Meeting: 1002, Pittsburgh, Pennsylvania, SS 13A, Special Session on Mathematical Biology

1002-92-222 **Yixin Guo*** (yigst@math.ohio-state.edu), **Jonathan Rubin** (rubin@euler.math.pitt.edu) and **David Terman** (terman@math.ohio-state.edu). *Thalamicortical cell relay fidelity with various pattern of internal pallidum input.* Preliminary report.

Experimental studies in humans and animals with Parkinson's disease (PD) provid evidence that the increased activity, the firing patterns and the correlation of neurons within the internal segment of globus pallidus (GPi) are likely related to motor symptoms of PD. We use a computational model to study the fidelity of thalamicortical (TC) responses to sensorimotor signals with different patterns of GPi input. The model shows that both increased activity with more burst discharges and higher correlation among GPi neurons, characteristic of a parkinsonian state, jeopardize the capability of TC cells to respond to sensorimotor signals. As we reduce the burst activity and the correlation, the inhibition from GPi to TC becomes irregular and uncorrelated. The TC response is faithful to the signals, which corresponds to the normal state. If the burstiness in GPi neurons is increased to a significant level, the resulting GPi firing mimics the increased and regularized GPi activity during deep brain stimulation of either the subthalamic nucleus (STN) or the GPi. This tonic, high frequency inhibition from GPi restores the TC responsiveness to sensorimotor signals. The model effectively explains the contribution of various GPi firing patterns to parkinsonian, normal and DBS states. (Received September 14, 2004)