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# Predicting the effects of knee extensor muscle weakening and strengthening on a post-stroke gait

Gilmar F. Santos<sup>1</sup>, Eike Jakubowitz<sup>1</sup>, Nicolas Pronost<sup>2</sup>, Thomas Bonis<sup>2</sup>, Christof Hurschler<sup>1</sup>

<sup>1</sup>Laboratory for Biomechanics and Biomaterials, Department of Orthopedics, Hannover Medical School, Hanover, Germany

<sup>2</sup>Université de Lyon, Université Claude Bernard Lyon 1, CNRS LIRIS, Lyon, France

Email: [FernandesdosSantos.Gilmar@mh-hannover.de](mailto:FernandesdosSantos.Gilmar@mh-hannover.de)

## Summary

Stroke may cause different gait abnormalities, such as knee hyperextension and stiff-knee gait. This study used predictive simulation to investigate how the weakening and strengthening of the knee extensor muscles affect the gait pattern of a post-stroke patient. The prediction result showed impairments similar to those observed in the gait obtained by the inverse dynamics. While the predictive simulation of muscle weakening corrected the stiff-knee gait, the gait prediction of muscle strengthening decreased the knee hyperextension exhibited in the gait pattern of the patient.

## Introduction

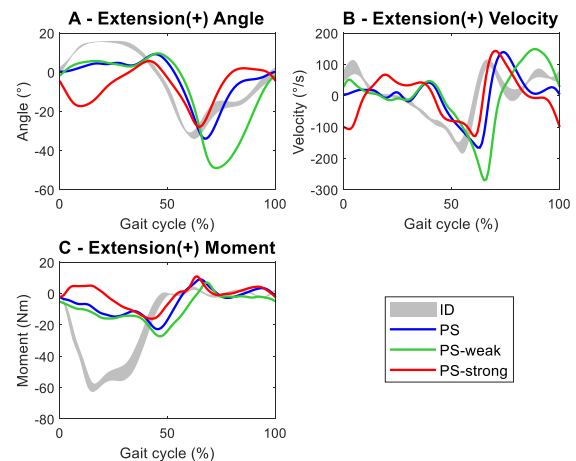
Individuals who suffered stroke may present impaired gait. Knee hyperextension and stiff-knee gait (SKG) are deviations commonly exhibited by this population, but these gait patterns could have different causes [1]. Understanding how to improve these impairments may support the rehabilitation. Predictive simulation was used in order to investigate the cause-effect relationship between changes in the musculoskeletal system and the observed gait abnormalities in a typical post-stroke patient. In a simplified approach, the purpose of this study was to predict the effects of the weakening and strengthening of the knee extensor muscles on pathological gait, and to investigate whether the specific changes could improve the gait pattern of the patient.

## Methods

The gait analysis was performed for a female patient (age: 46 years; height: 1.60 m; mass: 63.8 kg) walking overground at a self-selected gait speed ( $0.55 \pm 0.04$  m/s). Inverse dynamics (ID) results were obtained using a scaled lower body model (gait2392) and the tools available in OpenSim (v. 3.3). The 3D musculoskeletal model used for predictive simulations (PS) was based on the one used in ID. The PS was formulated as an optimal control problem [2]. The observed speed in the gait analysis was imposed. Personalized muscle-tendon parameters (optimal fiber length, maximal isometric force and tendon slack length) were used for PS. An optimal control problem was solved using the ID joint moments to estimate the personalized muscle-tendon parameters [2]. With this parameter set, it was possible to predict a pathological gait without directly relying on the kinematics and kinetics of the collected gait data. In order to investigate the effect of muscle weakening (PS-weak), maximal isometric force of the knee extensor muscles in PS was decreased by 50%. Similarly, muscle strengthening (PS-strong) was predicted by increasing maximal isometric force by 50%.

## Results and Discussion

The knee hyperextension and SKG observed in the ID were predicted, but PS resulted in less knee extension during the stance phase than the ID (Fig. 1A). PS-weak increased knee hyperextension and corrected the SKG, while the opposite was observed for PS-strong (Fig. 1A). These results indicate that weak knee extensor muscles is related to knee hyperextension in this patient, as also reported by Mulroy et al. [3]. The decreased peak of knee flexion in the swing phase was accompanied by low knee flexion velocity (Fig. 1B), which has been identified as a cause of SKG [4]. The change of dominance from knee flexion to extension moment in early stance phase in PS-strong (Fig. 1C) allowed knee flexion, which corrected knee hyperextension.



**Figure 1:** Knee extension (A) angle, (B) velocity and (C) moment for inverse dynamics, predictive simulation, knee extensor muscle weakening and knee extensor muscle strengthening results.

## Conclusions

The alteration of the knee extensor muscle strength predicted improvements in the post-stroke gait and identified possible causes of the knee hyperextension and SKG. Future studies may investigate other gait abnormalities and should include more patients in the analysis.

## Acknowledgments

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