A COMPUTATIONAL MODEL OF LOCAL MEMORY IN THE PRIMATE PALLIDAL-SUBTHALAMIC CIRCUIT.

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Previous models of the primate basal ganglia have postulated a dichotomy in flow of information: a "direct" pathway from the striatum to globus pallidus internus (GPi) and an "indirect" pathway from striatum to globus pallidus externus (GPe) to subthalamic nucleus (STN); however, recent tracer studies have revealed the existence of a more complex connectivity including a reciprocal STN-GPe pathway as well as a substantial GPe-GPi connection. In an earlier computational model, we demonstrated how the basal ganglia can learn action sequences, but this required short-term memory in the PFC. We now hypothesize that one function of the GPe-STN-GPe loop is to store short term local traces of activity in the basal ganglia. The loop was simulated on a computer using spiking GPe and STN neurons with an inhibitory GPe→STN synapse, an excitatory STN→GPe synapse, an inhibitory striatal input to the GPe, and an excitatory cortical input to the STN. Each neuron was modeled as an integrate-and-fire unit with spike afterhyperpolarization, post-inhibitory rebound, and psp's modeled by alpha functions. With a tonic cortical input to the STN, the circuit settled into a regular pattern of firing in both the STN and GPe cells. When perturbed from this state by transient striatal inhibition of the GPe, a postinhibitory rebound of activity in the GPe resulted in a transient inhibition of the STN followed by rebound activity, which then reexcited the GPe neuron. In this manner, the transient striatal inhibition led to an extended period of alternating quiescence and bursting in the GPe-STN loop. Because this loop is also connected to the GPi, the GPe-STN loop is ideally suited to storing activity traces which can be used in the production of action sequences. Lesions of the loop are predicted to impair the execution of previously learned sequences because of decreased local memory storage.