

SHAPING ANKLE MECHANICAL IMPEDANCE IN SPASTICITY DURING THE STANCE PHASE OF GAIT

Sebastião Oliveira¹, João Gouveia¹, Rui Coelho¹, and Jorge Martins¹

¹ IDMEC, Instituto Superior Técnico, Universidade de Lisboa, Portugal

sebastiao.e.oliveira@tecnico.ulisboa.pt; rui.coelho@tecnico.ulisboa.pt; jorgemartins@tecnico.ulisboa.pt

joao.g.gouveia@tecnico.ulisboa.pt

KEY-WORDS: Ankle Impedance, Neuromuscular Model, Reflexes, Spasticity.

1 Introduction

Neurological disorders like stroke and cerebral palsy impair movement control, reducing independence and quality of life. The central nervous system (CNS) regulates movement through dynamic motion primitives, modulated by reflexes to maintain walking stability. The ankle, essential for propulsion and balance, is particularly impacted by upper motor neuron disorders, which disrupt voluntary control and reflex pathways, altering its mechanical impedance. This study developed a reflex-based locomotion model, building on Geyer and Herr [1], and extended by Bruel et al. [2], integrating skeletal, muscular, and neurological components. Spasticity was incorporated to analyze its effects on ankle impedance—stiffness, damping, and inertia—during the stance phase of gait.

2 Summary of the Work Developed

The work developed in the scope of this study can be divided onto three main categories, depicted by the following subsections.

2.1 Construction of the model and optimization of its parameters

The model framework was developed in Simulink, heavily inspired by the work of Bruel et al. [2]. It extends Geyer and Herr's reflex circuits by adding pathways for the soleus (SOL), gastrocnemius (GAS), and their antagonist, the tibialis anterior (TA) muscles. These extensions include direct force, spindle, and length pathways for all three muscles, along with reciprocal spindle pathways between the plantar flexors (SOL and GAS) and TA, resulting in a more complex and physiologically accurate model that in the following stage of this study allowed for the detailed simulation of spasticity. The gain parameters of the new model were optimized using the covariance matrix adaptation evolution strategy (CMA-ES), a technique well-suited for non-linear, non-convex functions [3]. The objective function incorporated parameters commonly used to quantify gait, such as metabolic cost, joint angle limitations, and joint torques.

2.2 Implementation of spasticity

The effects of neurological impairments on gait were simulated by adjusting bilateral plantar flexor reflex parameters to mimic hyperreflexia, reduced presynaptic and reciprocal inhibition, and neural weakness—conditions associated with spasticity, a disorder marked by hyperexcitability of stretch reflexes [2]. Reflex parameters for the soleus (SOL) and gastrocnemius (GAS) were altered simultaneously during the stance phase, as these agonist

muscles are often impaired together. Impairments were modeled across all three reflex types—force (F), length (L), and velocity (V)—affecting both direct and reciprocal pathways of the plantar flexors.

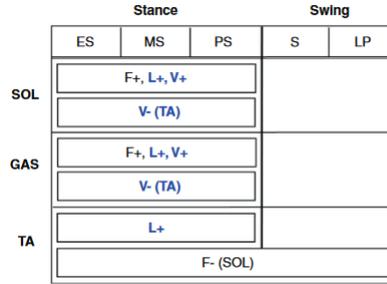


Figure 1: Implemented gait controller. Adapted from [2]

2.3 Estimation of Ankle Impedance

Ankle impedance was estimated by replicating the work by Rouse et al. [4] within a computational simulation framework. A perturbation was applied to the ankle torque, and its response in the angle was assessed. Impedance was estimated by isolating the perturbation-induced torque and angle responses, achieved by subtracting the average unperturbed profiles from the perturbed trials. A second-order parametric model (Eq.1) was used to characterize ankle impedance, where T_p is the torque response, I_{tot} represents total inertia, b_a and k_a are the damping and stiffness coefficients, respectively, and θ_p being the angular perturbation displacement.

$$T_p = I_{tot}\ddot{\theta}_p + b_a\dot{\theta}_p + k_a\theta_p \quad (1)$$

The derivatives were computed numerically in MATLAB by fitting a second-order polynomial to four points around each time point, using the polynomial coefficients to quantify the derivative. Impedance parameters were then estimated using least squares estimation over a 100 ms window. The new model was validated using various metrics, including a direct comparison of muscle activation profiles with those from previous models. Additionally, the method for estimating ankle impedance performance was assessed using the Variance Accounted For (VAF).

Acknowledgements

The authors acknowledge Fundação para a Ciência e a Tecnologia (FCT) for its financial support via the projects LAETA Base Funding (DOI: 10.54499/UIDB/50022/2020) and ReflexES (DOI: 10.54499/2022.04834.PTDC).

J. Gouveia acknowledges support by FCT through the PhD scholarship 2021.06844.BD

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