COMPARTMENTAL models of thalamic reticular (RE) neurons were investigated based on current-clamp and voltage-clamp data. Spontaneous oscillations in the model arise from the interaction between inhibitory synaptic currents and the rebound burst of RE cells. These oscillations critically depend on the level of the resting membrane potential. A network of RE neurons can be switched between silent and sustained oscillatory behavior by modulating a leak potassium current through neuromodulatory synapses. These results suggest that neuromodulators, such as noradrenaline, serotonin and glutamate, can exert a decisive control over the oscillatory activity of systems of RE cells. The model may explain why the isolated RE nucleus oscillates spontaneously in vivo but not in vitro.

Key words: Sleep; Noradrenaline; Serotonin; Glutamate metabotropic; GABA; Brain stem biophysical model; Spindle oscillations; Inhibitory networks; G-protein

Modeling the control of reticular thalamic oscillations by neuromodulators

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Introduction

The reticular thalamic nucleus of the thalamus has been shown spontaneously,¹²-¹⁴ in sharp contrast to the oscillations found in the isolated RE nucleus in vivo¹ and all models

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current, both based on current clamp data.\textsuperscript{5,6} \(I_{\text{Na}}, I_{k}\) are the fast Na\(^{+}\) and K\(^{+}\) currents responsible for the generation of action potentials (taken from Ref. 17). Details of the kinetics of these currents, based on estimates from physiological data, are given in Ref. 10.

\[
g_{\text{KL}} = \tilde{g}_{\text{KL}} m \quad \text{[4]}
\]

\[
\frac{dm}{dt} = \alpha [S] m - \beta (1 - m) \quad \text{[5]}
\]

where \(\tilde{g}_{\text{KL}} (= 1 \text{ nS})\) is the maximal leak conductance for K\(^{+}\) and \([S]\) represents the concentration of second...
Control of reticular thalamic oscillations

This behavior was extremely robust to changes in parameters such as the reversal potential of GABAergic currents ($E_{Cl}$), the values of the synaptic conductances or the amount of leak $K^+$ current affected by NE/5HT synapses. Typically, $E_{Cl}$ and the resting level were varied in a range of 5 mV around the present values; the simulations showed that sustained oscillations arose only if there was a sufficient 'driving force', of at least several millivolts (around 10–15 mV, depending on the maximal conductance of GABAergic synapses), between the resting membrane potential and $E_{Cl}$. If the membrane is hyperpolarized too close to $E_{Cl}$, the resulting shunting inhibition between RE cells prevents them from sustaining oscillations.
FIG. 2. Dependence of RE oscillatory behavior on the membrane potential. Simulation of a network of 100 RE cells interconnected with their neighbors through GABAergic synapses.