance, and increased knee extension and hip flexion throughout the gait cycle except for terminal swing. The effect of using stroke instead of control gain modulation patterns for vasti and rectus was limited (Figure 1).

a. Difference in ankle angle
(increased plantar flexion is negative).




b. Difference in knee angle
(increased knee extension is positive).

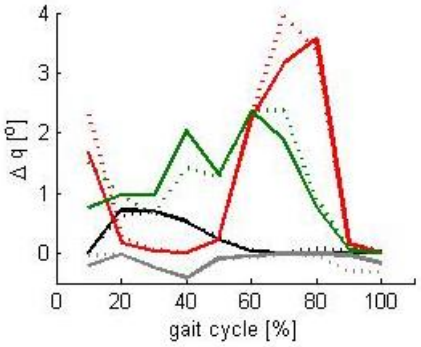



**Figure 1**: Difference between reference and predicted joint angles when length and velocity gains were increased by a factor three for soleus (black), gastrocnemii (gray), vasti (red), and rectus femoris (green) using control (solid line) or stroke (dotted line) gain modulation patterns.

The predicted increased plantar flexion, knee flexion, and hip extension during terminal swing resulting from augmented feedback gains for the gastrocnemii is in accordance with the foot-floor contact with foot flat or toes first often observed in hemiparetic subjects [1]. The predicted increased plantar flexion resulting from higher feedback gains for all studied muscles is in accordance with the observations of Mulroy et al. [12] in one group (Extended) of hemiparetic subjects. The predicted increased knee extension resulting from augmented feedback gains for soleus, vasti, and rectus femoris is in accordance with the knee hyperextension during stance and the decreased knee flexion during swing observed in a large number of hemiparetic subjects [1, 12].

Our study has some limitations. First, the base excitations and initial states were determined from a simulation of normal gait. Hence, we cannot predict compensatory strategies adopted by hemiparetic subjects. Second, the determination of the reference gains was based on assumptions regarding the magnitude of length and velocity feedback excitations. However, we believe that the proposed framework is sufficiently accurate to predict in which direction joint angles will change when altering length and velocity feedback gains. The advantage of predictive simulations is that the effect of isolated changes in control parameters can be assessed.

## CONCLUSIONS

We presented a simulation framework to predict the effect of enhanced length and velocity feedback on joint kinematics during gait. The predicted changes in kinematics resulting from increased length and velocity feedback in soleus, gastrocnemii, vasti, and rectus femoris are in accordance with gait deviations observed in a large number of hemiparetic subjects. Hence, our predictive simulations support the idea that higher length and velocity reflex excitability might be at the origin of some of the gait deficits of spastic, hemiparetic subjects.


## ACKNOWLEDGEMENTS

The support of KU Leuven-BOF PFV/10/002 Center-of-Excellence Optimization in Engineering (OPTEC), G.0395.09 of the Research Foundation – Flanders, KU Leuven's Interdisciplinary Research Program IDO/07/12, and Materialise Chair for Image-based, Patient-specific Biomechanics is gratefully acknowledged.


## REFERENCES


1. Knutsson E, Richards C. *Brain*. **102**:405-430, 1979.
2. Delp SL et al. *IEEE T Biomed Eng*. **54**:1940-1950, 2007.
3. Hamner S et al. *J Biomech*. **43**:2709-2716, 2010.
4. Zajac FE. *Crit Rev Biomed Eng*. **17**:359-411, 1989.
5. De Groote F et al. *J Biomech*. **41**:3390-3398, 2008.
6. Thelen DG et al. *J Biomech*. **39**:1107-1115, 2006.
7. Ogihara N, Yamazaki N. *Biol. Cybern.* **84**: 1-11, 2001.
8. Larsen B. *Exp Brain Res*. **170**:555-566, 2006.
9. Faist M. et al. *Brain*. **122**:567-579, 1999.
10. Yang JF et al. *Can J Neurol Sci*. **18**: 443-452, 1991.
11. Péréz-González, A et al. *Med Eng Phys*. **30**:387-398, 2008.
12. Mulroy S et al. *Gait & Posture*. **18**:114-125, 2003.