CONTRALATERAL ACTIVATION AND ENHANCED RIGIDITY IN PARKINSON’S DISEASE: NEURAL AND NON-NEURAL CONTRIBUTIONS

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INTRODUCTION
Parkinsonian rigidity is described as a uniform increase in resistance to passive motion throughout the entire range of motion. Evidence indicates that rigidity is influenced by both non-neural and neural factors. The non-neural component of rigidity is the result of alterations in the mechanical properties of the muscle and connective tissues [1]. The neural mechanisms underlying rigidity include aberrant reflex responses to passive stretch and shortening. Previous research has revealed that a contralateral contraction enhances parkinsonian rigidity [2]. However, the changing contributions of the neural and non-neural components of rigidity in response to the contralateral contraction have not been previously investigated. The purpose of the current study was to compare the changes in neural and non-neural contributions to parkinsonian rigidity in response to contralateral activation. It was hypothesized that the contralateral contraction would result in greater contributions of the neural component of rigidity.

METHODS
Nine people with Parkinson’s disease (PD) participated in the current study. Inclusion criteria for PD subjects were presence of mild to moderate rigidity but minimal presence of tremor. Informed consent was obtained from each subject prior to participation in the current study. A custom-built apparatus generated passive wrist flexion and extension movements equal to 5° at 50°/s using a pseudorandom binary sequence (PRBS) waveform [3].

PD subjects were tested after a 12-hour withdrawal from dopaminergic medication (OFF-MED) and again one hour after administration of medication (ON-MED). The PRBS waveform was imposed on a relaxed wrist joint under two conditions: passive (PA) and contralateral active (CA). Joint position and torque were recorded using an encoder (100 Hz, Pacific Scientific, USA) and strain gauge (1000 Hz, Transducer Techniques, USA), respectively. Surface EMG (Delsys, Inc., MA, USA) was recorded from the flexor carpi ulnaris, flexor carpi radialis, flexor digitorum superficialis, extensor carpi ulnaris, extensor carpi radialis and extensor digitorum communis. EMG signals were band pass filtered (10-450 Hz) before being sampled at 1000 Hz. System Identification (SID) modeling approach was used to identify the intrinsic (non-neural) and reflex (neural) components of rigidity.

The SID was applied to separate overall rigidity into the neural and non-neural components for each subject under the PA and CA conditions in the OFF-MED and ON-MED states. An analysis of variance was used to determine the effects of the contralateral contraction and medication on the intrinsic and reflex components of rigidity in the PA and CA conditions under the OFF-MED and ON-MED states. Significance level was set at 0.05.

RESULTS
Neural and non-neural components of rigidity were enhanced with the contralateral contraction (neural: p=0.03; non-neural: p=0.01) in the OFF-MED state. In the ON-MED state, both components of rigidity were reduced in the presence of a contralateral contraction (neural: p=0.03; non-neural: p=0.04). Medication did not significantly reduce the neural and non-neural components of rigidity (p=0.17).

DISCUSSION AND CONCLUSIONS
Previous research has shown that a contralateral contraction increases rigidity in PD [2]. While the current data seem to be in conflict with previous findings, the mechanism previously suggested relied predominantly on input from the transcortical long-latency stretch reflex (LLSR). These data suggest the underlying mechanisms for the observed changes in rigidity may not be solely attributed to the LLSR.

REFERENCES