I-66. Identifying the neural initiation of a movement

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The preparation of voluntary movements takes time (Rosenbaum, 1980), suggesting that such actions depend on sequentially readying and then executing a motor plan. When these two phases are separated by a delay, neurons in the motor cortex often modulate their firing during both, indicating that the cortical circuits involved in the two computations overlap. Thus, to start the movement, the cortex must switch from a preparatory mode to an executory one. Is the moment of this switch evident in the neural firing? Reasoning that the switch should manifest as a change in computational dynamics, we fit a "hidden switching linear dynamical system" model (Petreska et al., NIPS, 2011) to multi-neuron data from macaque dorsal premotor (PMd) and primary motor (M1) cortices, recorded as the animal performed delayed reaches. Modelling the spike trains alone—without reference to external events—we could indeed see a trial-specific change in dynamics in both areas that appeared to anticipate initiation of arm movement. The change in M1 tended to follow that in PMd (mean shift = 36 ± 0.13 ms), and its estimated timing on each trial was more tightly correlated with the beginning of movement (cross-validated r2 = 0.70 ± 0.03) than was the estimated change in PMd (cross-validated r2 = 0.51 ± 0.06). It has been suggested that movements are triggered when population firing reaches a threshold. If so, then applying a similar threshold to the recorded population might better predict movement onset. In fact, no matter how we estimated or thresholded firing rates, this approach could explain no more than 0.37 ± 0.02 of the reaction-time variance. Thus, we conclude that the neural initiation of movement is better identified with a change in estimated circuit dynamics than with the crossing of a firing-rate threshold.

I-67. How does pacemaking in the globus pallidus affect striatal microcircuits?

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The basal ganglia (BG) are a collection of highly interconnected forebrain nuclei that network the cortex, thalamus and brainstem and are critically involved in action-selection and reward-based learning. The circuit structures underlying this functionality are poorly understood, but likely involve coordinated activity in cell assemblies across the BG. The globus pallidus (GP) is a hub of this network, containing GABAergic projection neurons that innervate all of the BG. Most GP neurons are spontaneously active yet their firing is desynchronized, providing independent inhibitory pacemaking throughout the BG. Here we use transgenic mice, optogenetics, and viral synaptic tracing to define the physiological properties and anatomical topography from a single class of genetically defined GP neurons. We focus on projection from the GP to the striatum, the major input nucleus of the BG. Recordings from acute brain slices demonstrate that GP neurons expressing the calcium binding protein parvalbumin are spontaneously active around gamma frequency and selectively innervate two types of striatal interneurons with different synaptic properties. Thus while inhibitory projections from the striatum heavily innervate the GP and are sufficient to pause GP pacemakers, these pacemakers indirectly affect striatal output by differential modulation of two types of local interneurons. Are these synaptic loops between the GP and striatum "closed" or "open"?