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CORTICAL INPUT MAY ABOLISH SYNCHRONY IN A COMPUTER MODEL OF 2-4 Hz THALAMIC OSCILLATION. W. W. Lytton*, D. Contreras, A. Destexhe, T. J. Sejnowski, and M. Steriade, University of Wisconsin, Madison, WI; HHMI, The Salk Institute, La Jolla, CA; Laval University, Quebec City, Canada.

In vivo study of spike-wave (SW) complexes at 2-4 Hz shows that only a subset of thalamocortical cells (TC) fire spike-bursts in synchrony, while 60% of TCs undergo sustained hyperpolarization (Steriade and Contreras, this meeting). We performed computer simulations to determine conditions that would allow some TCs to oscillate while others are inactive. A pair of interacting neurons, consisting of a model TC and a model reticularis neuron (RE), oscillated in synchrony at 2 Hz, driven by the TC. A single simulated cortical shock disrupted this rhythm, resulting in an isolated RE intrinsic oscillation that hyperpolarized the TC continuously. The efficacy of this disruption depended primarily on the degree of activation of the RE, a function of the strength of the NMDA component of its cortical synaptic input. Alternatively, in the absence of cortical input, different parameters produced TC-leading synchrony that eventually disappeared as the RE asserted its intrinsic rhythmicity. With these parameters, the transient TC rhythm was abolished prematurely even with relatively weak cortical synaptic input. Rhythmic cortical stimulation was also effective in altering the oscillation frequency. Cortical driving at 4 Hz resulted in patterns where the TC followed on every other cycle or where the TC was driven in sets of about 10 bursts followed by a quiescent period. We suggest that variation in synaptic strength or intrinsic properties among TC cells could produce subpopulations of TC cells that would not participate in an apparently generalized SW process.

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