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Early Cortical Orientation Selectivity: How Fast Inhibition Decodes the Order of Spike Latencies

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Abstract. Following a flashed stimulus, I show that a simple neurophysiological mechanism in the primary visual system can generate orientation selectivity based on the first incoming spikes. A biological model of the lateral geniculate nucleus generates an asynchronous wave of spikes, with the most strongly activated neurons firing first. Geniculate activation leads to both the direct excitation of a cortical pyramidal cell and disynaptic feed-forward inhibition. The mechanism provides automatic gain control, so the cortical neurons respond over a wide range of stimulus contrasts. It also demonstrates the biological plausibility of a new computationally efficient neural code: latency rank order coding.

Keywords: shunting inhibition, visual cortex, rank order coding, population coding, contrast invariance, single spike

1. Introduction

The question of how neurons encode and decode information is central in neuroscience (Perkel and Bullock, 1968). Almost all computational models of processing in the visual system start from the premise that neurons transmit information in the form of a firing rate code. Thus, the pattern of activity in the geniculate afferents reaching the visual cortex can be described as a set of continuous variables, one for each afferent, where the value corresponds to the neurons firing rate (Douglas et al., 1988; Ferster and Jagadeesh, 1992; Suarez et al., 1995; Carandini et al., 1997; Hirsch et al., 1998; Adorjan et al., 1999). However, an increasingly large number of studies have looked at aspects of activity in sensory neurons that cannot be described simply in terms of firing rate. In particular, there is now considerable evidence that the timing of the response to transient stimuli is considerably more precise than had previously been thought (Mainen and Sejnowski, 1995; Gawne et al., 1996; Reich et al., 1997; Buracas et al., 1998; Meister and Berry, 1999). This temporal precision opens up a whole range of computational possibilities that merit further attention.

One such alternative takes advantage of the fact that strongly activated neurons will tend to fire early, with the result that information about the stimulus can be obtained by looking at the latencies at which a population of cells fires (Thorpe, 1990). In this paper, I test the plausibility of using such latency differences as part of a mechanism for generating one of the most widely studied functional properties of cortical neurons, namely the orientation selectivity of neurons in primary visual cortex (V1). Orientation selectivity has been studied intensively for decades (Vidyasagar et al., 1996; Sompolinsky and Shapley, 1997; Anderson et al., 2000), but the underlying mechanisms are still controversial. Some authors starting with Hubel and Weisel (1962) have argued that the pattern of geniculate inputs is sufficient to explain basic orientation selectivity,

whereas others have argued that intracortical feedback is important (Suarez et al., 1995; Carandini and Ferster, 1997; Carandini et al., 1997; Adorjan et al., 1999). The spike time based mechanism proposed here allows these two apparently opposing views to be reconciled. Following a transient visual stimulus, a wave of spikes is generated in the lateral geniculate, the time of discharge of neurons being a function of the local contrast in the image. Electrophysiological studies actually report such waves of discharge for lateral geniculate neurons presented with bars at high contrast (Maunsell et al., 1999; Reinagel et al., 1999). This wave can produce orientation selective responses very rapidly in the primary visual cortex by using a combination of excitatory feed-forward connections and feed-forward inhibitory connections involving fast shunting inhibition processes demonstrated recently (Borg-Graham et al., 1998; Anderson et al., 2000; Ferster and Miller, 2000). The resulting mechanism is interesting for a number of reasons. First, its speed means that it is compatible with the timing constraints imposed by behavioral and neurophysiological data on the speed of processing in the visual system. Second, it provides a simple gain control mechanism that allows the system to respond in an orientation-selective manner over a wide range of stimuli contrasts. Obtaining contrast invariance for such early responses is a feature that cannot be generated using conventional techniques that involve feedback mechanisms.

2. Material and Methods

2.1. Cortical Neuron Characteristics

A single compartment model of a cortical cell from cat visual cortex was implemented in the NEURON simulation software system (Hines, 1989). Detailed modeling has shown that dendritic inputs tend to linearize at the soma (Jaffe and Carnevale, 1999), implying that the single compartment simplification is not unreasonable. The soma contained Hodgkin-Huxley type voltage dependent sodium and potassium channels ($g_{\text{Na}} = 180\ \text{mS/cm}^2$; $E_{\text{Na}} = 50\ \text{mV}$; $g_{\text{K}} = 30\ \text{mS/cm}^2$; $E_{\text{K}} = 90\ \text{mV}$). Values from various models (Suarez et al., 1995; Carandini et al., 1997; Destexhe, 1997; Mel et al., 1998) were adjusted to fit single compartment modeling (*surface* $= 15000\ \mu\text{m}^2$; $R_m = 4\ \text{k}\Omega\text{cm}^2$; $C_m = 1.0\ \mu\text{F/cm}^2$; $V_{\text{rest}} = -70\ \text{mV}$; the simulation file is available at www.sccn.ucsd.edu/~arno/model.html). Excitatory synapses from the lateral geniculate nuclei (LGN) cells included only fast AMPA-type synaptic conductances ($\tau_{\text{AMPA}} = 5$ ms; $E_{\text{rev-AMPA}} = 0$ mV). Inhibitory neurons in the primary visual cortex implemented fast GABA_A inhibition ($\tau_{\text{GABA}_A} = 10$ ms; $E_{\text{rev-GABA}_A} = -70$ mV). For both inhibitory and excitatory receptive fields, synaptic delays were ignored and post-synaptic potentials were modeled using instantaneous exponential decay processes.

2.2. Stimuli

Input images were adjusted to contain a 2-cycle period grating stimulating about 100 ON-center neurons and 100 OFF-center neurons. Each input image consisted of a 17×17 pixel array containing a stationary Gabor patch grating (8 bit gray level pixels) at a given orientation and contrast:

$$I(x, y) = C \sin(\text{Re}\{(x + iy)e^{i\theta}\}\phi)e^{\frac{x^2+y^2}{2\pi\sigma^2}}$$

where $I(x, y)$ is the pixel value (between -1 and 1) at position (x, y), x and y being integer between -8 and 8 relative to the ON or OFF neuron location ($\phi = 0.36$ radians and $\sigma = 2.3$ pixels). Orientation θ in radian was varied by 5° steps over the full 360° range and contrast C was varied from 1.6 to 100%. An array of 11×11 ON-center cells values—excluding borders of the initial image—was computed by a direct application of a 7×7 difference of gaussian contrast filter on the image. The standard deviation of the central positive gaussian was 0.63 pixels (surround 1.9 pixels), and the amplitude of the surround negative gaussian was divided by 3 with respect to the central region. The array of OFF-center cells was calculated using the same filter on the negative image with inverted pixel values.

2.3. Input from the LGN

Gabor patch gratings at various orientation and contrast were presented to an array of ON-center and OFF-center cells modeling the LGN neurons. These cells were modeled as noisy leaky integrate-and-fire neurons, whose inputs corresponded to the local contrast in the image as depicted in the previous paragraph. The earliest latencies correspond to those cells for which the value of contrast is the highest (positive for ON-center cells and negative for OFF-center cells), whereas lower activation levels resulted in progressively longer latencies (Fig. 1). The noise added to the LGN neurons

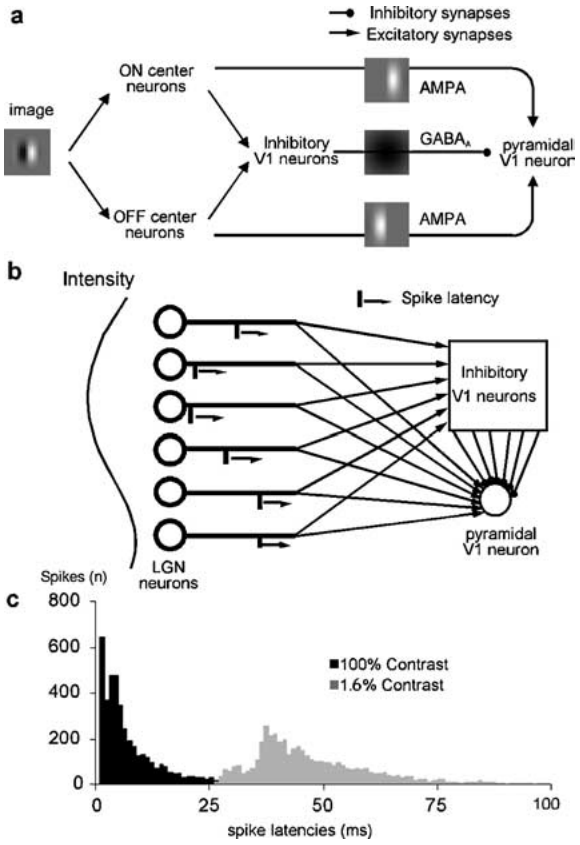

Figure 1. Model architecture and dynamics. (a) Transformation of an input image into neuronal discharges by arrays of ON-center and OFF-center neurons and connectivity of these neurons with a V1 pyramidal neuron. Synaptic weights are depicted in gray scale; mean gray values standing for 0, brightest ones for excitatory AMPA synapses and dark ones for inhibitory $GABA_A$ synapses. (b) Dynamic of neuronal activation. In the LGN, the latency of discharges of neurons depends on the local intensity of activation. For the V1 neuron to discharge, highest synaptic weights must be activated first, before the inhibition kicks in. (c) Latency of spikes of ON-center neurons pooled for all 72 grating at 100% and 1.6% contrast (2 ms time bin).

corresponded to a 3.6 ms standard deviation of spike timing at the highest contrast and 12.6 ms at the lowest contrast (corresponding to a signal to noise ratio of 1 at the lowest contrast). Averages spike latencies were 9 ms at the highest contrast and 47.3 ms at the lowest contrast. These values appear reasonable with respect to neurophysiological studies (Reich et al., 1997; Maunsell et al., 1999). The latencies of LGN neurons' spikes were generated with the neural network simulator SpikeNET with a membrane time constant of 20 ms (Delorme et al., 1999). For a given stimulus and at each precise location, either an ON or an OFF-center cell fires one spike, thus ruling out the possibility of using a conventional rate code based on inter-spike interval.

2.4. *Connectivity*

Each of the 242 LGN neurons—11×11 ON-center and 11×11 OFF-center cells—forms a single excitatory synapse onto the cortical cell's soma. Synaptic weights were set according to a Gabor function that makes the neuron selective to contours oriented at 0° (Fig. 1).

$$W_i = \left| \sin(A \cdot d(r_i, n)) e^{\frac{-d(r_i,n)^2}{2\sigma^2}} \right|^+ + B$$

where W_i is the synaptic weight between ON-center neuron r_i in the LGN and the unique neuron n in V1, and $d(r_j, n)$ represents the Cartesian distance in pixels separating the two neurons ($A = 0.75$; $B = 0.1$; $\sigma = 2.5$). The B parameter was added to ensure that the selectivity of the neuron could not simply be explained by the activation of sub-populations of synapses. Electrophysiological studies explicitly show that the absence of discharge for non-optimal orientations is not simply due to the withdrawal of excitation (Hirsch et al., 1998; Anderson et al., 2000). Parameter B contributes to about half the sum of weights. OFF-center cells make connections with the V1 neuron using the same function but with a phase shift of 180°.

Inhibitory interneurons were not explicitly modeled. For simplicity, I supposed that the LGN cells trigger an inhibitory neuron at the same location sufficiently rapidly so the onset of the inhibitory post-synaptic potentials (IPSP) coincided with the onset of excitatory post-synaptic potentials (EPSP). As I will discuss later, this would be the case if the intradendritic propagation of EPSP compensated for IPSP time lag. The amount of inhibition slowly decreased with the distance separating the input LGN neuron to the cortical neuron. Synapses were modeled using fast $GABA_A$ synapses but preliminary studies show that the addition of slow $GABA_B$ synapses does not modify the model's behavior.

3. Results

For a given amount of inhibition, excitatory synaptic weights afferent to the unique cortical neuron were adjusted such that at 100% of contrast the neuron discharged over a 65° orientation range centered on the

preferred orientation (here 0°). Even without inhibition, some orientation selectivity will be present: only at the preferred orientation will the high amplitude EPSPs all arrive within a sufficiently short period to trigger a spike. This period depends on the duration the membrane time constant ($\tau = 9.5$ ms) in our case and by setting the spike threshold at a suitable value, the cortical neuron can be made orientation selective. However, the neuron's selectivity is not robust against a drop in stimulus contrast since the increased latency spread of the incoming spikes prevents the neuron from reaching its threshold (Fig. 2, black area).

In contrast, with feed-forward inhibition, the neuron can cope with a wide range of different contrasts. This can be seen from the other curves in Fig. 2, which show that, as the total inhibition conductance increases, the neuron becomes more and more robust to contrast changes: for the condition of highest inhibition, the neuron's orientation selectivity can remains roughly constant down to contrasts as low as 3%. Note that there is a linear relationship between the sum of inhibitory conductances and the sum of excitatory conductances (linear fit: $R = 0.9996$) so the balance between excitation and inhibition is similar in every condition. The neuron fails to respond at 1.6% of residual contrast irrespective of the amount of inhibition: 1.6% residual contrast would correspond to a standard deviation between spike latencies of 12.6 ms. Since the excitatory AMPA time constant is 5 ms, the inhibition mechanism for contrast invariance breaks down because EPSPs become independent.

The selectivity of the neuron depends mainly on the timing of the excitatory inputs relative to the rapidly increasing inhibition. If strong excitatory inputs are triggered fast enough, the neuron can reach its threshold before significant inhibition kicks in (Fig. 3). On the other hand, if the early activated excitatory synapses are relatively weak, the relative contributions of excitation and inhibition will be biased in favor of inhibition and the neuron will not fire. This property was preserved using a 1-ms delay for the inhibitory synapses but not using a 2-ms delay (data not shown). However, as pointed out in the Methods section, the effect of excitation on the soma is delayed by intracellular potential propagation. The issue of the relative timing between excitation and inhibition will require detailed simulations of the V1 neuron geometry.

As illustrated in Fig. 4, the neuron's spike latency is shortest for its preferred orientation. This means that when a population of orientation-selective neurons is presented with a grating stimulus, the optimally activated neuron will tend to respond faster than the other ones. Thus, the model V1 neuron is orientation selective in terms of spike latency. As shown in Fig. 4, the latency of the first spike was shortest when the orientation of the stimulus matched the neurons preferred orientation. Response latency increased when stimulus orientation was moved away from the preferred value, and the neuron did not discharge at all for orientations beyond a certain value. Neurophysiological studies have

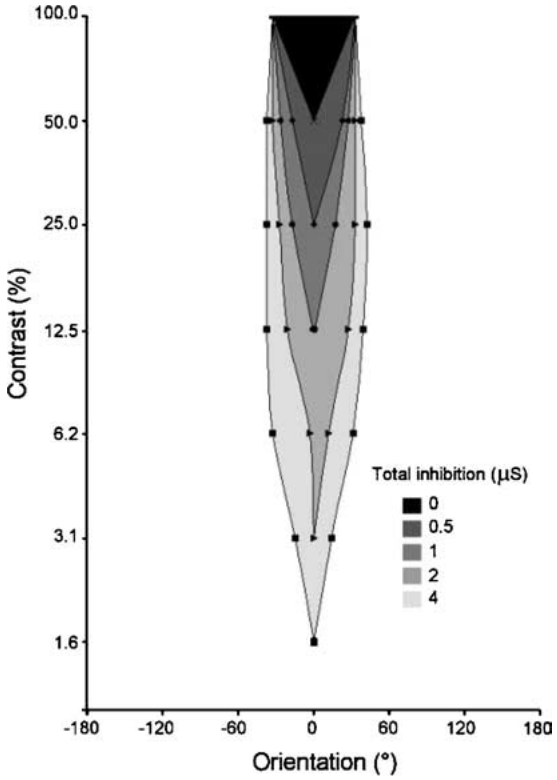

Figure 2. Range of orientations to which the neuron discharged as a function of contrast and for different degrees of shunting inhibition (depicted by different gray levels). The figures represent the sum of all the inhibitory conductances in μS. The strength of the excitatory input was adjusted so that the neuron responded over a range of 65° at 100% contrast. Each colored surface represents the range of discharge of the output neuron in the contrast-orientation 2-D space for a given level of inhibition. The limit was defined by the range of orientations to which the cell responded on at least 5 of the 10 trials when noise was added. The standard deviations at the borders are always less than 5° (not shown). With no inhibition (black surface), the neuron no longer responded when contrast was reduced to 50%. In contrast, with increasing shunting inhibition, the neuron could remain selective even when stimuli were presented at very low contrasts. Thus the presence of fast shunting inhibition allows the neuron to keep responding even at low contrasts.

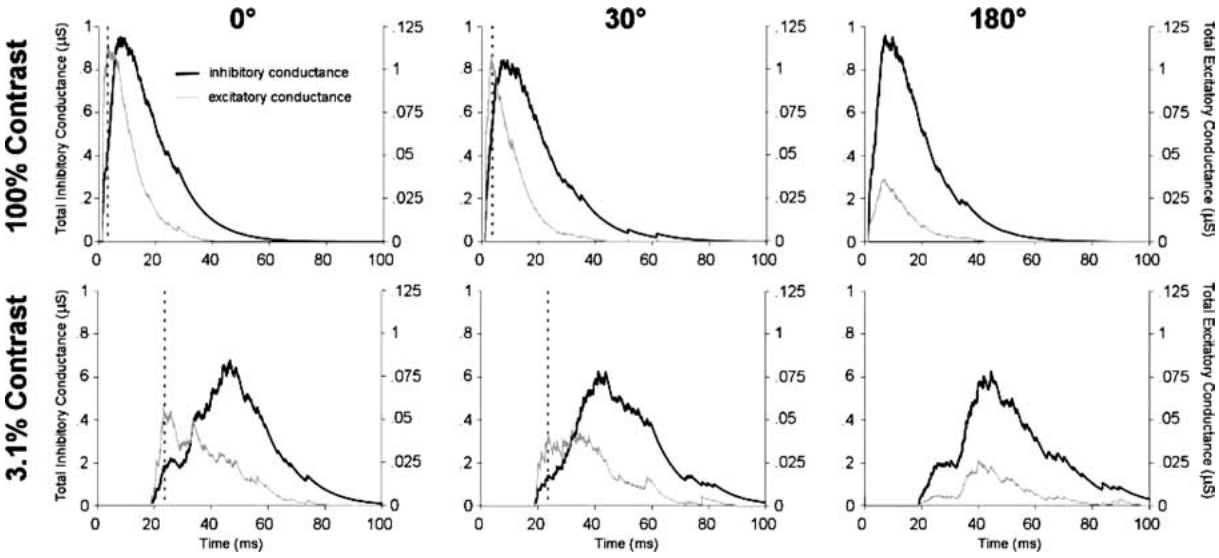

Figure 3. Total excitatory (light gray curves) and inhibitory (dark gray curves) post-synaptic conductances of the model of V1 neuron calculated for 3 orientations, 0°, 30° and 180° and 2 levels of contrast. Dotted lines if present indicate the latency of the neuron's discharges. The inhibitory conductance is constant for a given contrast and it is the dynamic of the excitation that determine the neuron's behavior. The neuron spikes for the 0° and 30° oriented grating but not for the 180° one. The latency of discharge is faster at 0° than at 30° which indicate that the latency code can be used at the next stage of processing. The total inhibitory conductance was 4 μS and the transient increase in shutting inhibition is compatible with electrophysiological recordings (see text for details).

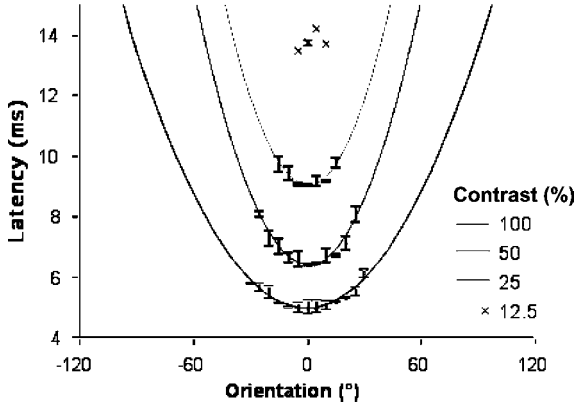

Figure 4. Spike latency of the first spike of the V1 neuron model as a function of orientation for 4 different degrees of contrast and inhibition fixed to an intermediate value (sum of 1 μS). The Standard deviation of spikes' timing was computed using 10 different initial conditions. The data were fitted with curves based on second-degree polynomial functions. For all contrast conditions, the latency of discharge of the neuron is a function of the orientation of the stimulus: earliest latencies corresponded to the preferred orientation. This behavior is in agreement with electrophysiological recordings.

shown that real neurons can behave very similarly in primate V1. Both Celebrini et al. (1993) and Gawne et al. (1996) observed increases in latency between the preferred orientation and one shifted by 30°. As in our model, for larger orientation shifts, they did not observe any responses. Thus, for the next processing stage, the information can again be encoded in the pattern of spike latency across the neuronal population.

Additional simulations showed that inhibitory currents did not have to be proportional to the output neuron potential. In these simulations, inhibitory synapses injected simple exponentially decreasing current pulses ($\tau = 5$ ms with current proportional to synaptic weight). In these conditions, the output neuron still responded to a wide range of contrast (not shown).

I also tested whether the model required the precise pairing of individual excitatory and inhibitory spikes by randomly suppressing inhibitory synapses. The output neuron behavior remained qualitatively unchanged when up to 70% of the inhibitory synapses were randomly suppressed (Fig. 5). Note however that, because of the random suppression of synapses, the neuron latency orientation tuning curve became discontinuous and asymmetric with respect to the neuron's preferred orientation. This effect was even more pronounced when 90% of the inhibitory synapses were suppressed (not shown).

In the previous models, each neuron discharged only once. To address the issue of multiple spikes, after the first spike, each input neuron was allowed to discharge

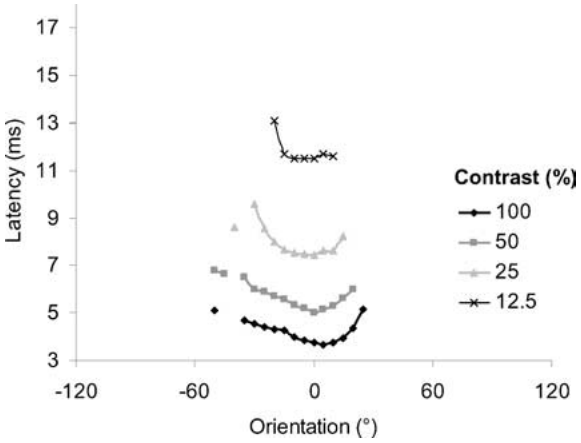

Figure 5. Spike latency of the first spike of the V1 neuron model (with 70% of inhibitory input suppressed) as a function of orientation for 4 different degrees of contrast and inhibition fixed to an intermediate value (sum of 1.2 μS). The output neuron was still able to respond to a wide range of contrast under these conditions.

an additional 4 Poisson spikes with a mean rate of 100 spikes per second (2-ms refractory period). Fast synaptic depression was implemented for excitatory connections, with synaptic efficacy divided by 3 for the second spike, 4 for the third, 5 for the fourth, and 6 for the fifth. As shown in Fig. 6, in the absence of inhibition, the model could not respond to changes in stimulus contrast. Note that absence of synaptic depression led to similar results (not shown). Thus fast feed-forward inhibition leads to responses at different levels of contrast even if input neurons discharge multiple spikes.

4. Discussion

I proposed a hierarchical scheme in which the outputs of LGN cells are pooled together to drive an oriented simple cell in V1. The simulations reported here should be taken as proof-of-concept that fast inhibition can shape excitatory input at the very beginning of the integration of incoming spikes, thus providing early orientation selectivity and contrast invariant responses for the highest level of inhibition tested. I proposed that non-orientation selective fast inhibition shapes early responses and accounts for early orientation selectivity and contrast invariance properties of V1 membranes (Celebrini et al., 1993; Volgushev et al., 1995; Carandini et al., 1997). The results show that, even in a reasonably detailed neurophysiological model, order sensitivity based on fast fast inhibition could indeed provide a very rapid mechanism for producing orientation selectivity. I will now review converging evidence arguing in favor of such a neuronal dynamics.

4.1. Fast Feed-Forward Shunting Inhibition

In real neurons, the onset of inhibition following a transient stimulation corresponds to shunting inhibition. Hyperpolarizing inhibition lasts as long as the IPSP itself, which can be tens of ms, depending on the time constant of the cell. Shunting effects however, last only as long as the post-synaptic ion channels remain open, i.e. for a period of a few ms in the case of $GABA_A$ receptors. In the model presented here, shunting

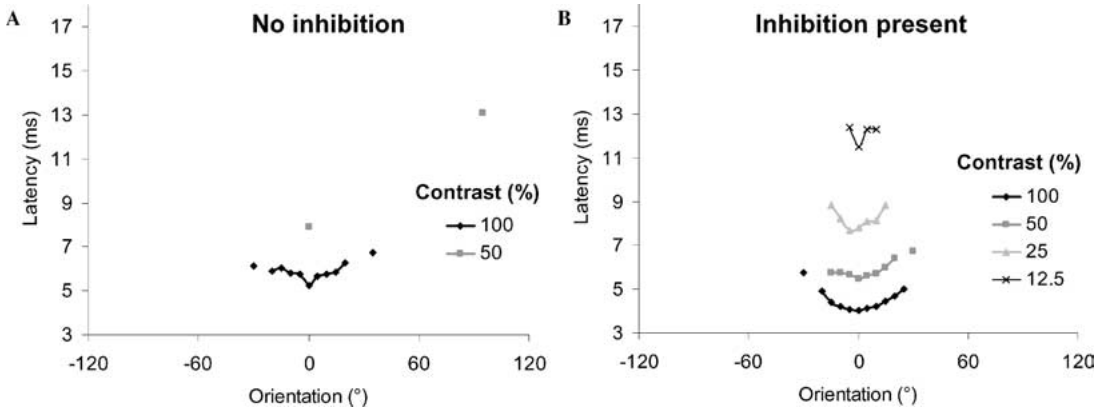

Figure 6. Using the model allowing several spikes per neuron (see text for details), latency of the first spike of the V1 neuron as a function of orientation and contrast. (A) Without inhibition, the output neuron discharged only at the highest stimulus contrasts. (B) With inhibition (sum of 4 μS), the neuron discharged selectively to a wide range of contrast.

effects—increases in inhibitory channel conductance as shown in Fig. 3—are present throughout EPSP integration.

Shunting effects result in an increased membrane conductance and recent experiments showed that after a flashed stimulus, visual inputs evoke strong shunting inhibition in visual cortical neurons (Berman et al., 1991; Borg-Graham et al., 1998; Anderson et al., 2000; Ferster and Miller, 2000). Both Borg-Graham et al. (1998) and Anderson et al. (2000) showed that membrane conductance for stimuli at the preferred orientation can rise to more than three times that of the resting state and it has been argued that the magnitude of such changes can easily be underestimated. This shunting inhibition seems to occur very rapidly after the stimulus onset (within a few ms) and can precede spike discharge (Anderson et al., 2000).

Most of the studies published on the topic of inhibition are directly or indirectly compatible with our model (Gabbott et al., 1988; Celebrini et al., 1993; Volgushev et al., 1995) and show that fast feed-forward inhibition might occur even before the visually evoked first spike of a neuron. Hirsch et al. (1998) made intracellular recordings from neurons in V1 while stimulating the retina with flashed black or white squares of various sizes in different locations. They showed that for white squares overlapping the ON and OFF regions of a neuron's receptive field, the very first milliseconds following excitation showed net effects that could be inhibitory even if the neuron received substantial excitatory input. Such data show that inhibition can indeed be fast enough to counteract the effects of excitatory postsynaptic potentials. Hirsch et al. (1998) also showed that for non-optimal stimuli—positive contrast on the OFF receptive field location—the decrease in activity cannot be explained by the suppression of thalamic inputs. Rather, as in our model, it seems to be due to a fast inhibitory process that is relatively independent of the stimulus. Volgushev et al. (1995) showed very similar results with flashed bars of different orientations. Extracellular recordings have also shown that a reduction of spontaneous activity triggered by non-optimal stimuli can have similar latencies to the spike onsets produced by optimal orientations (Celebrini et al., 1993). These studies demonstrate that inhibitory processes take place at the very beginning of an event-related neuronal response.

While all these studies strongly support the existence of the sort of fast feed-forward inhibition used in our model, even apparently contradictory studies that favor linear integration of spikes are compatible with transient shunting inhibition. Experimental and modeling works have argued that long-lasting shunting inhibition does not play a major role in orientation selectivity (Douglas et al., 1988; Ferster and Jagadeesh, 1992; Hirsch et al., 1998). However, most of these studies start from the basic assumption that stimuli are represented by the firing rate of neurons. Thus, even when modeling spiking neurons in V1 (Maex and Orban, 1996) or when implementing feedforward inhibition (Troyer et al., 1998), the discharge patterns of geniculate inputs involved in generating orientation selectivity are typically modeled using Poisson spike distributions. It is commonly supposed that an initial linear integration phase (Ferster and Jagadeesh, 1992; Hirsch et al., 1998) precede cortical amplification (Suarez et al., 1995; Adorjan et al., 1999) and/or normalization (Carandini and Ferster, 1997; Carandini et al., 1997). However, these models and the model I presented in this article act on a different time scale and they are by no means incompatible. The kind of processing I presented here would only occur in response to very fast changing stimuli or following the release of saccade-related inhibition in the geniculate at the end of an eye movement, when the spike timing is more reliable (Mechler et al., 1998).

4.2. Rapid Visual Processing

One of the other aspects of the current model that merits discussion is its relevance to rapid visual processing. The speed with which the visual system can process complex scenes poses a major challenge for current models of visual processing. Neural recording and behavioral data (Thorpe et al., 1996; Delorme et al., 2000; Keysers et al., 2001) imply that the underlying processing at each stage of the visual pathway must be extremely rapid, and at least some information needs to be made available within the first 10 milliseconds following the arrival of inputs from the preceding stage. These very severe temporal constraints are problematic for conventional rate codes because few cells will be able to emit more than one spike in less than 10 ms. Such data argue strongly in favor of a mainly feed-forward processing strategy in which computations need to be performed very rapidly. The model reported here is compatible with these time constraints and can account for very fast contrast invariant responses of V1 cells. Celebrini et al. (1993) showed for instance that a 10 ms

oriented bar presentation followed by a perpendicular mask is sufficient to elicit selective responses in V1 neurons.

My model also demonstrates the biological plausibility of using computationally efficient population codes for hierarchical processing in the visual system. If the amount of shunting inhibition in the modeled neuron depends on the number of spikes that have been received from the LGN, excitatory synaptic potential will be shunted to a degree that depends on their order of arrival rather than their time of discharge. This form of Rank Order Coding has a number of features that make it very interesting from a computational point of view (Thorpe, 1990; Gautrais and Thorpe, 1998; Van Rullen and Thorpe, 2001). In a previous model, I implemented a hierarchical neural network of two layer of integrate-and fire neurons, the first one implementing orientation selectivity as in the model presented here and the second one implementing face recognition. I have shown that such a network could perform complex visual processing tasks that include the localization and recognition of new faces in photographs and that it could cope with high level of noise (Delorme et al., 1999; Delorme and Thorpe, 2001). According to the Rank-order-coding hypothesis, the main aspect in the current model that plays a role in the encoding of orientation at different contrasts is the extent to which real neurons behave as perfect integrators.

I have shown that a simple mechanism that uses fast feed-forward shunting inhibition, coupled with the fact that strongly activated neurons will tend to fire early, provides a form of automatic gain control that allows selectivity to be maintained over a very wide range of stimulus contrasts. This new biologically plausible mechanism may be used to produce a model for orientation selectivity than can operate very quickly, even under conditions where each input only has time to emit one spike. This mechanism has also been shown to be computationally efficient to perform image processing. Finally, it is theoretically compatible with rate based model that operate over longer time scales.

The model leads to predictions that could be tested experimentally. Suppressing fast inhibition in a whole cell clamped cortical neuron should result in broader initial orientation selectivity and the loss of contrast invariant selective responses. However this blockage might have no visible effect at a longer time scale (Nelson et al., 1994). In addition, I would also expect fast shunting inhibition and membrane conductance increases to occur not only for the preferred orientation (Borg-Graham et al., 1998; Anderson et al., 2000) but also for other orientations.

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