

CORTICAL SYNCHRONIZATION AND PERCEPTUAL SALIENCE

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ABSTRACT

We present a striate-cortical model which proposes a direct relationship between cellular synchronization and perceptual salience. The model focuses on the role of the long-range horizontal connections between oriented simple cells in striate cortex and is able to account for current physiological and psychophysical results on contour salience. We demonstrate that horizontal connections between realistically-modeled multi-compartment pyramidal cells and interneurons can generate robust context-dependent synchronization. Closed contours induce better synchronization in the network than open contours, and closure thus increases perceptual salience, as observed psychophysically by Kovács and Julesz. This result is a general topological property of synchronization. The model supports a temporal synchronization solution to the binding problem, in that changes in synchronization are directly linked to changes in visual perception.

INTRODUCTION

Recent theoretical and experimental studies suggest that temporal synchronization mechanisms may be involved in solving the binding problem¹. A missing link in these studies, is the demonstration that synchronization/de-synchronization affects perception, and that switching which sets of cells are synchronized results in a switch in percepts, and vice versa. We believe that a critical experiment in this regard has been performed by Kovács and Julesz² who showed that, in complex visual displays, closed contours have greater perceptual salience than open contours. The stimuli used by Kovács and Julesz consist of an array of Gabor patches placed at random positions and orientations, as shown in Figure 1. A subset of the Gabor patches are arranged so as to form either an open or

closed contour. They find that the salience of the contour is governed by \bar{d} , the average separation between the contour elements. At the threshold of detection, elements on a closed contour can be separated farther apart than those on an open contour. Most interestingly, at the threshold separation for open contours, d_{Open} , adding additional elements increases the salience of the contour monotonically. However, for elements on a closed contour separated by d_{Closed} , additional elements provide no increase in the salience until the last element is added and the contour becomes closed, at which point there is a large increase in salience.

We propose that the results of their experiment can be explained in terms of cortical synchronization in striate cortex, and that their results reflect a change in *perception* that accompanies changes in cortical synchronization.

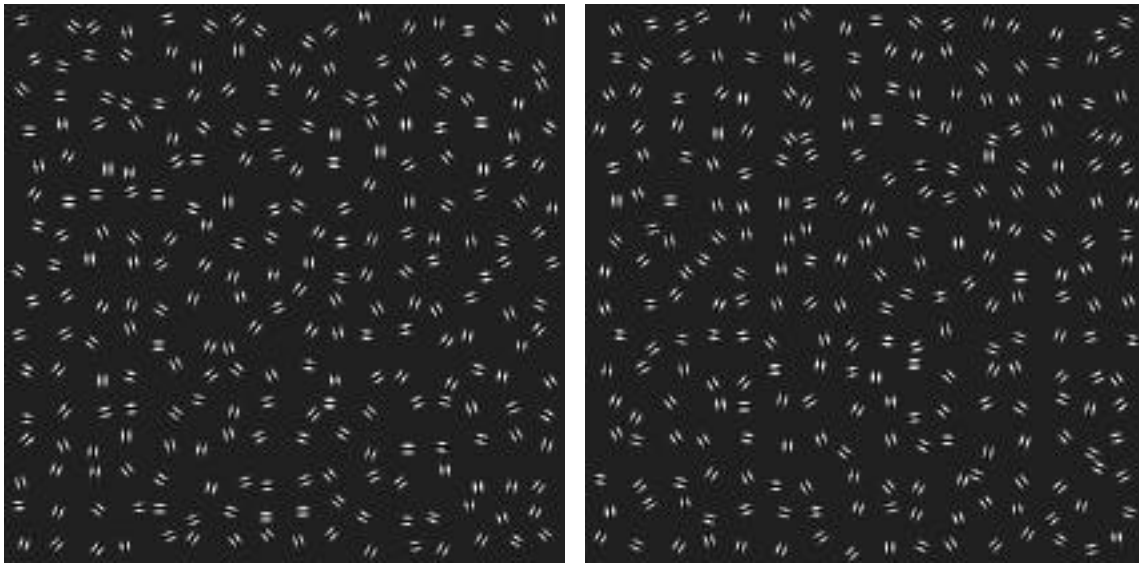


Figure 1. Stimuli used by Kovács and Julesz². The stimulus on the left contains an open contour made up of a subset of Gabor elements, while the stimulus on the right contains a closed contour.

MODEL

The model^{3, 4} consists of oriented simple cells, interconnected with long distance horizontal connections in a pattern similar to that suggested by Parent and Zucker⁵ and Field *et al.*⁶. The long-distance horizontal inputs mediate both facilitation and inhibition between cortical cells (depending upon their relative positions and orientation preferences), and cause cells lying along a smooth contour to become facilitated relative to the background elements. The facilitation mediates the transition of the cortical cells to a high-frequency bursting mode (as seen in the “chattering” cells described by Gray and McCormick⁷). In this bursting state, cortical cells can synchronize with other similarly bursting cells via the horizontal connections. The salience of the contour is then represented by the sum of synchronized activity.

Synchronization was studied using realistically-modeled, multi-compartment models of pyramidal cells and interneurons. The synchronization mechanism was adapted from Traub *et al.*⁸, in which coupled interneurons mediate the synchronization of pyramidal

cells*. We used Traub's 66-compartment pyramidal cell¹¹ and the 51-compartment interneuron of Traub and Miles¹². Both cells contained 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 afterhyperpolarizing potassium (I_{K_AHP}), and high-threshold calcium (I_{Ca}) currents.

Each orientation column stimulated by a Gabor element was modeled as a local group of 8 pyramidal cells and 8 interneurons (see Figure 2). Input to the pyramidal cells was modeled as a current injection of 1.6 ± 0.2 nA. Interneurons received a small (0.05 nA) hyperpolarizing current to suppress spontaneous firing. Pyramidal cells make both AMPA and NMDA mediated synapses onto apical and basal arbors of interneurons and other pyramidal cells. Interneurons make “fast” GABA_A-ergic synapses (decay constant of 10 ms) onto the perisomatic region of pyramidal cells and interneurons and slower “dendritic” GABA_A-ergic synapses (decay constant of 50 ms) on the apical arbor of pyramidal cells. The cells within a group had all-to-all connectivity with negligible synaptic delays and peak synaptic conductances that remained unchanged for all simulations. Each cell was also connected to half of the cells in each of its two neighboring groups, except the cells at the ends of the open contour, which had connections to only one neighboring group. These inter-group connections had axonal delays of 5 ms and peak AMPA, NMDA, and GABA_A conductances inversely proportional to the separation between the Gabor elements (this serves to simulate the decrease in the density of synaptic connections with increasing separation of the orientation columns). Simulations were carried out using the Parallel GENESIS¹³ simulation environment.

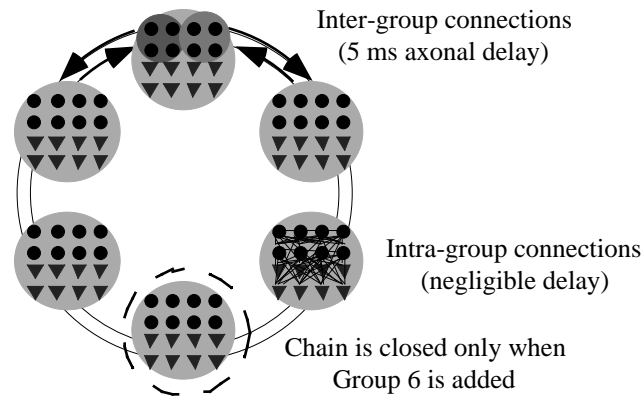


Figure 2. Connections within and between orientation columns. Each group in the figure represents cells in one orientation column. In this set of simulations, a closed chain is made up of six groups. Only nearest neighbor groups are connected; thus there are no connections between Group 1 and Group 5 in an open chain.

RESULTS

We were able to reproduce the synchronization reported in Traub *et al.*⁸ which depends upon the production of "doublet" spikes in the interneurons. The interneurons

*Similar mechanisms have also been proposed by Wang and Buzsaki⁹ and Bush and Sejnowski¹⁰.

control network synchronization when the time delay between groups matches the local, inter-doublet time delays within a group. The voltage traces of all the pyramidal cells in the simulation are shown in Figure 3. The cells within each group are tightly synchronized across all stimulus configurations, with an inter-burst interval of approximate 25 ms. The cells in each group are thus oscillating at 40 Hz and it is the inter-group connections that mediates the global synchrony across groups. When the Gabor elements form a closed contour, with the elements separated at d_{Open} , the pyramidal cells are tightly synchronized across all groups. This is due to the fact that the high peak conductances of the inter-group connections (AMPA: 8 nS, GABA_A: 1.5 nS, NMDA: 2 nS) contributes to the production of spike doublets in the interneurons, which are responsible for creating global synchrony. As the separation between the elements of the closed contour approaches d_{Closed} , the pyramidal cells are still synchronized across all groups, but not as tightly as before. The reduction in the peak conductances of the inter-group connections (AMPA: 0.8 nS, GABA_A: 0.15 nS, NMDA: 0.2 nS) reduces the frequency of the spike doublets, which reduces the global synchrony. When one of the contour elements is removed such that the contour is no longer closed, the inter-group connections to the groups on the ends are effectively reduced by half (since they now have connections to only one neighboring group). Spike doublets are rarely seen in the interneurons across the groups and the synchrony between the groups falls apart. The cells on the open contour are able to synchronize once again when the separation between the elements is reduced to d_{Open} such that the increase in the conductances of the inter-group connections is able to compensate for the loss in half the inter-group connections for the groups on the ends of the open contour.

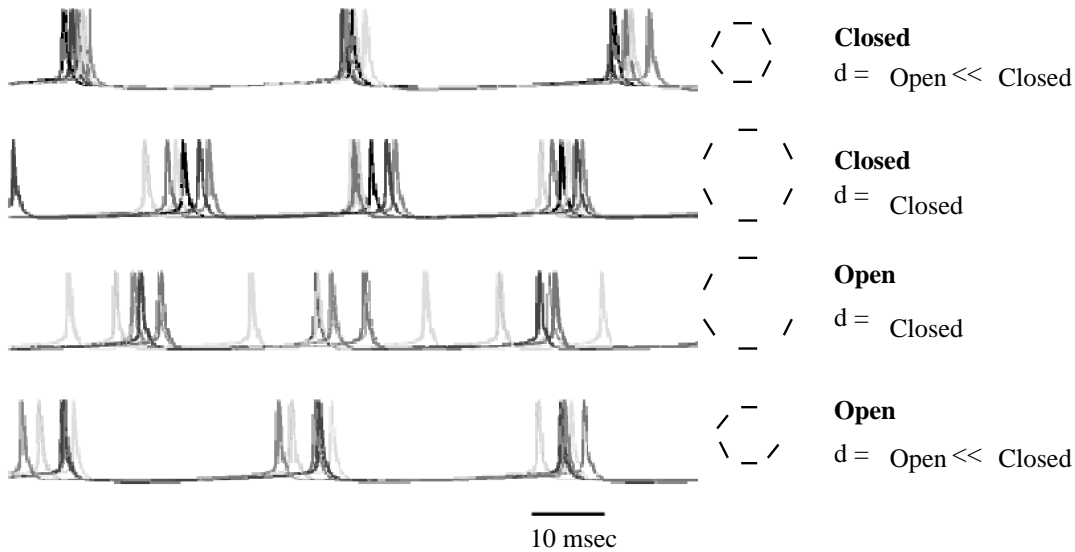


Figure 3. Synchronization using realistic, multi-compartment cells. The voltage traces of all the pyramidal cells (48 cells on closed chains, and 40 on open chains) in each simulation are plotted in the figure. Spikes from each group are shaded differently. The peak conductances of the intra-group and inter-group connections in the first and fourth traces were AMPA: 8 nS, GABA_A: 1.5 nS, NMDA: 2 nS. This corresponds to the strong connection strengths between groups when the contour elements are closely spaced (d_{Open}). In the second and third traces, the inter-group conductances were: AMPA: 0.8 nS, GABA_A: 0.15 nS, NMDA: 0.2 nS, while the intra-group conductances remained unchanged. This corresponds to widely-spaced contour elements (d_{Closed}).

We can therefore account for Kovács and Julesz's experiment as follows. Elements on closed contours may be separated further than elements on open contours ($\text{Closed} > \text{Open}$) because of the boundary conditions affecting the synchrony (and thus the salience) of cells on open and closed contours. At Open , the cells on the contour are synchronized; this means that each additional contour element adds to the sum of synchronized activity. The monotonic increase in salience of the open contour may thus be the perceptual correlate of this increase in synchronized activity. At Closed , the cells on the contour are not synchronized unless the contour is closed. This means that each additional contour element is not going to add to the sum of synchronized activity (and thus the salience), until the last element (which causes the contour to become closed) is added. At that point, all the cells on the contour are able to synchronize and the salience of the contour increases dramatically.

DISCUSSION

Temporal synchronization serves several roles in this model. As in previous models¹, synchronization serves as a means of distinguishing different contours in the scene. Synchronization also serves as the representation of the contour--a "contour" is perceived only when the orientation columns responding to individual contour elements are synchronized. Most importantly, we propose that the amount of synchronized activity correlates with the salience of the contour.

Changes in synchronization parallel changes in salience, and thus provide an explanation for the difference in salience between open and closed contours. This finding appears to be a general property of synchronization, independent of the exact mechanism. For example, we have previously demonstrated that the same result holds for synchronization of chains of phase-coupled oscillators³. Kopell and Ermentrout¹⁴ have proven analytically that synchronization in chains of coupled neural oscillators is enhanced when the chain is closed. Similar results also hold with relaxation oscillators¹⁵. In all cases, as a consequence of the boundary conditions, closure yields better synchronization.

The synchronization mechanism in the model depends upon the existence of long-range inhibitory connections in cortex¹⁶⁻¹⁸, which extend up to 10 mm. Conduction delays along these connections are less than 5 ms^{19, 20}. Even connections between cortical areas, or hemispheres have conduction delays within the required time window²¹. We predict that even longer delays can be consistent with synchronization if, instead of doublets, the local networks produce triplets or longer bursts.

An alternative explanation of the Kovács and Julesz result is to assume that salience is defined solely on the basis of activity. Cells on smooth contours may have higher activity due to recurrent facilitatory activity. Cells on closed contours may have higher activity due to the absence of weakly supported end elements. An increase in activity across a hypercolumn should be perceived as an increase in stimulus contrast. Thus, an activity-based mechanism might lead to confusions between the encoding of contrast and salience. In addition, several studies have shown that there is no change in apparent contrast for Gabor elements on a contour^{22, 23}. It remains possible that salience is encoded by a selective increase in the activity of a subpopulation of cells in the hypercolumn. Such a profile of enhanced activity might not generate a change in perceived contrast, particularly if contrast perception depends upon a population code.

In any activity-based model, the detection of the contour is presumably mediated by cells in higher cortical areas that are able to differentiate the enhanced activity of the facilitated cells in striate cortex. Although higher visual areas are undoubtedly involved in the perception of contours, a synchronization mechanism allows an implicit representation of contours to be maintained at the level of striate cortex, the area with the highest spatial resolution. Higher visual areas may modulate synchronization, perhaps via cholinergic effects on interneurons²⁴ and/or pyramidal cells⁷, thereby altering the salience of particular stimuli.

These two models predict opposite physiological results for cells whose receptive fields lie along a contour. An activity model predicts increased firing rates, at least in cells whose preferred orientation matches the local contour orientation. The synchronization model predicts little or no increase in activity, but a significant change in correlated firing.

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REFERENCES

1. W. Singer and C.M. Gray, Visual feature integration and the temporal correlation hypothesis, *Annu Rev Neurosci*, 18:555-86 (1995).
2. I. Kovács and B. Julesz, A closed curve is much more than an incomplete one: effect of closure in figure-ground segmentation, *Proc Natl Acad Sci U S A*, 90:7495-7 (1993).
3. S.-C. Yen and L.H. Finkel, Cortical synchronization mechanism for "pop-out" of salient image contours. In J. Bower (Ed.), *Computational Neuroscience: Trends in Research 1997*, Plenum, New York, 1997, pp. 553-560.
4. S.-C. Yen and L.H. Finkel, Extraction of perceptually salient contours by striate cortical networks, *Vision Research* (in press).
5. P. Parent and S.W. Zucker, Trace inference, curvature consistency, and curve detection, *IEEE Transactions on Pattern Analysis and Machine Intelligence*, 11:823-839 (1989).
6. D.J. Field, A. Hayes and R.F. Hess, Contour integration by the human visual system: evidence for a local "association field", *Vision Res*, 33:173-93 (1993).
7. C.M. Gray and D.A. McCormick, Chattering cells: superficial pyramidal neurons contributing to the generation of synchronous oscillations in the visual cortex, *Science*, 274:109-13 (1996).
8. R.D. Traub, M.A. Whittington, I.M. Stanford and J.G. Jefferys, A mechanism for generation of long-range synchronous fast oscillations in the cortex, *Nature*, 383:621-4 (1996).
9. X.J. Wang and G. Buzsáki, Gamma oscillation by synaptic inhibition in a hippocampal interneuronal network model, *Journal of Neuroscience*, 16:6402-13 (1996).
10. P. Bush and T. Sejnowski, Inhibition synchronizes sparsely connected cortical neurons within and between columns in realistic network models, *Journal of Computational Neuroscience*, 3:91-110 (1996).
11. R.D. Traub, J.G. Jefferys, R. Miles, M.A. Whittington and K. Toth, A branching dendritic model of a rodent CA3 pyramidal neurone, *J Physiol (Lond)*, 481:79-95 (1994).

- 12.R.D. Traub and R. Miles, Pyramidal cell-to-inhibitory cell spike transduction explicable by active dendritic conductances in inhibitory cell, *J Comput Neurosci*, 2:291-8 (1995).
- 13.N.H. Goddard and G. Hood, Large Scale Simulation with PGENESIS. In J. M. Bower and D. Beeman (Eds.), *The Book of GENESIS: Exploring Realistic Neural Models with the GEneral NEural SIMulation System*, Springer-Verlag, in press.
- 14.N. Kopell and G.B. Ermentrout, Symmetry and phaselocking in chains of weakly coupled oscillators, *Communications on Pure and Applied Mathematics*, 39:623-660 (1986).
- 15.D. Somers and N. Kopell, Rapid synchronization through fast threshold modulation, *Biological Cybernetics*, 68:393-407 (1993).
- 16.Z.F. Kisvarday and U.T. Eysel, Functional and structural topography of horizontal inhibitory connections in cat visual cortex, *European Journal of Neuroscience*, 5:1558-72 (1993).
- 17.C.T. McDonald and A. Burkhalter, Organization of long-range inhibitory connections with rat visual cortex, *Journal of Neuroscience*, 13:768-81 (1993).
- 18.C. Morin and S. Molotchnikoff, Influences of horizontal connections on visual responses in rabbit striate cortex, *European Journal of Neuroscience*, 6:1063-71 (1994).
- 19.A. Mason, A. Nicoll and K. Stratford, Synaptic transmission between individual pyramidal neurons of the rat visual cortex in vitro, *Journal of Neuroscience*, 11:72-84 (1991).
- 20.A.M. Thomson, D. Girdlestone and D.C. West, Voltage-dependent currents prolong single-axon postsynaptic potentials in layer III pyramidal neurons in rat neocortical slices, *Journal of Neurophysiology*, 60:1896-907 (1988).
- 21.M.E. McCourt, J. Thalluri and G.H. Henry, Properties of area 17/18 border neurons contributing to the visual transcallosal pathway in the cat, *Visual Neuroscience*, 5:83-98 (1990).
- 22.M.W. Pettet and P. Verghese, Enhanced contour detection is not mediated by apparent contrast. , *27th Annual Meeting of the Society for Neuroscience, Vol. 23*, New Orleans, LA, USA, 1997, pp. 176.
- 23.W.H. McIlhagga and K.T. Mullen, Contour integration with colour and luminance contrast, *Vision Research*, 36:1265-1279 (1996).
- 24.T.F. Freund and G. Buzsaki, Interneurons of the hippocampus, *Hippocampus*, 6:347-470 (1996).