Abstract View

MODEL FOR SLOW (2-3 HZ) NEOCORTICAL PAROXYSMAL OSCILLATIONS IN VIVO

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The oscillations observed in EEG and intracellular recordings during slow neocortical paroxysmal activity are larger than those during slow-wave sleep (SWS) activity as a consequence of increased synchrony of neuronal firing. The mechanisms underlying paroxysmal activity were investigated in computational network models of cortical neurons, which included voltage and Ca^{2+}-dependent currents. Intra- and extracellular K^+ and Na^+ concentrations were continuously updated based on the current flows, Na^+/K^+ pumps and glial buffering. A transition from SWS oscillations to 2-3 Hz seizure-like activity occurred in the model following increases in the extracellular K^+ concentration. The amplitudes of both hyperpolarizing and depolarizing membrane potentials were increased and neurons fired highly synchronously; in some cells depolarization led to spike inactivation lasting 50-100 ms. Enhanced activation of I_h in a small set of neurons was the main factor maintaining 2-3 Hz activity. Slow changes of K^+ and Na^+ reversal potentials reduced the frequency of paroxysmal oscillations during the first 3-5 sec after seizure onset and also decreased the hyperpolarizing potentials. In some cases, this led to fast oscillations in the frequency range 10-15 Hz, which were similar to the fast runs observed during seizures in vivo. These results suggest that modification of the intrinsic currents mediated by an increased concentration of extracellular K^+ can explain neocortical paroxysmal oscillations in vivo.

Supported by: NIH, HHMI, Human Frontier Science Program and the Canadian Institute of Health Research.

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