Neuromechanical Simulation in Berkeley Madonna

Camryn Lewis and Daniel P. Ferris

***Abstract*—Neuromechanics seeks to understand how muscles, motor pattern generators, and the brain interact to produce coordinated movement. To improve understanding on the neuromechanics of hand movements, a comprehensive musculoskeletal model is needed. The development of a mechanical model of rhythmic hand pronation and supination may serve as a tool for deeper understanding of the underlying mechanisms in motor symptoms during rhythmic upper extremity tasks. Control systems must face inevitable time delays that can complicate control. This model focuses in on the variability of muscle delay and can serve as the foundation for more complex models that characterize disordered motor movement. It was hypothesized that changes in muscle delay result in disrupted oscillations.**

I. Introduction

Supination describes the rotation of the wrist and forearm so that the palm of the hand is facing upward. Pronation indicates rotating so that the palm is facing downward. A joint with one degree of freedom connects the ulna and radius, enabling forearm pronation and supination [1]. Pronation to supination is one of the most important movements during rehabilitation exercises because it is critical for everyday activities [2]. This model attempts to simulate observations from experiments in which the subjects were asked to supinate/pronate and to turn the hand back to its original position after completion of the movement [3].

There are many conditions that may result is muscle delay, such as Parkinson's disease. Parkinson's disease is a neurodegenerative disorder that causes cognitive and motor deficits and affects over ten million people around the world [4]. People with Parkinson's disease tend to have slower reactions when compared with healthy individuals [5]. Thus, when programming and executing movements, a delayed reaction time can be expected. A variable that has been examined by previous studies when researching Parkinson's disease is tonic input [4]. Neurons receive tonic input from the brainstem.

Neuromechanical systems must face inevitable time delays that can complicate control [6]. Not only do they occur during the acquisition of sensory information, but also in the processing of said information for modulating motor output [6]. Due to the fact that biological systems have damping and inertial components, additional lag times arise from dynamic behavior [6]. To better understand the consequences of time delays for control of neuromechanical systems, a comprehensive musculoskeletal model is needed.

The Matsuoka Oscillator, a model of central pattern generators that is composed of two identical, spontaneously firing neurons [7], was essential to the framework of this study. Central pattern generators were described by Huang as spinal neural networks that produce rhythmic motor commands [8]. Past models have explored neuromechanical models for the functional range of motion during a pronation and supination task [4], but this model attempts to reduce the number of factors in order to finetune results. Therefore, this model can serve as the foundation for more complex models that characterize disordered motor movement.

II. Methods

The simulation utilized a modified Matsuoka Oscillator with the following equations:

$$X_1{}' = \begin{pmatrix} -X_1 - \beta V_1 - \omega Y_2 - H_1 MAX(O,0) \dots \\ -H_2 MAX(O,0) + C \end{pmatrix} \frac{1}{T_1} \quad (1)$$

$$X_2{}' = \begin{pmatrix} -X_2 - \beta V_2 - \omega Y_1 - H_1 MIN(O,0) \dots \\ -H_2 MIN(O,0) + C \end{pmatrix} \frac{1}{T_1} \quad (2)$$

$$T_2 = 3T_1 \quad (3)$$

$$V_1{}' = \left(Y_{1,delayed} - V_1\right) \frac{1}{T_2} \quad (4)$$

$$V_2{}' = \left(Y_{2,delayed} - V_2\right) \frac{1}{T_2} \quad (5)$$

$$Y_1 = MAX(X_1, 0) \quad (6)$$

$$Y_2 = MAX(X_2, 0) \quad (7)$$

$$Y = Y_1 - Y_2, \quad (8)$$

where X_1 and X_2 represent the average rates of discharge of a pair of mutually inhibitory pair of neurons, T_2 is the beginning of signal to neuron 2, V_1 and V_2 model the dynamics of self-inhibition, and remaining neural constants are defined in Table 1.

Additionally, the Matsuoka Oscillator was modified to include decreased muscle delay. Equations (6) and (7) were used to determine the magnitude of positive or negative neural signal based off the conditions described in (1), (2), (3), and (4). The Matsuoka Oscillator is illustrated in Fig. 1.

The authors are with the J. Crayton Pruitt Family Department of Biomedical Engineering, University of Florida, Gainesville, FL USA. Lewis (camrynlewis@ufl.edu) is corresponding author.

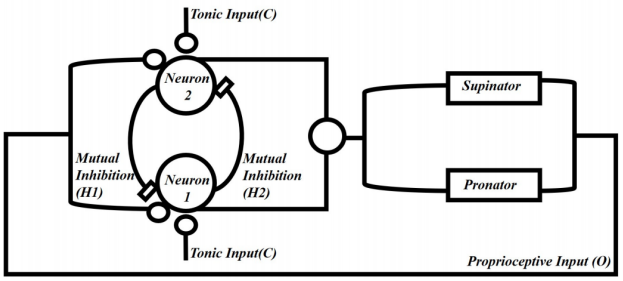

Figure 1. Adapted Matsuoka Oscillator. Acquired from [4].

TABLE I. NEURAL AND MUSCULOSKELETAL MODELING PARAMETERS

Constant	*Definition*	*Value*
β	Neuron Self-Inhibition (mV)	2.5
ω	Mutual Inhibition (mV)	2.5
H_1	Feeback Gain of Neuron 1 (mV)	30
H_2	Feeback Gain of Neuron 2 (mV)	30
T_1	Beginning of Signal to Neuron 1 (ms)	.6
C	Tonic Input (mV)	5.06
O	Proprioceptive Input (mV)	$\pm$1,0
I	Inertia (Kg cm^2)	14.7
G_P	Pronator Muscle Gain Ratio	5
G_S	Supinator Muscle Gain Ratio	5
R_P	Pronator Teres Moment Arm (cm)	-30
R_S	Supinator Moment Arm (cm)	0
D	Muscle Dampening Ratio	.136
k	Spring Constant (N/rad)	.4
Q	Starting Position (rad)	-.175
ρ	Reduction Ratio	.01

Conditional statements were inserted into the code in order to maintain realistic biological output during changes in position. If the position of the hand exceeded 1.725 radians, proprioceptive input was equal to 1 mV. If the position went below -1.725 radians, proprioceptive input was equal to -1 mV. Otherwise, proprioceptive input was set to 0. X_1,V_1, and V_2 were initialized to 0, and X_2 was initialized to 1.

The neural oscillator was coupled with two muscles using the following equations:

$$F_P = Y_{1,delayed} G_P \quad (9)$$

$$F_S = Y_{2,delayed} G_S \quad (10)$$

$$T_P = F_P R_P \quad (11)$$

$$T_S = F_S R_S \quad (12)$$

$$T = T_P - T_S \quad (13)$$

$$\theta'' = \left(\rho T \frac{1}{I}\right) - k(\theta - Q)^3 - (\theta' D), \quad (14)$$

where T_P and T_S were greater than 0 and θ represented the position of the hand relative to a neutral position, meaning that the thumb was pointing in a superior direction. Rather than multiplying by the muscle delay as previous works did [4], the delay function in Berkeley Madonna was used to create $Y_{1,delayed}$ and $Y_{2,delayed}$. Torque was calculated in (13) and angular acceleration was calculated in (14). The constants for the biomechanical equations are defined in Table 1. θ was initialized to 1.92 and θ' was initialized to 0, placing the starting simulated position of the hand at maximal supination. All simulations were modeled using Berkeley Madonna software with .01 millisecond increments for a duration of 500 ms.

III. RESULTS

The goal of this endeavor was to develop code that can simulate hand pronation and supination with varying muscle delays. Details of steps taken are provided in Methods. Below are the graphs generated by the Matsuoka oscillator simulation.

The extensor and flexor muscle torque values were plotted over time. As shown in Fig. 2, the flexor muscle torque greatly overpowered the extensor muscle torque, indicating the system was mainly relying on one muscle. Extensor and muscle torque peaks occurred at alternating time points.

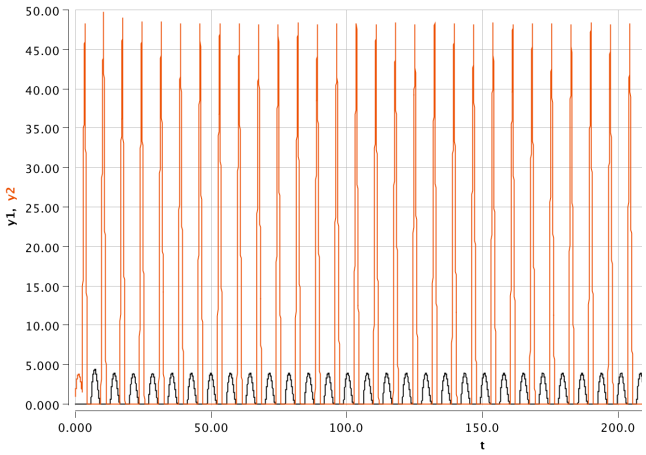

Figure 2. Graph of extensor muscle torque (y1) and flexor muscle torque (y2) produced by Matsuoka oscillator simulation vs. time in ms (t). Muscle delay of 1ms.

These muscle torques were then compared across different muscle delays through the examination of Fig. 2 and 3. Displayed in Fig. 3, the muscle delay was reduced to .9 ms. This reduction caused the flexor muscle torque to abruptly decrease just before 100 ms, while it caused little to no difference in extensor torque.

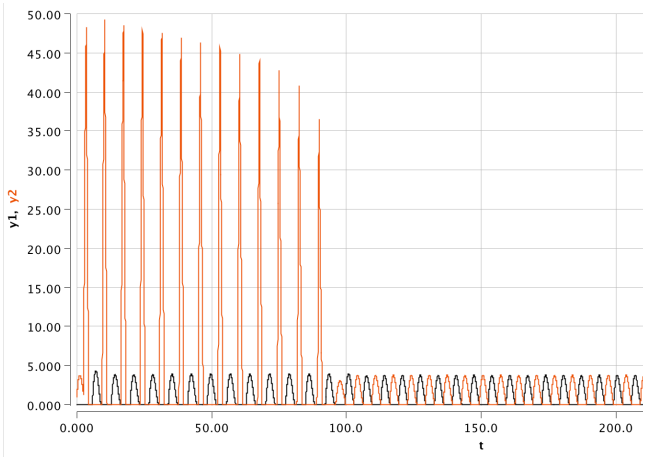

Figure 3. Graph of extensor muscle torque (y1) and flexor muscle torque (y2) produced by Matsuoka oscillator simulation vs. time in ms (t). Muscle delay of .9ms.

The physiologically realistic output of the hand position produced by the oscillatory movement is shown in Fig. 3. During hand pronation and supination, the position of the hand should have a range around 180°, or 3.14 radians. The position generated, represented by theta, goes up to around 1.3 and falls to -2, indicating the range would be around 3.3 radians.

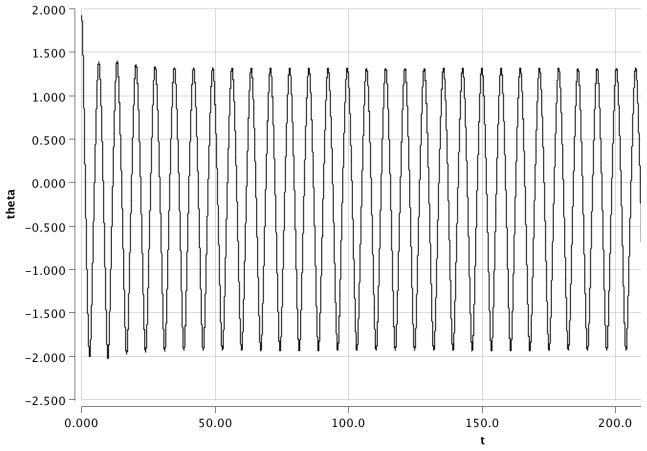

Figure 4. Graph of position (theta) of hand in radians vs. time in ms (t). Muscle delay of 1ms.

In comparison to the control condition (Fig. 4), the muscle delay of .9 ms was represented by disrupted oscillatory activity with decreased magnitude of supination and pronation signal (Fig. 5). The oscillations started off as expected, but then slowly started to decrease. At 100 ms, a decrease in amplitude and frequency occurred, followed by a sharper decrease in amplitue that then resulted in steady-state oscillations at a smaller amplitude.

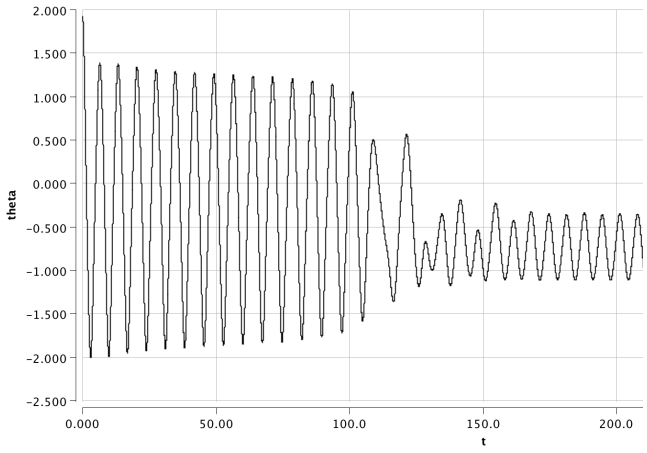

Figure 5. Graph of position (theta) of hand in radians vs. time in ms (t). Muscle delay of .9ms.

The final graph (Fig. 6) was generated using Parameter Plot to more easily visualize the changes in amplitude as a result of manipulated muscle delay. Spikes should be noted at .9 ms. This plot supports the results in the previous figures.

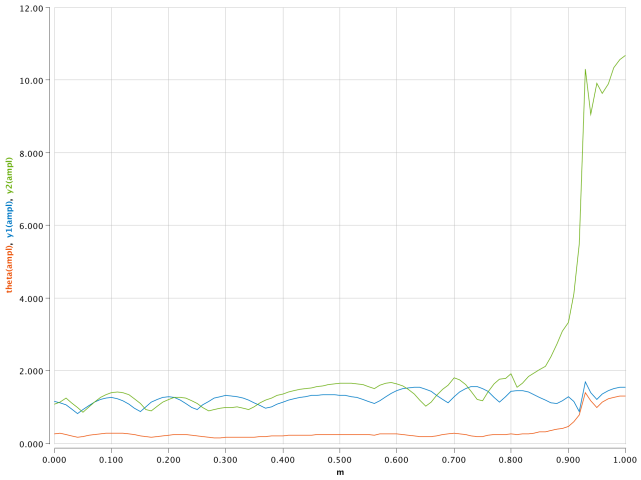

Figure 6. Parameter plot of the amplitude of theta (theta(amp)) in red, y1 (y1(amp)) in blue, and y2 (y2(amp)) in green vs. muscle delay in ms (m).

IV. Discussion

The graphs provided in the Results section demonstrate the functional differences between selecting a muscle delay of 1 ms or .9 ms. The position oscillations for a muscle delay of 1 ms maintained steady state (Fig. 4), as opposed to other simulations [4]. The result achieved in this study should be expected because the system is maintaining energy, not losing it.

Even though the position of the hand in Fig. 5 seems less than ideal due to changes in signal amplitude, this pattern is actually characteristic of the sporadic, slow, and weak neural communication that is observed in patients with conditions such as Parkinson's disease [4]. The occurrence of the decrease in Fig. 3 before the 100 ms mark aligns with research in which patients with Parkinson's disease showed a delay in onset of 66 $\pm$ 59 ms [5]. The introduction of irregular signal

can be followed by tremor [4]. Returning to the hypothesis, it was in fact shown that changes in muscle delay cause disruptions in oscillations of both flexor muscle torque (Fig. 3) and position (Fig. 5).

The values used in this study were chosen in order to increase physiological relevance. The average functional range of motion for the hands of healthy, young males is -1.92 to 1.73 radians [9], which was taken into account when initializing position and simulating proprioceptive input. A reduction ratio was added to (14) in order to reduce amplitude, but ideally this variable would not be necessary. A passive spring was added to (20) to represent the contributions of surrounding tendons. Muscle dampening refers to the internal stiffness of the muscle. Tonic input can be modeled with sin equations [4] but was left as a constant in this study in an attempt to simplify the model.

The modified Matsuoka oscillator's reliance on a central pattern generator for the creation of movement is what makes it ideal for this model [10]. The central pattern generator is capable of modeling movements in repetitive tasks. Using Berkeley Madonna allowed the constants to be manipulated easily through the addition of sliders. Incorporating torques also increased the similarity to actual kinematics.

It should be taken into consideration that this script was created for academic purposes, and further research into more advanced methodology should be conducted when studying neuromechanical simulation. Improvements to the code could include implementing moment arm equations to replace constants, equalizing the torque between flexor and extensor muscles (Fig. 2), and reducing the range of motion of the hand to fall within 3.14 radians (Fig. 4). Additionally, a larger time period could be examined to more accurately understand the effects of muscle delay. In the future, results should be compared to those of clinical research in order to quantitatively assess accuracy.

V. Conclusion

While the proposed neuromechanical model produced results that corresponded with previous studies [4], further validation is required. This model can serve as a basis for deeper research into upper extremity movement modeling.

References

[1] M. Mirakhorlo, N. Van Beek, M. Wesseling, H. Maas, H. E. J. Veeger, I. Jonkers, "A musculoskeletal model of the hand and wrist: definition and evaluation", *Taylor & Francis Online*, 2018.

[2] H. A. Rahman, Y. C. Fai, E. L. M. Su, "Analysis of Human Hand Kinematics: Forearm Pronation and Supination", *Journal of Medical Imaging and Health Informatics,* 2014.

[3] H. Teravainen, D. B. Calne, "Action tremor in Parkinson's disease", *Journal of Neurology, Neurosurgery, and Psychiatry*, 1980.

[4] L. Ziegelman, Y. Hu, M. E. Hernandez, "Neuromechanical Simulation of Hand Pronation and Supination Task in Parkinson's Disease," *IEEE,* 2018.

[5] K. A. Low, J. Miller, E. Vierck, "Response slowing in Parkinson's disease: A psychophysiological analysis of premotor and motor processes", *Brain*, 2002.

[6] K. Nishikawa, et al, "Neuromechanics: an integrative approach for understanding motor control". *Integrative and Comparative Biology*, 2007.

[7] K. Matsuoka, "Analysis of a neural oscillator", *Biological Cybernetics*, 2010.

[8] H. J. Huang, D. P. Ferris, "Computer simulations of neural mechanisms explaining upper and lower limb excitatory neural coupling", *Journal of NeuroEngineering and Rehabilitation*, 2010.

[9] National Aeronautics and Space Administration, "Anthropometry and Biomechanics," pp. 16–45, 2000.

[10] W. D. Byblow, J. J. Summers, G. N. Lewis, and J. Thomas, "Bimanual coordination in Parkinson's disease: Deficits in movement frequency, amplitude, and pattern switching," Mov. Disord., vol. 17, no. 1, pp. 20–29, Jan 2002.