

Abstract View

SLOW-WAVE SLEEP OSCILLATIONS IN COMPUTATIONAL MODELS OF DEAFFERENTED CORTEX

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When the brain falls asleep, tonic inputs from thalamic and cholinergic afferents are reduced resulting in a partial cortical deafferentation. A decrease in the release of acetylcholine during sleep hyperpolarizes pyramidal cells, enhances intracortical excitatory synapses and depresses afferent thalamocortical synapses. All these effects create a network with potentially very different properties from the awake cortex. Using Hodgkin-Huxley type network models we studied spontaneous and evoked activity in the sleep-like 'deafferented' cortex. In one-dimensional models, external stimulation resulted in waves of excitation propagating with constant velocity controlled by AMPA and NMDA conductances. The burst duration depended on short-term synaptic depression, NMDA conductance and I-KCa. When spontaneously firing cells were included, periodic bursting appeared in the network. In large-scale two-dimensional models, the network displayed transient or self-sustained oscillations in the form of traveling planar or spiral waves. Periodic activities in 1-D and 2-D network models were in the frequency range of slow-wave sleep rhythms, including cortical delta (1-4 Hz) and slow (<1 Hz) oscillations. Slow and delta cortical activities during sleep are similar in several aspects to those occurring during paroxysmal states, particularly in chronically isolated cortex. Discovering the cellular and network mechanisms underlying slow periodic activities in deafferented cortical networks may help explain the transformation of normal sleep oscillations to electrophysiological seizures. Supported by: NIH, HHMI, CIHR

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