During stage two sleep, neuron networks in the thalamus and the cortex alternate between up and down states of activity. The pyramidal and interneuron network in the cortex generate a slow wave oscillation (<0.5 Hz) that propagates through the cortex to the thalamus where it synchronizes bursting activity in thalamicocortical and reticular neurons. Sleep is induced by synchronized, rhythmic bursting activity in the thalamicocortical network. Bursting occurs when cells receive an initial hyperpolarizing current followed by a strongly depolarizing current, and is driven by a calcium current. During sleep, reticular neurons receive a constant cholinergic input, which induces constant hyperpolarization in the cell population. The theta model, which represents an excitable system, can be imposed on a neuron model and used to present a possible explanation for the synchronizing effect associated with the cortical slow wave oscillation. The model presents a thalamicocortical relay cell and a reticular neuron that alternate up and down states when coupled and given a constant depolarizing current and a slow wave oscillation. The model shows that when the thalamocortical cell and a reticular neuron that alternate up and down states of activity. The pyramidal and reticular neurons had the same intrinsic currents as the TC neuron, excluding the hyperpolarization-activated current. The slow wave oscillation (0.5 Hz) generated by the cortical network was represented by a step function that alternated between an up and down states every second.

Baseline parameters were taken from Bazhenov 2002 and altered.

The Role of Calcium in Burst Firing

In awake states, neurons fire in a tonic mode. During sleep, neurons enter a burst firing mode, which is hypothesized to be less sensitive to incoming signals. This characteristic bursting pattern is generated by an infrequent calcium spike that causes a neuron to burst. In this hyperpolarized state, a sufficiently strong depolarizing current will cause the neuron to burst, which sends an excitatory pulse to the coupled thalamocortical cell. The coupled thalamocortical and reticular neurons is only a segment of the thalamocortical network, has not yet been achieved.

The Theta Model: Synchronization

Although not specifically a neuron model, the theta model represents an excitable system that can be imposed on neuron behavior. The model provides an analytic approach to understanding the synchronizing effect of the slow wave oscillation, and is represented by the following equation:

$$\frac{d}{dt}L = 1 - \cos (t) + (1 + \cos(t))(t)$$

Where \(t\) represents the angle in radians and \(L(t)\) is the input current function. The system has period \(2\pi\) and behaves as follows:

- \(t < 0\): The system is excitable when \(t < 0\) - a sufficiently strong perturbation will generate an action potential. When \(t > 0\), the system enters a limit cycle and oscillates for all time.
- \(t = 0\): Two neurons (N1 and N2) of the same species will have the same intrinsic currents \(I(t)\) and period. If an extrinsic slow wave oscillation is added, the neurons maintain equal periods with faster frequencies.

The full model consisted of thalamicocortical and reticular neurons from the thalamus connected to pyramidal cells and interneurons in the cortex (Bazhenov 2002). Neurons sent pulses to coupled neurons when they fired. Reticular cells sent inhibitory pulses, and the others were excitatory.

In this hyperpolarized state, a sufficiently strong depolarizing current will cause the cell to burst. The coupled thalamocortical and reticular neurons is only a segment of the thalamocortical network, has not yet been achieved.

Multiple pairs of coupled neurons would become synchronized and exhibit this precise behavior.

The coupled thalamocortical and reticular neurons is only a segment of the thalamocortical network implicated in sleep. Moving forward, we will create a sufficiently robust network of interconnected thalamocortical and reticular neurons that bursts in synchrony. The cortical network comprised of pyramidal cells and interneurons will also be modeled in order to organically generate the synchronizing slow wave oscillation. With the complete model, we will be able to manipulate input functions that simulate sensory information received by the brain during sleep. Hypotheses about an existing threshold value of sensory input after which an individual switches from bursting mode to tonic firing, and sleep as a bistable system will be addressed using the full model.

References