Abstract View

EXTRACELLULAR POTASSIUM DYNAMICS CONTROLS TRANSITIONS BETWEEN FAST AND SLOW SEIZURE-LIKE OSCILLATIONS

F.Frohlich[1,3*]; M.Bazhenov[1]; I.Timofeev[2]; M.Steriade[2]; T.J.Sejnowski[1,3]

1. Salk Inst., La Jolla, CA, USA
2. Dept of Anat. and Physiology, Laval Univ., Quebec, PQ, Canada
3. UCSD, La Jolla, CA, USA

Computational models of cortical networks were used to investigate the effects of extracellular K^+ dynamics on seizure-like synchronous oscillatory activity similar to that observed in anesthetized cats in vivo and during Lennox- Gastaut types of seizures in humans. Active and passive K^+ transport through the cell membrane, glial buffering, and diffusion in the extracellular space were included in the model. In a single cell, increase of $[K^+]_o$ caused slow (2-3 Hz) periodic bursting; progressive decrease of $[K^+]_o$ triggered a switch to fast (~10 Hz) oscillatory firing (fast run). The cell eventually returned to its resting potential. In a network of cortical excitatory and inhibitory neurons, overall network activity prevented $[K^+]_o$ from decaying to the steady-state level for the single neuron. The sustained increased level of $[K^+]_o$ caused a spatially inhomogeneous pattern of switching between epochs of slow bursting and fast runs, each lasting several seconds. During fast runs, increase of $[K^+]_o$ up to a threshold level triggered a switch to a slow bursting mode. During repetitive slow bursting, $[K^+]_o$ decreased until it reached a different level for transition to a fast run. Bifurcation analysis of single cell dynamics revealed a bistability leading to hysteresis between slow bursting and fast runs as a function of $[K^+]_o$. Increased $[K^+]_o$ together with synaptic drive stabilized the network in the bistable regime leading to spatially inhomogeneous patterns of slow bursting and fast runs. The existence of hysteresis between slow and fast oscillations in a single neuron may account for the switching between slow bursting and fast runs observed in cat neocortex in vivo.
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