Modeling Postural Control in Parkinson‘s Disease

How damaged neural pathways change our movements

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Motivation

The fear of falling due to changes in gait and disturbances in equilibrium leads to a decrease in quality of life in Parkinson’s patients [1]. 13 % of all Parkinson’s patients require medical treatment due to falling each year [2]. The neuropathology of Parkinson’s Disease is characterized by neural loss in the substantia nigra (see Fig. 1). However, its correlation to the changed walking style still remains unknown [3].

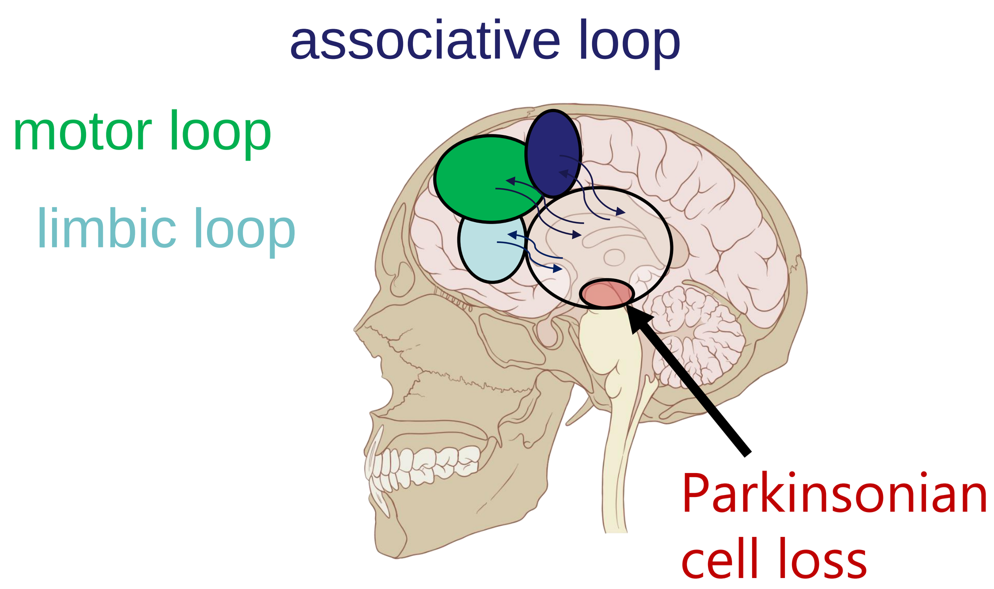

Fig. 1: Neurodegeneration in Parkinson’s Disease.

Goals

A simplified dynamic model is used to simulate muscular behaviors in Parkinson’s patients. An optimization of model parameters indicates if Parkinsonian walking results from changed modulation of central pattern generators, neural networks correlated to the creation of cyclic movement patterns such as in walking [4].

Procedure

Parkinsonian gait measurements were provided by Shiraz University and include marker trajectories and ground reaction forces for the patient during tests. In order to get an understanding of the Parkinsonian gait, a template model is optimized for fitting ground reaction forces and joint torques of the measured gait.

Methods

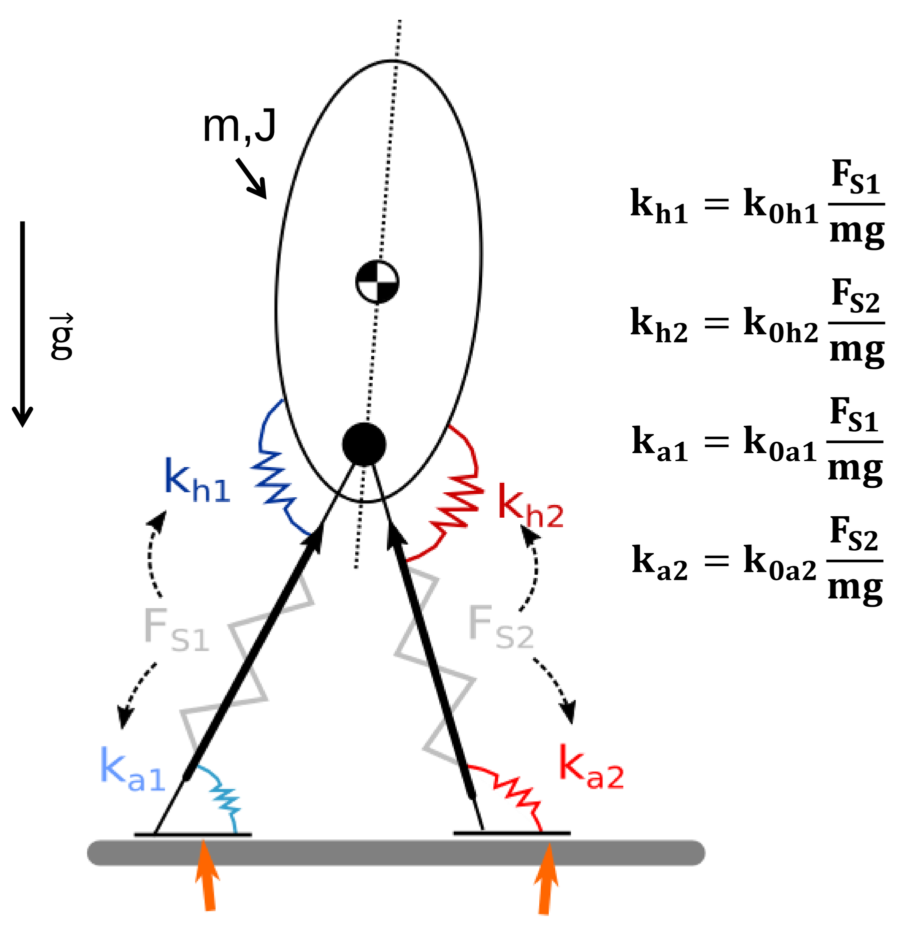

Fig. 2: FMCHA model and stance phase equations.

FMCHA (see Fig. 2) is the simulation model used during **stance phase**. FMCHA stands for “force modulated compliant hip and ankle”. It is a simplified representation of the human locomotion system consisting of a trunk paired with massless, compliant legs. Rotational springs represent ankle and hip stiffness modified over force feedback.
The model can create force and torque trajectories similar to healthy human measurements.

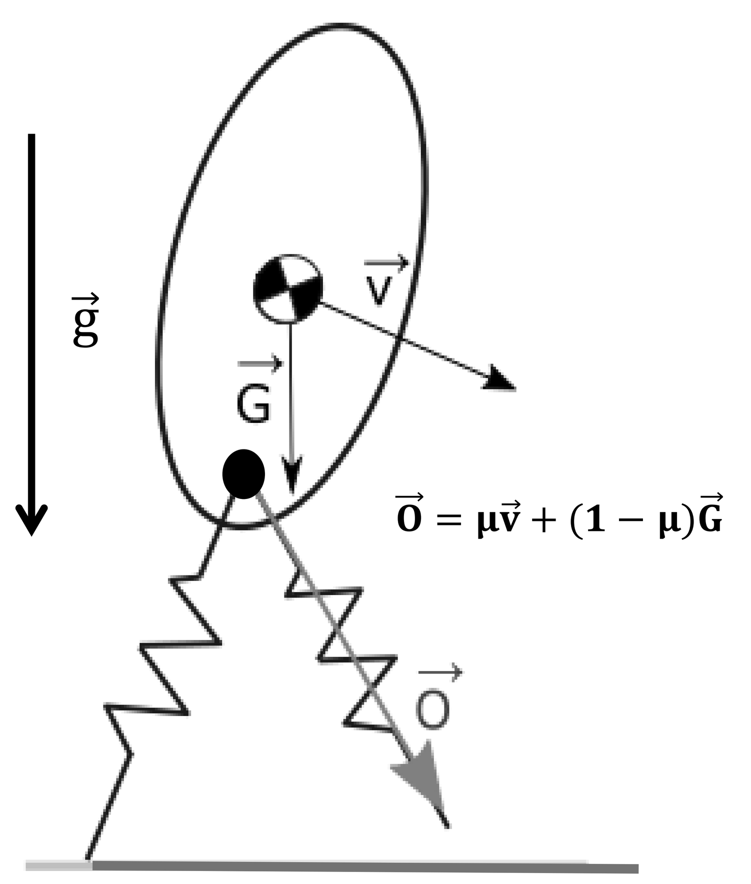

Fig. 3: VBLA model and swing phase equations [5].

For the **swing phase** leg adjustment, the **VBLA** model is used (see Fig. 3). It leads to robust and human-like gait [5].

Results

Parkinsonian-like ground stance duration, ground reaction forces and ankle torques were achieved with the model. However, the measured hip torques could not be represented by the model (see Tab. 1). In contrast to healthy subjects, Parkinsonian walking has a negative VPP position, a parameter that correlates with walking stability [6]. This aspect could not be reproduced in the simulation.

Parameter	Exp.	Sim.
Stance duration [%]	69	71
Max. vertical force [BW]	1.07	1.03
Max. horizontal force [BW]	0.17	0.22
Max. hip torque [BW*L0]	0.27	0.028
Max. ankle torque [BW*L0]	0.24	0.23
VPP position [L0]	-0.35	0.03

Tab. 1: FMCHA model and stance phase equations. Values are normalized to bodyweight [BW] and leg rest length [L0].

Conclusions

The negative VPP location shows that Parkinsonian gait is instable. The template lacks an additional control mechanism in order to stabilize it for negative VPP locations, which is why it cannot be fit to Parkinsonian gait. Equivalently, the outcomes suggest that the assumed change in CPG gains due to Parkinson’s Disease turns walking instable, which is why Parkinson’s patients require high level control mechanisms to stabilize locomotion. This coincides with the observation that Parkinson’s patients struggle with equilibrium and have to concentrate on walking in a safe manner [7].

References

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