

THE USE OF PREDICTIVE SIMULATIONS TO EVALUATE THE EFFECTS OF GASTROCNEMIUS HYPER-REFLEXIA ON GAIT KINEMATICS

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INTRODUCTION

People with cerebral palsy (CP) often show problems with gait due to both neural and non-neural impairments [1]. Optimal treatment selection is challenging because of the complex, multilevel nature of these problems. Neuro-musculoskeletal modelling might provide a valuable source of information to assist in treatment selection. Predictive simulations can be used to gain insights into how common impairments, such as hyper-reflexia (spasticity), affect gait outcomes. The aim of this study was to evaluate how gastrocnemius hyper-reflexia affects gait kinematics by using predictive simulations.

METHODS

We used a two-dimensional OpenSim [2] model with 14 Hill-type musculotendon complexes and 9 degrees of freedom for forward dynamic gait simulations. An impact model from Hunt & Crossley [3] was used to calculate contact forces. The reflex-based muscle control model described by Geyer and Herr [4] was implemented into SCONE (<http://scone.software>), a recently developed platform for forward dynamic, predictive simulations. In this controller, muscle excitations are a combination of constant signals and reflexes, based on the muscle length or muscle force and the active phase of gait. The initial pose and reflex gains were optimized by minimization of the cost of transport, using a metabolic energy cost model described by Wang et al. [5]. The optimization was performed using the Covariance Matrix Adaptation Evolution Strategy (CMA-ES) [6]. Each simulation was optimized twelve times with different initial guesses. The SCONE software used the OpenSim 3.3. engine for simulation, and simulation time was 10 seconds. Penalties were applied when the model walked slower than 0.3 m/s, when the center of mass was below 0.85 m, when the ankle angle was beyond 60 degrees dorsi- or plantarflexion, and when the knee limit force (which mimics knee ligaments) exceeded a threshold. The kinematics predicted by the forward dynamic simulations of the model were

compared to experimental data of healthy adults' gait [7].

Subsequently, an extra force-based reflex [8] with a constant gain was added to the gastrocnemius, mimicking hyper-reflexia. Different gains ($K_F=[0.5, 1.0, 1.5]$) of this extra reflex were implemented and after optimization, resulting kinematics were compared to the healthy gait simulations.

RESULTS AND DISCUSSION

Our predictive gait simulations resulted in an overall good agreement with experimental gait data (Fig. 1). These findings indicated that an optimized control model assuming that all muscle control during gait is based on reflexes matches experimental data well. Differences were mainly visible in the knee angle, in which the model showed too much extension in late-stance, as well as too much flexion during swing. The hip also showed too much flexion during swing.

Implementing low levels of force-based hyper-reflexia ($K_F=0.5$ and $K_F=1.0$) only resulted in slight changes in kinematics. The hyper-reflexia was mainly captured by some more ankle plantar flexion in swing and a slightly increased anterior tilt of the pelvis. However, when the gain of the extra reflex was further increased ($K_F \geq 1.5$), a relatively major change in kinematics appeared. The model showed a toe-walking pattern, with higher plantar flexion of the ankle during the entire gait cycle. During stance both the hip and the knee showed more extension compared to the healthy gait simulation. The pelvis tilt angle was restored towards a similar value and range of motion as in the healthy gait simulation.

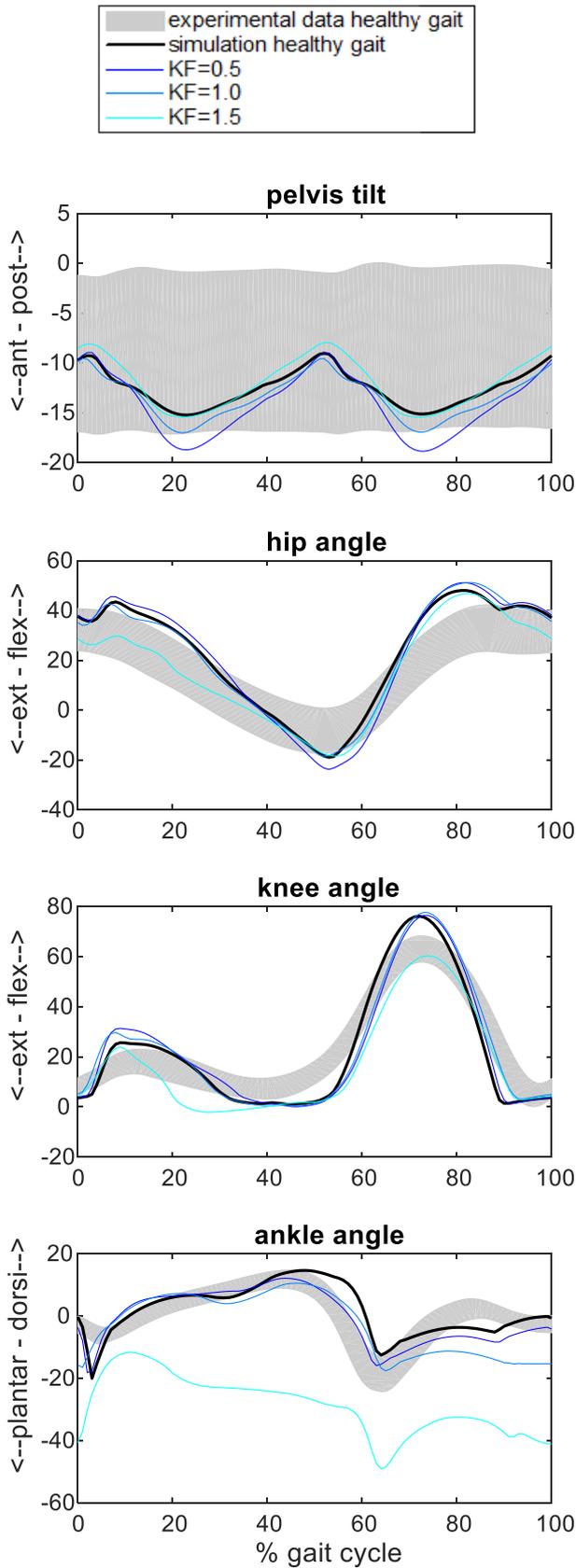


Fig 1: Predictive simulations showed an overall good agreement with experimental gait data. Low levels of gastrocnemius hyper-reflexia only had a slight effect on kinematics. When the hyper-reflexia exceeded a threshold, a toe-walking gait pattern occurred.

The results of this study showed how hyper-reflexia of solely the gastrocnemius could affect gait kinematics. Different levels of hyper-reflexia were displayed differently in the gait kinematics of the predictive simulations.

Interestingly, despite increased activity of the bi-articular gastrocnemius, the toe-walking pattern showed increased knee extension compared to healthy gait simulations. This phenomenon could be caused by the plantar flexion-knee extension couple which could result in this true equinus gait as described by Rodda et al. [9].

This study suggests that predictive simulations could become a powerful tool in gaining insight into how different impairments affect the gait pattern. This information may be relevant for clinical decision making, when using gait analysis in the process of deciding which muscle is affecting gait and should be treated. Further studies are needed to further evaluate the effects of hyper-reflexia of both the gastrocnemius and soleus. Moreover, it is important to validate the predictive simulations against known pathology and interventions.

CONCLUSIONS

By applying predictive simulations, these first results showed that different levels of gastrocnemius hyper-reflexia are manifested differently in the gait pattern. Follow-up studies can now focus on validation, by comparing the results of predictive simulations including different levels of hyper-reflexia to experimental data from matched individual cases.

REFERENCES

1. Graham HK, et al., *Nat Rev Dis Primers*, **2**: 1-24, 2016.
2. Delp SL, et al., *IEEE Trans Biomed Eng* **54**: 1940-1950, 2007.
3. Hunt KH & Crossley FRE, *J Appl Mech*, **42**: 440-445, 1975.
4. Geyer H & Herr H, *IEEE Trans Neural Syst Rehabil Eng*, **18**: 263-273, 2010.
5. Wang JM, et al., *ACM Trans Graph*, **31**: 25, 2012
6. Hansen N, *StudFuzz*, **192**: 75-102, 2006
7. Bovi G, et al., *G&P*, **33**: 6-13, 2011
8. Falisse A, et al., *PLoS ONE*, **13**: e0208811, 2018
9. Rodda JM, et al., *J Bone Joint Surg*, **86**: 251-258, 2003