798.3

A COMPUTATIONAL MODEL OF THE BASAL GANGLIA AND HOW PALLIDOTOMY ALLEVIATES SYMPTOMS OF PARKINSON'S DISEASE.

G.S. Berns*[1] and T.J. Sejnowski[2]. [1]Western Psychiatric Institute & Clinic, Univ of Pittsburgh, Pittsburgh, PA 15213, [2]Howard Hughes Medical Institute, Salk Institute, La Jolla, CA 92186.

We propose a systems-level computational model of the basal ganglia based closely on known anatomy and physiology. First, we assume that the thalamic targets, which relay ascending information to cortical action and planning areas, are tonically inhibited by the basal ganglia. Second, we assume that the output stage of the basal ganglia, the internal segment of the globus pallidus, selects a single action from several competing actions via lateral interactions. Third, we propose that a form of local working memory exists in the form of reciprocal connections between the external globus pallidus and the subthalamic nucleus (STN), which could store information about sequences. The potential actions were represented as parallel processing streams of information, each competing for access to the cortical areas that implemented them. In the computational model, these actions were represented by units that corresponded to pools of neurons in each of the proposed processing streams. The model was comprised of three layers of units: a striatal layer (STR), a globus pallidus layer (GP), and a STN layer. Both the striatal layer and GP layer sent a convergent projection to a single dopamine unit, which computed the difference between these projections and represented an error signal, which was subsequently used to modulate connection strength changes between the STN and the GP layers. Low learning rates, which would be hypothesized to reflect low levels of dopamine, as in Parkinson's disease, led to slow sequence shifting when the model was trained first on one sequence and then switched to another sequence. However, this could be partially offset by modeling a lesion of the globus pallidus resulting in an increase in gain of the STN units. The gain increase is predicted based on a GP lesion narrowing the distribution of biases in the STN population.