

Perceptual grouping in striate cortical networks mediated by synchronization and desynchronization

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Abstract

One advantage of cortical synchronization as a binding mechanism is its ability to account for phenomena such as perceptual grouping. Such a mechanism requires the ability to synchronize groups of cells and to desynchronize these groups from each other. We present a striate cortical model of perceptual grouping in which synchronization and desynchronization is carried out by a single, common mechanism. Cortical pyramidal cells and interneurons are simulated using multi-compartmental models. Cells in different orientation columns are inter-connected via two sets of long-distance connections that differ in axonal delays and spatial projections. The relative influence of these two connections determines whether synchronization or desynchronization occurs. Once one group of cells synchronizes, inputs from these cells facilitate synchronization in other orientation columns. We address the role of these synchronizing and desynchronizing connections in mediating perceptual grouping and metastable percepts.

Keywords: Synchronization; Striate cortex; Perceptual grouping; Compartmental models

1. Introduction

It has been proposed that neuronal synchronization in primary visual cortex may serve as the basis for the organization of elementary visual features into perceptual groups [7]. There are a number of theoretical advantages to the use of synchronization and spike timing in general. For instance, cells in primary visual cortex have relatively small receptive fields with a high degree of overlap, thus providing fine-grained visual resolution. Once the cells responding to different features of the same object synchronize, they are better able to drive cells in higher visual areas (with correspondingly larger receptive fields), which presumably carry out more complex processing tasks. More generally, use of precise spike timing provides higher information capacity and superior signal-to-noise properties for cellular and network computations [8].

One consequence of this synchronization-based framework is the requirement that groups of cells (corresponding to different perceptual objects) be synchronized independently so that they may be uniquely represented. A mechanism for “desynchronization” is thus required to distinguish cells responding to different perceptual groups. A number of models have achieved desynchronization through the use of a global inhibitory cell [12, 13]. Global inhibition requires a desynchronizing cell which has connections both to and from all the cells in striate cortex—a biological implausibility. We present a more biologically plausible desynchronizing mechanism that works in tandem with the synchronizing mechanism to allow different synchronized populations to emerge.

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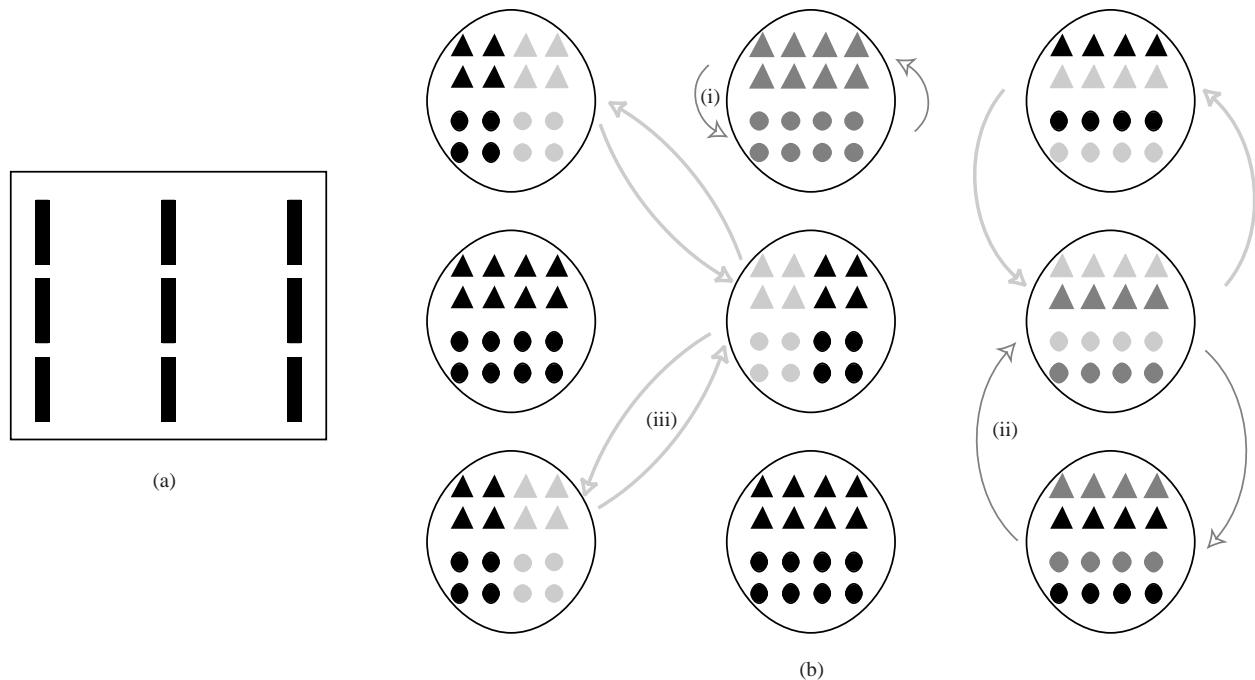


Fig. 1. Connections for network simulations. a) The figure on the left illustrates the stimulus configuration, made up of vertical bars arranged so that the dominant percept is for the bars to be grouped along the columns. b) the figure on the right illustrates the connections within the network. The connections labelled (i) connect all the cells in each group to one another, with no axonal delays. Half of the cells in each group make long-distance co-axial connections to half the cells in groups lying along the orientation axis. These connections labelled (ii) have axonal delays of 5 ms, and mediate the synchronization of the cells in a column. The oblique connections, labelled (iii), are formed between half of the cells in each group and half of the cells in groups that are not spatially aligned. These connections have axonal delays of 15 ms.

2. Model

To study the cortical mechanisms of synchronization and desynchronization, we implemented a compartmental model of cortical synchronization based on the mechanism proposed by Traub and colleagues [11]. In this model, synchronization is achieved through mutual inhibition of interneurons. Interneurons fire closely spaced doublet spikes, and synchronization is achieved when the timing of the doublet inter-spike interval matches the axonal delay between groups. In our simulations, each cortical orientation column is modeled as a group of 8 pyramidal cells and 8 interneurons. We used a slight modification of Traub's 66-compartment pyramidal cell [9] and his 51-compartment interneuron [10]. Both cells contain the following currents: fast sodium (I_{Na}), delayed rectifier ($I_{K_{DR}}$), calcium-dependent potassium ($I_{K_{Ca}}$), transient potassium (I_{K_A}), calcium-dependent after-hyperpolarizing potassium ($I_{K_{AHP}}$), and high-threshold calcium (I_{Ca}) currents. Within each orientation column, cells are densely inter-connected with excitatory (NMDA and AMPA) and inhibitory ($GABA_A$) synapses, with no axonal delay. Half of the cells in each column are also connected to cells of the same orientation in neighboring hypercolumns via long-distance horizontal connections (as shown in Figure 1b). The axonal delays of these connections are assumed to be a function of their spatial relationships as well as the separation between the orientation columns, and we assume that these inter-column connections fall into two classes. One set of connections extends roughly parallel to the axis of the orientation preference of the cell (co-axial connections) and has short axonal delays (roughly 5 ms). These reciprocal connections serve to synchronize the neuronal activity of cells that are spatially aligned with respect to each other. We have previously shown that these co-axial connections may underlie a number of physiological and psychophysical results on contour salience [14, 15]. A second set of connections extends obliquely with respect to the orientation axis of the cell, and has longer axonal delays (approximately 15 ms). Previous studies [1, 2] have shown that connections between neuronal oscillators with axonal delays of more than 5 ms are desynchronizing. These connections thus act to desynchronize cells that are not spatially aligned. The simulations were performed using Parallel Genesis [4] running on a SGI Origin 2000.

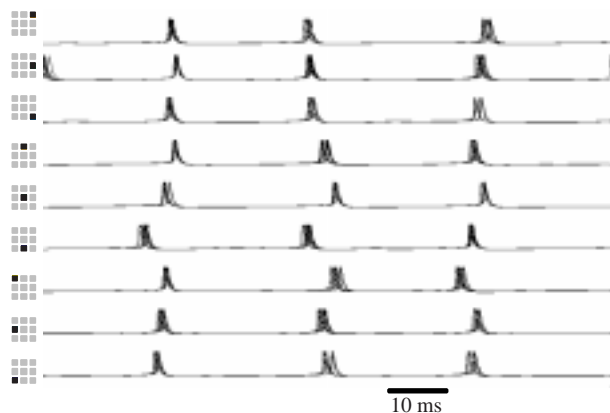


Fig. 2. Voltage traces of all pyramidal cells. The traces of the pyramidal cells in each group are plotted on top of each other. The position of the group in the stimulus array is shown in the left column. Current injection to the pyramidal cells is 1.6 ± 0.1 nA. In this simulation, there are no axonal delays in the intra-group connections, and the peak conductances are: AMPA - 8 nS, GABA_A - 1.5 nS, NMDA - 2 nS. The co-axial and oblique inter-group connections have 5 ms axonal delays, and peak conductances of: AMPA - 1.6 nS, GABA_A - 0.3 nS, NMDA - 0.4 nS. When the axonal delays of the co-axial and oblique connections are similar, the entire population synchronizes.

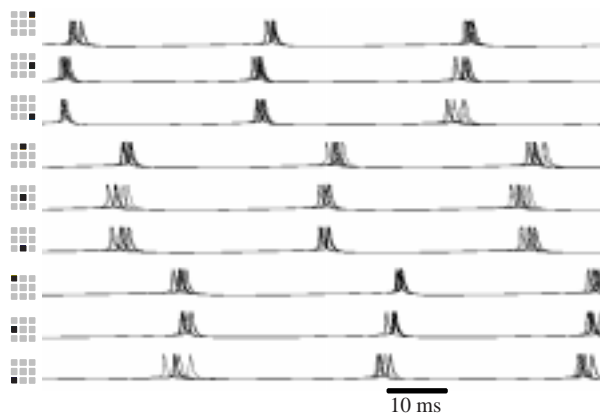


Fig. 3. Results of simulation with different axonal delays for co-axial and oblique connections. Current injection and the properties of the intra-group connections are the same as Figure 2. The co-axial inter-group connections have 5 ms axonal delays, and peak conductances of: AMPA - 8 nS, GABA_A - 1.5 nS, NMDA - 2 nS. The oblique connections have 15 ms axonal delays, and peak conductances of: AMPA - 1.5 nS, GABA_A - 0.3 nS, NMDA - 0.4 nS. The co-axial connections serve to synchronize cells in a column; once the cells in one column synchronize, the oblique connections desynchronize the cells in one column from the cells in other columns.

3. Results

The network was tested on a series of 3x3 arrays of vertically oriented bars as shown in Figure 1a. Networks of 9 groups of cells were connected as shown in Figure 1b. In the first experiment, the stimuli were arranged in a regular square array with equal spacing between all elements. Figure 2 shows the voltage traces of all the pyramidal cells from the simulation. In this case, the co-axial and oblique connections had the same axonal delays and all the cells become synchronized together, corresponding to a uniformly bound 3x3 square.

In the second experiment, the stimuli were grouped into 3 columns: the vertical inter-element spacing was smaller than the horizontal spacing. The results, in Figure 3, show that cells synchronize with other cells in the same column but are desynchronized from cells in other columns. This is consistent with our percept of 3 distinct columns, and illustrates the use of the oblique connections in providing local desynchronization.

We noticed that when the cells in a column became synchronized, the synchronous inputs provided by the oblique connections facilitate synchronization of cells in neighboring columns. In order to investigate this phenomenon, we removed the oblique connections, and reduced the connection weights of the co-axial connections to the point where the cells in each column were no longer synchronized. The results of the simulation are shown in Figure 4. We then added the oblique connections, and increased the co-axial connections for cells in only one of the columns so that they are now able to synchronize. The results are shown in Figure 5. The presence of the oblique connections allowed synchronization to propagate to the cells in the other columns. The cells are now once again, synchronized within the column and desynchronized between columns.

4. Discussion

Our results show that synchronization and desynchronization of cortical cells may be mediated by the same mechanism. In this model, co-axial connections have a shorter delay and thus allow cells to become synchronized. Oblique connections have a longer delay that desynchronizes a cell from populations in nearby orientation columns. Synchronous input from these oblique connections may enable post-synaptic populations to synchronize.

These results suggest a way for synchronization and *local* desynchronization to be obtained from very similar mechanisms. In addition, it is interesting that local desynchronization mechanisms might play a role in promoting and propagating synchronization. The use of separate co-axial and oblique connections is motivated by anatomical studies [6] and psychophysical studies, which demonstrate spatially specific interactions between stimuli [5]. However, the 5 ms and 15 ms axonal delays of the co-axial and oblique connections is arbitrary, and probably represents an unrealistic disparity for the range of visual angles used in the visual stimuli. In reality, perceptual

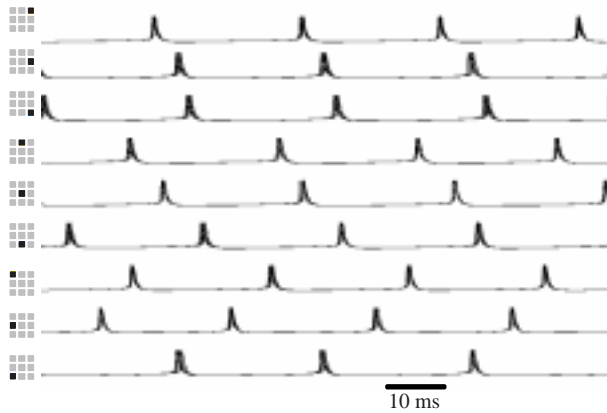


Fig. 4. Results of simulation with only weak co-axial connections. The current injection and the properties of the intra-group connections are the same as Figure 2. The co-axial inter-group connections have 5 ms axonal delays, and peak conductances of: AMPA - 0.4 nS, GABA_A - 0.75 nS, NMDA - 0.2 nS. The co-axial connections are too weak to allow the cells in each column to synchronize with each other.

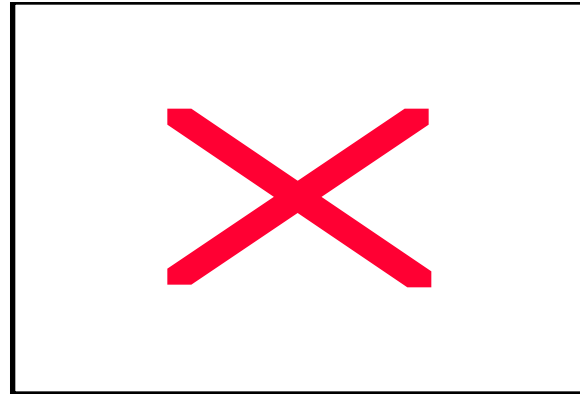


Fig. 5. Results of simulation with weak co-axial and oblique connections. The current injection and network connections are the same as Figure 4, with the exception that the co-axial connections of the groups in the right column have been increased to the levels in Figure 3. The oblique connections in this simulation have 15 ms axonal delays, and peak conductances of: AMPA - 0.8 nS, GABA_A - 0.15 nS, NMDA - 0.2 nS. The presence of the oblique connections allows the synchronization in one column to propagate to the other columns.

grouping depends upon the *relative* distance between elements. For example, in a 3x3 array, either columns or rows can be perceived depending upon the relative vertical and horizontal element spacing. A mechanism is therefore required to adjust the threshold for synchronization/desynchronization based on the actual distances involved. One possibility for such a mechanism might involve neuromodulators, which can alter both the connection strengths and synchronization properties of cortical cells. Another possibility might be to modulate the time constants of the cells in the orientation columns, which has been shown by [3], to influence the synchronizing or desynchronizing effect of delayed synaptic inputs. These mechanisms, as well as the effects of feedback connections on the ability to synchronize and desynchronize groups of cells, are directions for future research.

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