PERGAMON

Vision Research 38 (1998) 2007–2022

Vision Research

A model of cortically induced synchronization in the lateral geniculate nucleus of the cat: a role for low-threshold calcium channels

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Received 7 November 1996; received in revised form 14 July 1997

Abstract

Recently Sillito et al. (Nature 1994;369:479–82) discovered correlations in the spike trains of a relatively distant pair of cat lateral geniculate nucleus cells when simultaneously stimulated by a drifting grating; no such correlation occurs when the visual cortex is removed. In a further analysis of the data, we have found that short, high-frequency bursts contribute substantially to the synchronization and we hypothesize that the origin of the bursts is the low-threshold calcium spike. Guided by this hypothesis, our model of the corticogeniculate pathway and early visual system reproduces the experimental data in nearly every detail, as well as making predictions about cortical activity during the synchronizing process. We also discuss the possible behavioral relevance of correlations in the geniculo-cortical loop as well as other neural systems. © 1998 Elsevier Science Ltd. All rights reserved.

Keywords: Correlation; Feedback; Lgn; Corticofugal; Model

1. Introduction

The major pathway of visual information in the mammalian brain leads from the retina to the visual areas of the cerebral cortex via the lateral geniculate nucleus (lgn). The retina's function involves the transduction of photons to electrical activity as well as some preliminary signal processing, while the visual cortex is believed to be involved in perception and cognition of objects and their relationships. But the function of the lgn has remained somewhat mysterious despite a significant amount of research [1,2]; often the lgn has been thought of as a relay from the retina to the cortex.

However, anatomical and physiological studies give strong indications that the lgn's function is something more than a relay. Frequently, lgn cells appear to operate in one of two different modes: burst and tonic [3–5] and in the burst mode these cells do not simply relay retinal ganglion cell spikes to the cortex. Also, there are a significant number of extra-retinal inputs to the lgn, including the hypothalamus, visual cortex and some brainstem nuclei [3]; these inputs can potentially modulate the transmission of visual information to the cortex [6]. One of the most numerically impressive extra-retinal inputs is the visual cortex, comprising about 50% of the lgn synaptic input [3,7] and this corticogeniculate pathway is topographic [8,9].

While the general function of the corticogeniculate projection is believed to be the modulation of signal transmission through the lgn, a more specific understanding of its function has remained elusive. Early studies examining lgn responses in the cat after cooling or ablating the visual cortex produced mixed or inconclusive results [10–12]. Results from other species were also unclear [13,14]. Evidently, cortical effects on the lgn are not purely excitatory or inhibitory in all circumstances. Anatomical studies support this by showing that the cortex is potentially capable of both exciting and inhibiting the lgn, since corticogeniculate axons, which are most probably excitatory, make synaptic contacts on both projection cells and interneurons of the lgn [7,15], as well as with the perigeniculate nucleus [8,16], which in turn provides an inhibitory projection to the lgn [17,18]. This excitation/inhibition duality is also indicated in the electrophysiological study of Tsumoto et al. [19].

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PII: S0042-6989(97)00385-4

It is evident that the cortical input to the lgn is not as strong as the retinal input, since the center-surround receptive field (rf) structure of lgn cells is very similar to retinal ganglion cells, but is quite different from typical layer VI geniculate-projecting cortical cells, which have orientation-tuned rfs [20–22]. Comparison of the responses of lgn cells with their retinal ganglion cell inputs [23,6] also indicates the importance of the retina in driving lgn cells.

Several studies have implicated cortical feedback in a variety of functional aspects of the cat lgn, including length tuning [24], binocular effects [25] and rf center-surround interactions [26]. In another recent experiment, Sillito et al. [27] discovered a temporal interaction between pairs of (anesthetized) cat lgn cells that was dependent on an intact visual cortex. In this experiment, extracellular recordings were simultaneously made from two lgn cells while the cells were co-stimulated with a moving bar or drifting grating. The stimulus was aligned with the lgn rfs, with the rf centers separated by 1–4 visual degrees. Cross-correlation analysis was performed on the spike trains of the lgn cell pairs, with corrections made for the increased firing rates due to stimulation; in many cases, the cells showed significant correlation of their spike trains. No such correlations were found after the visual cortex was removed.

The synchronization of lgn cell responses can have a major influence on the impact of the lgn's output on the cortex, due to the temporal summation of post-synaptic potentials. Thus, the Sillito et al. [27] experiment demonstrates that the visual cortex can exert very important functional control over its geniculate input, despite evidence cited above that it generally does not exhibit strong excitatory or inhibitory influence on the lgn. How the cortex can achieve this is not obvious. In this paper we report a further analysis of the experimental data of Sillito et al. [27] and we present a model, based on our analysis, which employs low-threshold calcium channels and a dual excitatory/inhibitory action of the corticogeniculate pathway. Some of the work reported here has been previously presented in abstract form [28,29].

2. Methods

2.1. Data analysis

The tool that we used for analysis of both the experimental and simulation data is called the joint peristimulus time histogram (jpst). The jpst is like a 2-dimensional cross-correlogram, where the calculation is spread out over the duration of the stimulus response of the cells; using the jpst allows the possibility of detecting modulation of the correlation over the course of the stimulus response. A description of the jpst calculation and its properties can be found in Aertsen et al. [30] and Palm et al. [31]. In this subsection we will briefly summarize how the jpst is computed and interpreted.

Fig. 1a is an example of a jpst, corrected for the stimulus modulation of firing rates, computed using a pair of simultaneously recorded lgn cells from the data of Sillito et al. [27]. On the left-hand side is a matrix of bins, with one cell's peristimulus time histogram (psth) plotted below and the other cell's psth plotted on the left edge. The matrix is computed as follows: for each stimulus trial, the spike times for one cell are plotted along the bottom (the X-axis) and the other cell along the left edge (the Y-axis); the spikes times are measured relative to a stimulus marker that precedes the stimulus by a consistent time interval. Matrix bins are incremented such that they represent logical ANDs of the X and Y spike times; for instance, whenever cell X spikes at time j and cell Y spikes at time k, the bin that contains the point (j,k) is incremented. This process continues for every stimulus trial, gradually producing the psth for each cell along the bottom and left edge as well as the raw coincidence matrix. Unfortunately, the raw matrix generally contains a large number of coincidences simply because both cells were simultaneously stimulated; since these coincidences are not related to any interaction between the cells and are thus uninteresting to us, we correct for this factor by subtracting the bin-wise product of the two cells' psth from the raw matrix (this procedure is similar to the shift predictor correction for ordinary one dimensional cross-correlograms). This also eliminates the influence of the cells' firing rate on the correlation results. The matrix is then normalized by the following procedure: each matrix bin is divided by the product of the standard deviations of the corresponding psth bins, giving a range of -1 to 1 for each matrix bin; the bins are now correlation coefficients. See Aersten et al. [30] for details.

On the right-hand side of Fig. 1a is a diagonal histogram, called the 'coincidence histogram', which sums the strip of bins indicated by the bracket displayed on the top-right corner of the matrix. This permits detailed visualization of the correlation at some time delay, set by the bracket's position over the matrix, throughout the course of the stimulus response.

Finally, perpendicular to the coincidence histogram is another histogram which is the sum of the bins along each para-diagonal of the matrix (corrected for varying lengths of the para-diagonals). It conveys roughly the same information as the ordinary one-dimensional cross-correlogram.

We also use a statistical form of the jpst called the 'surprise' matrix [31]. An example is seen in Fig. 1b for the same data used for the jpst in Fig. 1a. The matrix in this calculation is computed by a statistical test on each