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A neuromusculoskeletal model to simulate the constant angular velocity elbow extension test of spasticity

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Abstract

We developed a neuromusculoskeletal model to simulate the stretch reflex torque induced during a constant angular velocity elbow extension by tuning a set of physiologically-based parameters. Our model extended past modeling efforts in the investigation of elbow spasticity by incorporating explicit musculotendon, muscle spindle, and motoneuron pool models in each prime elbow flexor. We analyzed the effects of changes in motoneuron pool and muscle spindle properties as well as muscle mechanical properties on the biomechanical behavior of the elbow joint observed during a constant angular velocity elbow extension. Results indicated that both motoneuron pool thresholds and gains could be substantially different among muscles. In addition, sensitivity analysis revealed that spindle static gain and motoneuron pool threshold were the most sensitive parameters that could affect the stretch reflex responses of the elbow flexors during a constant angular velocity elbow extension, followed by motoneuron pool gain, and spindle dynamic gain. It is hoped that the model will contribute to the understanding of the underlying mechanisms of spasticity after validation by more elaborate experiments, and will facilitate the future development of more specific treatment of spasticity.

Keywords: Elbow spasticity; Stretch reflex; Neuromusculoskeletal modeling; Constant angular velocity extension

1. Introduction

Spasticity has been clinically defined as a velocity-dependent increase in resistance to externally imposed joint movement [1]. Although neuroscientists have identified distinctly different ways (e.g., increase in motoneuronal excitability, and increase in the amount of excitatory synaptic input elicited by muscle stretch) that the neuromuscular system could produce an enhanced reflex response to muscle stretch, the exact mechanisms and their relative contributions on the enhanced reflex response are still far from conclusive [2].

In the past decade, a few theoretical models have been developed to help better understand the pathophysiology of elbow spasticity [3–6]. These models were of different complexity and based on different experimental protocols. Feng and Mak [3,4] modified the pendulum model of the knee joint proposed by He and his co-workers [7,8] to study elbow spasticity. In their study, the pendulum motion of the elbow joint was simulated by optimizing a set of stretch reflex parameters within a phenomenon-based stretch reflex loop model. No explicit muscle spindle and motoneuron models were incorporated in their scheme. Their protocol required the subjects to be tested in a prone position, which could affect the subject's muscle tone. Their model was applicable to subjects with mild spasticity. For subjects with severe spasticity, the tone might keep the elbow in a flexed position, which prohibited the pendulum test. Schmit et al. [5] modeled the reflex torque response of the spastic elbow flexors to a large amplitude, constant angular velocity elbow extension and used a simple torque equation, which was composed of the physiological cross-section areas, the moment arms, and the so-called activation functions of the biceps, brachioradialis, and brachialis to estimate the stretch reflex induced muscle torque. Their formulation did not explicitly take into account the force–length and force–velocity relationships of the contractile element. No explicit muscle spindle and mo-

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toneuron pool models were incorporated in their analysis. Zhang and Rymer [6] used a system approach to identify the reflex and intrinsic mechanical properties simultaneously as a function of background muscle torque for the elbow joint. Randomized, small amplitude angular position (±1.5°) perturbations were applied, and the joint angle and torque were measured. A lumped parametric model, which incorporated state-dependent limb inertia, joint viscosity, elastic stiffness, static and dynamic stretch reflex gain parameters, was employed to quantify the effects of reflex and non-reflex actions as function of background muscle torque. However, individual muscle responses cannot be differentiated.

The objective of this study is to develop a neuromusculoskeletal model to simulate the stretch reflex response induced during a constant angular velocity extension at spastic elbow. This model extended the past modeling efforts in the investigation of elbow spasticity by incorporating explicit musculotendon, muscle spindle, and motoneuron pool models and defining a set of physiologically-based parameters at each prime elbow flexor to analyze the effects of changes in motoneuron pool and muscle spindle properties as well as muscle mechanical properties on the biomechanical behavior of the elbow joint observed during a constant angular velocity elbow extension. It is believed that this model can provide additional insights that could not be easily gained by previous models.

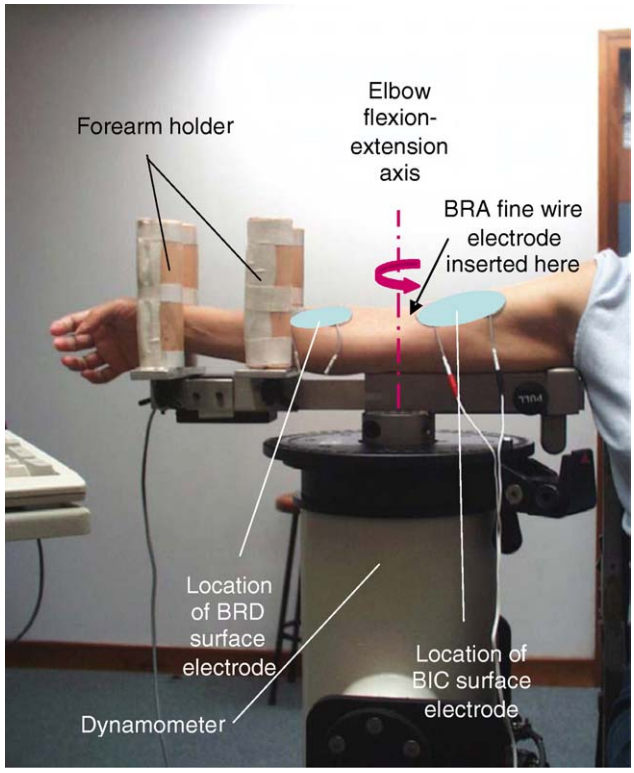

Fig. 1. The experimental setup.

2. Methods

To illustrate how the model can contribute towards better understanding of the underlying mechanisms of elbow spasticity, a male hemiparetic subject (age: 44 years; weight: 65.9 kg; Ashworth score [9]: 1+) was recruited and gone through a series of constant angular velocity elbow extension. The subject gave his informed consent to the investigation, according to the procedure approved by the Hong Kong Polytechnic University human ethics committee. The model was applied to mimics the stretch reflex response measured experimentally by varying a set of physiological-based parameters. Details of the testing protocol, model development, parameter estimation, and model simulation are described as follows.

2.1. Constant angular velocity elbow extension

Displacements of elbow angle were imposed using Cybex Norm Testing System (Computer Sports Medicine Inc., MA) at a constant angular velocity of 80° s^{-1} from 120° flexion to fully extended position (i.e., 0° flexion). During the test, the subject sat on an assessment chair with the trunk restrained by straps to minimize movement. The shoulder assumed 80–90° abduction and 0° flexion and the forearm was placed in a supinated position and fixed over a custom-made forearm holder so that the elbow flexion–extension axis was aligned with the motor axis (Fig. 1). The subject was asked to relax and not to intervene as the elbow was being extended by the servomotor. The torque measured during the extension can be regarded as a summation of the passive elastic torque (T_p), the passive viscous torque (T_v), the reflex torque (T_r), and the inertia torques due to the forearm holder and the limb (T_{in}), respectively.

$$\text{i.e., } T_m(\theta) = T_p(\theta) + T_v(\theta) + T_r(\theta) + T_{in}(\theta) \quad (1)$$

Since the servomotor accelerated and decelerated at the initial and final phases of the extension, respectively, only the range between 90° and 30° flexion can be regarded as constant angular velocity extension. During the constant angular velocity interval, the measured torque response would be independent of the inertia of the limb and the forearm holder. Therefore, the reflex torque response elicited by the elbow flexors over the range with constant angular velocity could be estimated by subtracting the passive elastic and viscous components from the measured torque. To estimate the passive elastic torque, a slow velocity extension (5° s^{-1}) was applied to the elbow joint through the testing range and the passive elastic stiffness was estimated by fitting a least squares linear regression equation to the torque–angle curve covering the angular range that was absence of reflex response as indicated by the EMG signals recorded simultaneously. This manipulation is possible because the passive elastic torque responses of the elbow joint are essentially linear and velocity independent [10]. $T_v(\theta)$ was estimated as summation of the viscous component of each prime elbow flexor (i.e., long and short heads of biceps (LHB, SHB), brachialis (BRA),