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The effect of feedback inhibition on throughput properties of the dorsal lateral geniculate nucleus

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Abstract

The effect of feedback inhibition from thalamic reticular cells on retinogeniculate transmission by thalamocortical neurons of the dorsal lateral geniculate nucleus is analyzed using a minimal integrate-and-fire-or-burst network model. Potassium leakage conductances control the neuromodulatory state of the network and eliminate rhythmic bursting in the presence of spontaneous input. During oscillatory full-field stimulation, feedback inhibition from thalamic reticular neurons leads to thalamocortical relay neuron burst responses. Depending on average input rate, contrast level, and temporal frequency of modulation, the response of the aroused network may or may not be phase-locked to the visual stimulus.

Keywords: Thalamus; Vision; dLGN; Integrate-and-fire; Burst

1. Introduction

Like other sensory thalamic nuclei, the dorsal lateral geniculate nucleus (dLGN) controls the flow of sensory information to cortex, acting as a state-dependent gateway between the sensory periphery and higher cortical centers [4]. During sleep the principle cells of the dLGN exhibit rhythmic bursts of action potentials (APs) which do not reflect the excitatory glutamatergic drive they receive from

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spontaneously active retinal ganglion cells (RGCs). These emergent network properties of the sleeping thalamus require the interaction between excitatory (glutamatergic) thalamocortical (TC) cells and inhibitory (GABAergic) thalamic reticular (RE) neurons, as well as the low-threshold Ca^{2+} current (I_T) that both TC and RE cells express. During arousal thalamocortical relay neurons of the dLGN cease rhythmic bursting, enter tonic mode, and respond with conventional APs that faithfully relay EPSPs received from spontaneously active or visually stimulated RGCs.

We present and analyze a minimal network model of retinogeniculate transmission by the dLGN that includes feedback inhibition from RE cells of the associated perigeniculate nucleus (PGN). We thus test the hypothesis that feedback inhibition from RE cells can significantly modify TC cell responses to visually driven RGC input. The dLGN/PGN network is constructed from TC and RE-like integrate-and-fire-or-burst (IFB) neuron models and is consequently more idealized than Hodgkin–Huxley-type models of thalamic oscillations and waves with [1] or without [3] AP generating currents. These simulations are novel in that the input/output properties—as opposed to autonomous network rhythms—of the dLGN/PGN model are characterized and, importantly, the sleep/awake transition occurs in the context of spontaneous retinal input.

2. Model

Simulations were performed using a minimal dLGN/PGN network model composed of TC-like and RE-like IFB neuron models [5]. Briefly, an IFB model is constructed by adding a slow variable to a classical integrate-and-fire neuron,

$$C\frac{\mathrm{d}V}{\mathrm{d}t} = -\underbrace{g_{\mathrm{KL}}(V-V_{\mathrm{KL}})}_{I_{\mathrm{KL}}} - \underbrace{g_{\mathrm{NL}}(V-V_{\mathrm{NL}})}_{I_{\mathrm{NL}}} - \underbrace{g_{\mathrm{T}}\Theta(V-V_h)h(V-V_{\mathrm{T}})}_{I_{\mathrm{T}}},$$

$$\frac{\mathrm{d}h}{\mathrm{d}t} = \begin{cases} -h/\tau_h^- & (V > V_h), \\ (1-h)/\tau_h^+ & (V < V_h), \end{cases}$$

where the leakage current $I_{\mathrm{L}} = I_{\mathrm{KL}} + I_{\mathrm{NL}}$ is the sum of potassium and non-specific components. A spike occurs when V reaches the threshold V_θ, and an absolute refractory period of length t_{R} is imposed during which $V = V_{\mathrm{reset}}$. The slow variable h represents de-inactivation of I_{T} and the Heaviside function $\Theta(V - V_h)$ is an idealization of I_{T} activation. The TC-like IFB model originally presented in [5] reproduces the salient response features of TC cells to sinusoidal current injection.

A subtle change in parameters converts the TC-like IFB model into an RE-like version (see Fig. 1A and B). In the TC model, the resting membrane potential ($V_{\mathrm{L}}^{\mathrm{TC}}$, *filled circle*) is more depolarized than V_h^{TC}(vertical branch of h-nullcline, *dotted line*), while in the RE model $V_{\mathrm{L}}^{\mathrm{RE}} < V_h^{\mathrm{RE}}$. Thus, the TC neuron exhibits tonic spiking when depolarized and post-inhibitory rebound bursts, while the resting RE model is primed to burst because I_{T} is de-inactivated ($h = 1$) when $V^{\mathrm{RE}} = V_{\mathrm{L}}^{\mathrm{RE}}$.