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Synchrony-asynchrony transitions in neuronal networks

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Irregular spike timing over long durations or relative asynchronous spiking between cells is a ubiquitous phenomenon observed in many brain nuclei both *in vivo* and *in vitro*. Subthalamic neurons (STN) and globus pallidus (GP) neurons of the basal ganglia are good examples of such neuronal irregularity. They fire asynchronously normally, but become synchronous in Parkinson's disease [1,2]. These cells have been shown to be autonomous, i.e., their oscillations are sustained in the absence of synaptic transmission. The prevalence of irregularity in such oscillatory neurons, and their tendency to become synchronous under disease conditions poses several challenges. They necessitate a detailed study of the interaction of oscillatory mechanisms that may contribute to synchronization between neurons with the sparsity of their interactions, the size of the network, and the external synaptic input. We study synchrony-asynchrony transitions in a model network of synaptically coupled STN or GP neurons with varying degrees of network size (N) and connectivity (number of presynaptic neurons per postsynaptic neuron, M <= N). We find the critical size (Ncrit) and critical number of connections (Mcrit for given N) needed in order to achieve global synchrony, as well as explore the nature and size of local cluster sates in the asynchronous state. The STN and GP neurons are modeled using Hodgkin-Huxley type of equations that incorporate sodium, potassium, calcium, a low-threshold calcium (T), and an afterhyperpolarization (AHP) activated potassium current [3]. In a network of two or more mutually excitatory neurons, for example, spike-to-spike synchrony can be achieved by increasing the coupling conductance beyond a critical level. In the unsynchronized regime, a phase drift between spike times of the neurons as well as multiple-frequency locked states can result. The level of mutual coupling required for synchrony in an all-to-all coupled network is found to increase as square root of N. In a sparsely connected network local cluster states emerge for weaker coupling, but spike-to-spike synchrony is also achieved for stronger coupling. Synchrony breaks down if the number of presynaptic neurons is fewer than a critical number. An external common synaptic inhibitory input given at Poisson intervals with a fixed arrival rate can cause asynchrony as well as enhance the frequency of the network at all network sizes.

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