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A NEUROMUSCULOSKELETAL MODEL TO SIMULATE PASSIVE WRIST EXTENSION TEST OF SPASTICITY

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Clinical Biomechanics Award: No

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Emerging Scientific Award sponsored by Professor J De Luca: Yes

Promising Scientist Award sponsored by Motion Analysis: Yes

Introduction and Objectives: Spasticity is a motor disorder characterized by a velocity-dependent increase in tonic stretch reflexes [1], commonly seen in many neurological disorders. Clinically, spasticity is measured by an examiner rotating a joint and simultaneously estimating the resistance according to an ordinal scale. However, the limited reliability of the measurement and the impossibility to discriminate between the underlying neural (stretch reflex) and non-neural contributions have been the motivation to develop methods describing resistance joint torque quantitatively. The aim of this study is to develop a forward neuromusculoskeletal model consisting of the explicit musculotendon, muscle spindle, and motoneuron pool models, which can simulate passive wrist extension test of spasticity.

Methods: Neuromusculoskeletal model

The neuromusculoskeletal modeling includes muscle spindle modeling, motoneuron pool modeling, muscle activation dynamics and musculotendon modeling of the wrist flexors.

Muscle spindle modeling: The modified hybrid $V^{0.6}$ model was used to model the firing characteristics of the muscle spindle [2].

Motoneuron pool modeling: The motoneuron pool transformation was modeled as a sigmoid function which ranges from 0 to 1, representing the motoneuron neural excitation as a function of the muscle spindle-firing rate.

Activation dynamics: Activation dynamics can be described as the process of converting the neural excitation to muscle activation as a first order differential equation [3].

Musculotendon modeling: The musculotendon model consists of one lumped muscle actuator which represented synergistic wrist flexors and one passive plant. The passive plant can be considered as the passive viscoelastic properties of the joint. Wrist extensors were not included in the model. The Hill-type musculotendon model was used to simulate musculotendon dynamics of the lumped muscle actuator [4].

Experimental Setup and Protocol

Passive extension of the wrist at a slow (5°s^{-1}) and a fast (236°s^{-1}) velocity were performed using a newly developed device Neuroflexor (Aggero MedTech AB, Solna Sweden). The resistance torque induced by the passive wrist extension was measured. Range of wrist movement was set between 20° flexion and 30° extension. An example data from a chronic stroke patient (male, hand length = 0.09m, weight = 69kg) was reported.

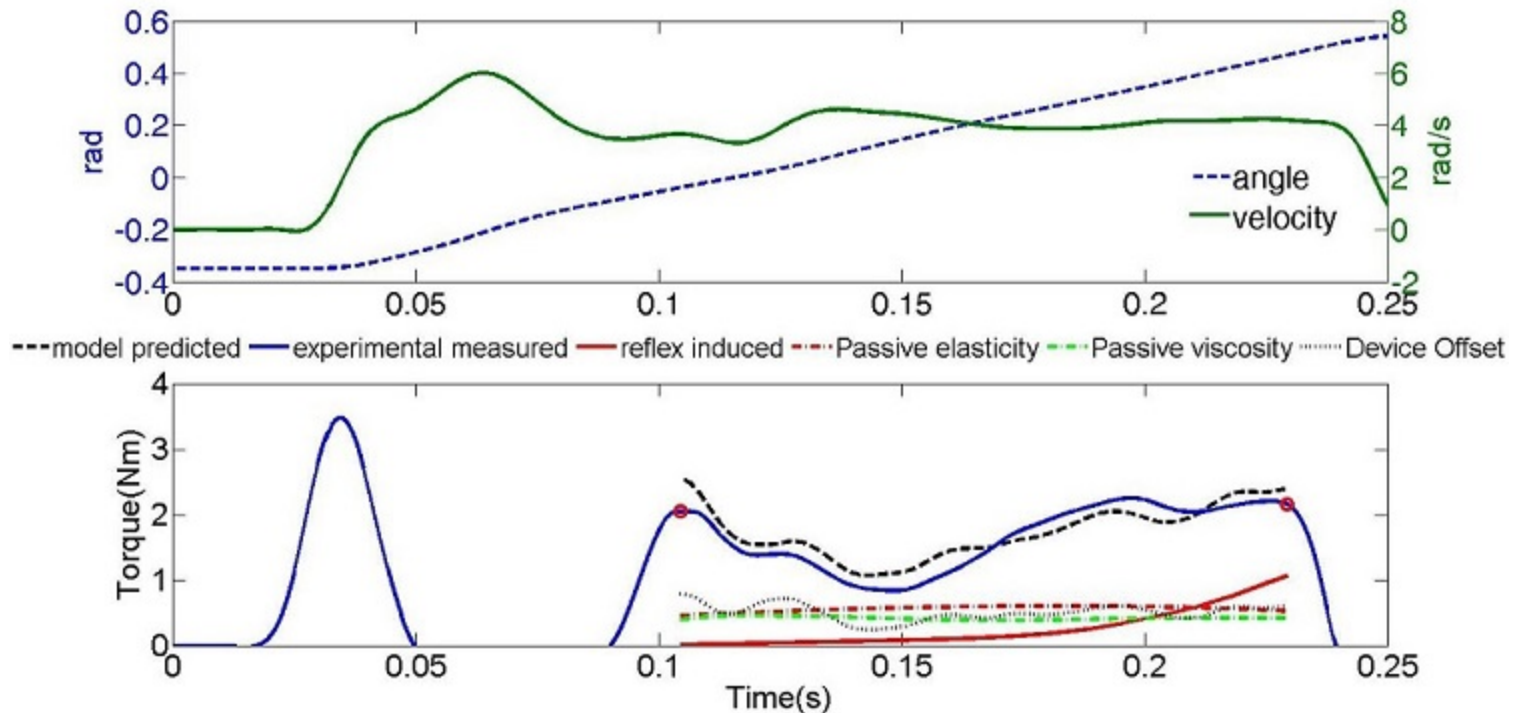
Optimization

It is assumed that no reflex induced muscle resistant torque in the slow movement; hence the resistant torque is purely caused by the passive plant. Nonlinear least squares was used to approximate four passive parameters: elasticity coefficient K_p , viscosity coefficient B_p , and non-linear exponential coefficients k_1 and k_2 . Genetic algorithm (GA) was used to identify four optimal stretch related parameter: motoneural pool parameter μ and σ , dynamic and static spindle gain G_d and G_s , so that the root-mean-square error between the measured and predicted resistant torque in part of fast movement (approximately from 0.1 to 0.23ms, Fig 1) was minimized. Previous studies have reported that the static

and dynamic gains of the spindle in the pathological group did not differ significantly from the healthy controls [6]. Therefore, constraints were set to vary $\pm 10\%$ from the reported values (2 and 4, respectively).

Results: Simulation results from one chronic stroke patient are illustrated in the Fig 1. Four passive parameters ($[K_p = 1.54$, $B_p = 0.1$, $k_1 = 0.05$, $k_2 = 3.2]$, $R^2 = 0.998$) were determined. Four stretch-related (neural) parameters ($[\mu = 157.3$, $\sigma = 10.1$, $G_l = 2.1$, $G_v = 4.1]$) were found and the minimal root-mean-square error (cost function) was 5.4.

Figure:



Caption: The top: the angular velocity and position were prescribed in the simulation of the fast wrist extension. The bottom: the contributions of the torques from passive muscle properties (elasticity and viscosity) and the stretch reflex to the total resistance torque were specified. The optimization was only performed during the period marked with red circles.

Conclusion: By identifying important passive and reflex related parameters using optimization, the neuromusculoskeletal model can reliably describe non-neural and neural related contributions to the total joint resistant torque. The figures illustrate a good fit between the predicted and experimental measured resistant torque. Due to the large measurement error during the period 0.05-0.09ms and the latency of the reflex, the optimization was only performed for part of the fast movement, where the angular velocity maintained stable. Due to the fact of interdependency of the parameters and the difficulties of obtaining gradient, GA was used in the current study. In conclusion, the proposed neuromusculoskeletal model can simulate the passive wrist extension test of spasticity.

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