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B. Li, M. R. Peterson, J. K. Thompson, T. Duong and R. D. Freeman

J Neurophysiol, August 1, 2005; 94 (2): 1645-1650.

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Intracortical Origins of Interocular Suppression in the Visual Cortex

F. Sengpiel and V. Vorobyov

J. Neurosci., July 6, 2005; 25 (27): 6394-6400.

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Binocular Cross-Orientation Suppression in the Cat's Striate Cortex

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Walker, Gary A., Izumi Ohzawa, and Ralph D. Freeman. Binocular cross-orientation suppression in the cat's striate cortex. *J. Neurophysiol.* 79: 227–239, 1998. When a cortical cell is activated by an optimal sinusoidal grating, its response can be attenuated by a superimposed second grating oriented orthogonally to the optimal stimulus. This effect is known as cross-orientation suppression (COS). In previous work, monocular characteristics have been explored and interocular tests have been conducted in an attempt to locate the origin of the suppression. In this study, we have recorded extracellularly from cortical cells to investigate the binocular characteristics of COS. Our hypothesis is that binocular disparity influences the strength of the effect. Our results do not support this supposition. We find that binocular COS is as strong as monocular COS, but disparity changes are of no consequence. We also conducted interocular tests in which the optimal grating and the orthogonal mask were seen by separate eyes. Although most interocular effects were weak, they were present in almost every cell and spanned a wide range of suppression strengths. We also tested the effect of asynchronous presentation of optimal and orthogonal gratings. These temporal offsets did not affect the strength of COS. We conclude that the suppressive mechanism underlying COS is primarily monocular and acts prior to the convergence of the two monocular streams.

INTRODUCTION

An optimal grating for a cortical cell has a reduced excitatory effect when a second stimulus is superimposed on the first. This effect was first demonstrated with orthogonally oriented stimuli (Bonds 1989; Morrone et al. 1982; Petrov et al. 1980) and is commonly referred to as cross-orientation suppression¹ (COS). However, it also occurs when the second stimulus is presented at any orientation (DeAngelis et al. 1992). This effect does not require an extended developmental process since it is manifest in kittens at 4 wk postnatal (Green et al. 1996). Thus COS is a fundamental property of primary visual cortex.

Gratings have been used to study various parameters of COS. As noted above, orientation of the masking grating is not critical for the suppressive effect. However, an orthogonal grating is preferable since it does not usually produce any excitation when presented alone, which allows observation of the suppression without additional excitation. Spatial phase and contrast of the masking grating have also been investigated. Spatial phase appears to be inconsequential but reduced contrast of the mask causes a substantial reduction in the magnitude of the suppression (Bonds 1989; DeAngelis et al. 1992). In most previous work, the optimal and mask gratings were shown to the dominant eye alone. A second configuration has also been attempted in which the one eye views the optimal

grating while the other views the masking grating. The goal of this test is to try to localize the site of COS. Results of these tests are mixed. While strong interocular effects have been reported (Ohzawa and Freeman 1986a,b), interocular COS has generally been found to be weak or absent (DeAngelis et al. 1992; Ferster 1981; Freeman et al. 1987). Recently, it has been reported that the timing between presentation of the primary and masking stimuli is important (Sengpiel and Blakemore 1994; Sengpiel et al. 1995).

The study we describe here is concerned with the binocular properties of COS. Although, as noted above, relative monocular phase appears not to matter in COS, binocular phase may be an important factor. Essentially all simple cells and around 40% of complex cells in striate cortex are sensitive to the relative interocular spatial phase of dichoptically presented sinusoidal gratings (Ohzawa and Freeman 1986a,b). These neurons presumably serve as the first stage in the processing of stereoscopic signals (Barlow et al. 1967). Since the interocular results vary considerably, we have used a more thorough and direct approach to study the binocular nature of COS. We first determined an optimal grating stimulus for a cortical cell and presented it binocularly. Next, we tested the cell with an orthogonal mask grating superimposed on the optimal grating at one of several disparities (relative spatial phases). Third, we presented the optimal grating to both eyes and the orthogonal mask to one eye only. Fourth, we conducted dichoptic tests in which the optimal grating was presented to one eye and the orthogonal mask was viewed by the other. Finally, we used several onset asynchronies between presentation of the optimal grating and the orthogonal mask to explore the temporal dynamics of COS.

In general, we find that the magnitude of COS does not depend on the relative phase of the orthogonal mask grating. In addition, the strengths of binocular and monocular COS are approximately equal. Dichoptic tests reveal interocular effects that are mainly weak but that cover a broad range of suppression. Asynchronous presentation of optimal and mask gratings does not alter the general effect. We conclude, therefore, that COS is mainly a monocular process.

METHODS

Surgical preparation

Extracellular recordings were made from cells in area 17 of anesthetized and paralyzed adult cats with tungsten-in-glass microelectrodes (Levick 1972). Thirty minutes prior to anesthesia, acepromazine maleate (0.5 mg · kg⁻¹) and atropine sulfate (0.06 mg · kg⁻¹) were injected subcutaneously to provide tranquilization and to suppress secretion, respectively. Femoral veins were cannulated for intravenous infusion, a tracheal tube and a rectal thermometer were inserted, and electrocardiographic (ECG) leads and elec-

¹ Extracellular techniques do not allow us to make a distinction between true suppression and withdrawal of excitation. Therefore, in this paper we use the terms suppression and inhibition interchangeably.

troencephalographic (EEG) screw electrodes were positioned. A craniotomy (approximately 5 mm in diameter) was performed around Horsley-Clarke coordinates P4L2 and the dura was carefully removed. Two electrodes were positioned just above the surface of the cortex at an angle of 10° medial and 20° anterior and the hole was covered with agar and sealed with wax to form a closed chamber.

During recording, animals were anesthetized and paralyzed by intravenous infusion of a mixture of thiamylal sodium (Bio-tal; 0.8 mg·kg⁻¹·h⁻¹) and gallamine triethiodide (Flaxedil; 10 mg·kg⁻¹·h⁻¹), combined with a 5% dextrose and lactated Ringer's solution (0.5 ml·kg⁻¹·h⁻¹). Steady-state hydration was provided by a drip system by which lactated Ringer's was infused (10 ml·kg⁻¹·h⁻¹). Animals were artificially respired with a mixture of N₂O (70%) and O₂ (30%) at 25 strokes per minute. Temperature was maintained near 38°C and end-tidal CO₂ at 4–4.5%. EEG, ECG, heart rate, and intratracheal pressure were monitored continuously. The pupils were dilated with 1% atropine sulfate, and nictitating membranes retracted with 5% phenylephrine hydrochloride. Contact lenses with 3-mm artificial pupils were placed on both corneas. Every 8–12 h, the contact lenses were removed and cleaned, and the clarity of the refractive media checked with an ophthalmoscope.

Experiments typically lasted 4 day after which the animals were given an overdose of pentobarbital sodium (Nembutal). After perfusion and fixation (with a buffered 0.9% saline solution followed by 10% Formalin), the cortex was frozen and sectioned into 50- μ m-thick slices. Tissue was stained with thionin, electrode tracks were reconstructed, and laminae identified. Histological reconstructions confirmed that all cells were in area 17.

Visual stimulation and receptive field mapping

The cat was positioned in front of a tangent screen on which a bar stimulus of variable size and orientation can be manually swept in any position and direction for initial mapping of the receptive field (RF). Two cathode ray tube (CRT) displays (Nanao T2–17, refresh rate 76 Hz), were used to allow independent stimulation of each eye. Each CRT was placed 57 cm from the eye such that the active screen area subtended 28 × 22° of visual angle, and the RFs were located near the middle of the screen. The mean luminance at the front surface of the contact lens is 23 cd/m².

For presentations requiring two superimposed gratings, the component gratings are displayed on alternate scan lines (line interleaving) to avoid any interaction of the two components resulting from the bandwidth limitations of the video amplifiers in the displays (Pelli and Zhang 1991). Some conditions call for two gratings to be superimposed on one monitor while the other monitor displays only one grating. In these conditions, the solo grating is line-interleaved with mean-luminance lines to equal the effective contrast of its matched grating in the other monitor. Frame refresh on the two displays is synchronized.

We sought balanced binocular cells but tested all cells that showed binocular interaction. When a cell was isolated, the RF was first qualitatively mapped by hand, and a rough measure of its orientation and spatial frequency tuning was determined. Following these initial results, the orientation and spatial frequency were determined quantitatively by computer controlled runs that randomly present an appropriate range of orientations and spatial frequencies of a drifting grating (2-Hz temporal frequency). The length and width of the stimulus patch was varied to determine the degree of end and side inhibition (DeAngelis et al. 1994), and subsequently the stimulus was constrained to lie within the excitatory center of the RF (typical grating patch sizes were 3–7° in diameter). Next, orthogonal gratings of various spatial frequencies were superimposed monocularly to determine the spatial frequency which produced the most suppression. This spatial frequency was used in the orthogonal grating

for all future runs. This value was typically the same or similar to the optimal excitatory spatial frequency, confirming earlier results by DeAngelis et al. (1992).

RESULTS

Altogether, 84 cells were studied in area 17 [33 simple (S), 51 complex (Cx)]. Forty-five (15 S, 30 Cx) were used in the binocular experiment and an additional 39 (18 S, 21 Cx) were used in the interocular experiment. The RFs for all cells were located within 15° of the area centralis. Simple and complex cell classifications were determined by use of standard criteria (Hubel and Wiesel 1962). We also used the ratio of the first harmonic and mean of the response to a drifting grating stimulus (Skottun et al. 1991).

Binocular suppression

IS BINOCULAR CROSS-ORIENTATION SUPPRESSION SENSITIVE TO DISPARITY? Most neurons in the striate cortex are sensitive to the relative disparity of binocular stimuli (Barlow et al. 1967; Ferster 1981; Ohzawa and Freeman 1986a,b). We expect, therefore, that the inhibitory processes invoked during COS should also be modulated by the relative disparity of the suppressive orthogonal grating. A possible way in which this may occur is that the cycle of facilitation-suppression shown by phase-shifted grating stimuli is duplicated for suppression from the orthogonal stimuli.

To investigate this, we first determined the optimal monocular driving stimulus for a given cell. Optimal monocular stimuli were then presented dichoptically, and one grating was phase shifted with respect to the other over 360° in a series of randomly interleaved presentations to determine the disparity which yielded the maximum response for the cell (Ohzawa and Freeman 1986a,b). This phase disparity was used for all subsequent binocular presentations of the optimal stimulus. Next, we superimposed an orthogonal mask on the optimal grating for each eye to generate suppression. The spatial phase of the mask for one eye was randomly shifted with respect to the phase of the mask in the other eye to probe the disparity sensitivity of the suppression. When possible, this measurement was repeated, but switching the stimuli on the two monitors so that the orthogonal mask was phase-shifted through the other eye. Results are independent of which eye viewed the phase-shifted orthogonal mask. Figure 1 shows an example of the stimuli which consist of an optimally oriented grating at a fixed optimal disparity superimposed on an orthogonal mask at variable disparities. The cyclical nature of the grating stimuli guarantees that all possible disparities for the mask are sampled. Monocular tests were also included in which the mask was presented to one eye while the optimal stimulus was presented to both. This condition allowed us to compare the strength of suppression and the tuning in the binocular and monocular conditions. If there is modulation of suppression in the monocular condition, it implies a monocular phase interaction between the two gratings, and this can serve as a baseline for comparison with the phase tuning observed in the binocular condition. Finally, we presented optimal stimuli to each eye alone and to both eyes simultaneously to establish control response levels for no-mask conditions. Within a set of presentations, all trials were randomly

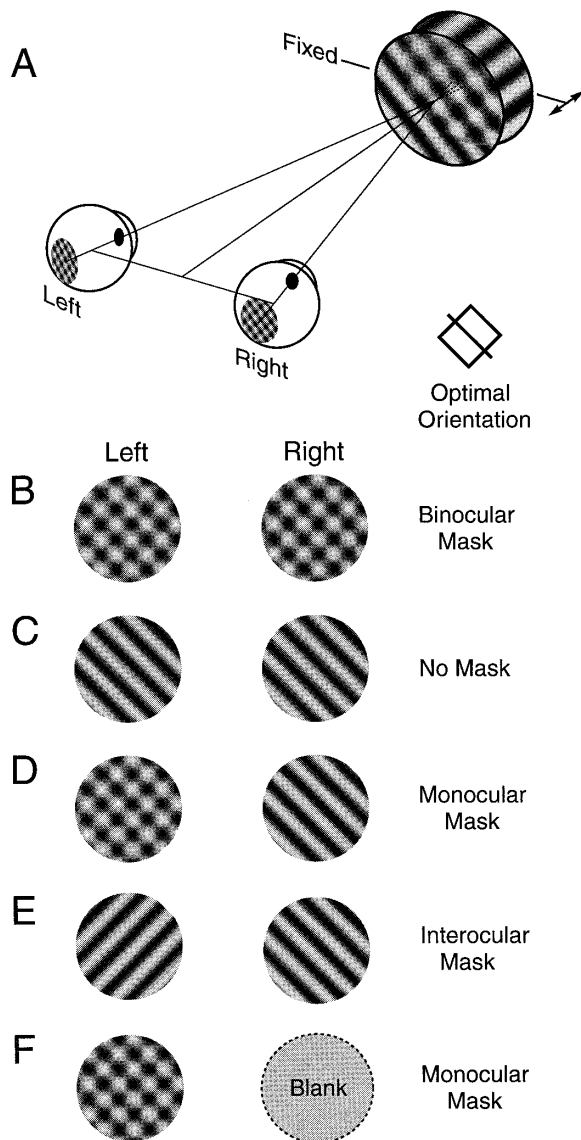


FIG. 1. A schematic illustration of the visual stimuli. *A*: 3-dimensional interpretation of the visual stimuli associated with the primary condition in the binocular COS experiment. An optimal grating is presented dichoptically, and a second orthogonal grating is superimposed at variable disparity. *B*: monocular images of stimuli depicted in *A*. Each eye is monocularly masked, thus forming a plaid, but the cyclopean percept is two gratings at different depth planes. *C*: left and right eye images for the optimal binocular stimulus with no mask. *D*: monocular mask superimposed on the optimal dichoptic stimulus. *E*: interocular masking configuration. *F*: monocular mask superimposed on an optimal monocular grating. For each stimulus set *B*–*F*, there is a corresponding pair in which the left and right eye stimuli are switched. Monocular masking in *D* and *F* can be distinguished by the underlying excitatory grating. In *D*, the cell is dichoptically excited, whereas in *F* there is only monocular excitation.

interleaved and temporally separated by a period of 3 s during which a blank screen was presented at the same mean luminance as the gratings. Spontaneous activity was assessed during null stimulus trials interleaved in the stimulus set.

Typical responses from a simple cell and a complex cell are shown in Fig. 2, *A* and *B*, respectively. There are three conditions in which the effect of varying the phase of one

of the orthogonal gratings is measured. For all three conditions, the optimal grating is presented binocularly. Open symbols represent trials in which the orthogonal grating was presented to one eye only. The curves obtained from these trials indicate the degree to which the monocular phase of the orthogonal grating influences the response. The filled symbols are obtained from binocular presentation of the orthogonal mask. The phase differences in these stimuli correspond to changes in relative disparity of the orthogonal grating. Binocular suppression is uniformly stronger than mon-

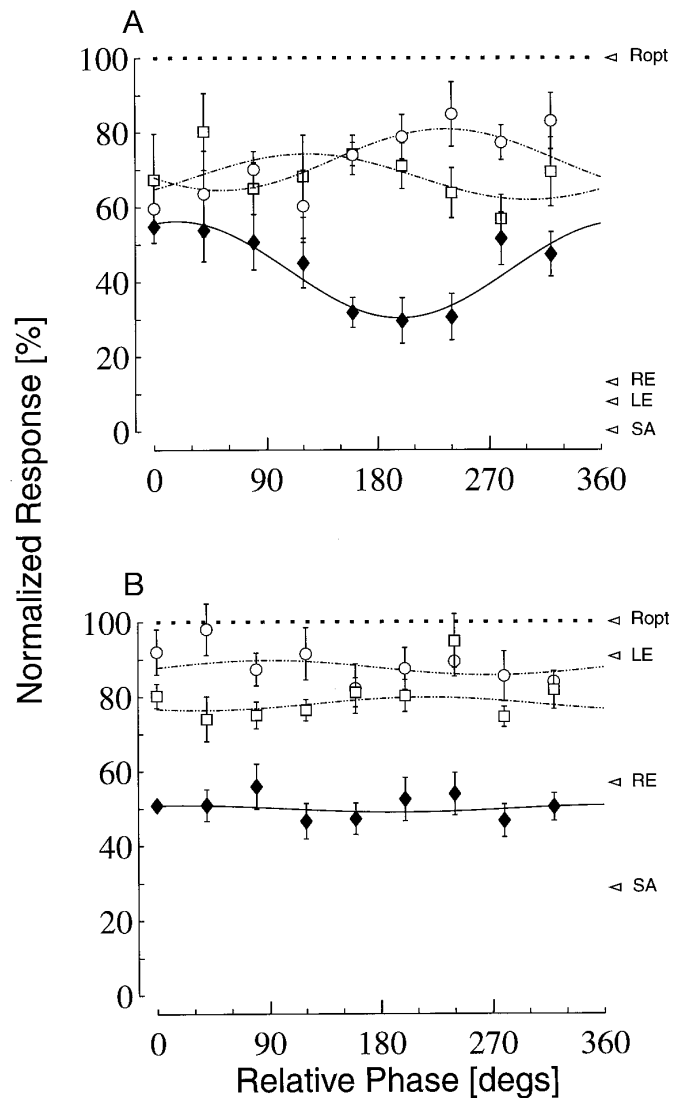


FIG. 2. Binocular cross-orientation suppression (COS) vs. relative interocular phase difference (disparity) for a simple cell (*A*) and a non-disparity tuned complex cell (*B*). The filled symbols are from binocular masking presentations, and the open symbols are from monocular masking conditions (circles = right eye masked; squares = left eye masked). All curves are least-squared fits with 1 cycle of a sinusoid. The dashed line at 100% is the response to the optimal binocular stimulus alone (R_{opt}). Error bars are ± 1 SE of the mean. Response to the optimal stimulus, presented alone to the left and right eyes, is denoted by the LE and RE pointers, respectively. SA, spontaneous activity during presentation of a uniform gray screen. The data have been normalized because the monocular masking data are collected in separate trials and R_{opt} typically varies slightly over time. The average R_{opt} for *A* and *B* is 56.20 and 68.22 spikes/s, respectively.

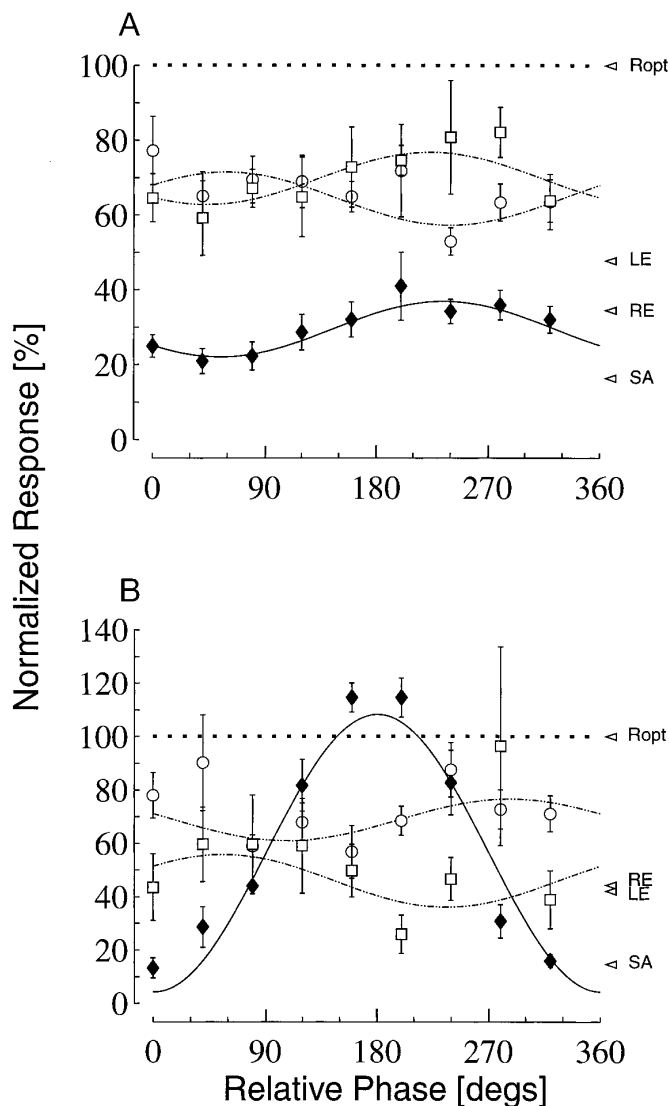


FIG. 3. Responses of 2 additional complex cells. Symbols are as in Fig. 2. *A*: disparity sensitive complex cell with weak COS tuning in both the monocular and binocular conditions (average $R_{opt} = 20.45$ spikes/s). *B*: complex cell showing binocular tuning of suppression and a lack of monocular tuning. Optimal grating parameters: orientation = 150° ; spatial frequency = 0.65 cycles/°; size = 5° grating patch (square window). Note that this cell shows a large degree of excitatory binocular summation, as seen by comparing the monocular responses (LE, RE) with R_{opt} (average = 8.73 spikes/s).

ocular suppression,² and in both cases, responses are weaker than the response to the optimal stimulus (R_{opt} , dashed line). In the case of the nondisparity tuned complex cell (Fig. 2*B*), responses are generally unchanged as a result of varying the disparity of the mask. An example of results from a phase sensitive complex cell is shown in Fig. 3*A*. Although tuned for the disparity of optimally oriented stimuli, there is minimal modulation of COS for the binocular condition.

The data are fit with one cycle of a sine wave using the Levenberg-Marquardt method of least-square errors which takes into account the variance of each data point (Press et

² In this experiment, monocular suppression refers to the suppression obtained by presenting an orthogonal grating to one eye while providing binocular excitation with an optimal grating.

al. 1992). For a number of cells in the current study, the amplitude of the sinusoid is so small that it results in a nearly straight line (for example, Fig. 2*B*). Clearly, in these cases, there is no tuning.

The data from most cells are similar to those in Figs. 2 and Fig. 3*A*. However, there are a few exceptions. Figure 3*B* shows the response of a cell which exhibited striking modulation of suppression that is dependent on the disparity of the orthogonal grating. Furthermore, this cell's monocular tuning is essentially flat, implying that the binocular effect is due solely to binocular disparity interactions and not to monocular phase dependence. We repeated the entire protocol for this cell and obtained equivalent results. Aside from the phase tuning of the suppressive effect, this cell was not extraordinary. Histological reconstructions show that this cell was in layer 6.

To quantify the tuning of the suppression, a modulation index (MI) was computed. We define the MI as the ratio of the amplitude (A : measured as peak-to-trough height) of the sinusoidal fit and the response to the optimal stimulus (R_{opt} : spontaneous response subtracted first)

$$MI = A/R_{opt}$$

An MI of 0 represents a flat tuning curve (no modulation) and MI of 1 represents complete modulation between the spontaneous level and the maximum excitatory level. If the amplitude of the sinusoid brings the response above R_{opt} and below the spontaneous levels, as in Fig. 3*B*, MI will be greater than one.

A summary of the results for all cells, in terms of the MI, is shown in Fig. 4. Most of the points are clustered near the origin and 83% (66/80) of the data points have an MI of <0.4 for both the binocular and monocular conditions. Clearly, interocular phase disparity has a minimal modulatory effect on the strength of suppression for both monocular and binocular conditions.

A COMPARISON BETWEEN BINOCULAR AND MONOCULAR SUPPRESSION. For most cells, binocular suppression is consistently stronger than monocular suppression at all phase disparities. To quantify the strength of suppression, we computed a suppression index (SI)

$$SI = 1 - M/R_{opt}$$

where M is the mean of the fitted sinusoid. Spontaneous activity is subtracted from M and R_{opt} before calculating the index. In this formulation, an SI of 1 represents complete suppression, with the spontaneous response used as the zero level. An SI of 0 represents no suppression. The contrasts of the optimal and orthogonal gratings were chosen with the aim of attaining an SI close to 0.5 in the binocular conditions. It should be noted though, that because monocular suppression is generally weaker, some responses yield negative SIs, indicating that the response with the mask was stronger than the response without the mask. However, in no case was there strong facilitation of the response when the orthogonal grating was presented to one or both eyes.

In the binocular case, suppression and tuning should not depend on which eye is presented the phase shifted grating because the stimuli are symmetric with respect to the two eyes. But for monocular suppression, there are two distinct cases since the mask grating may be presented to the left or right eye, which

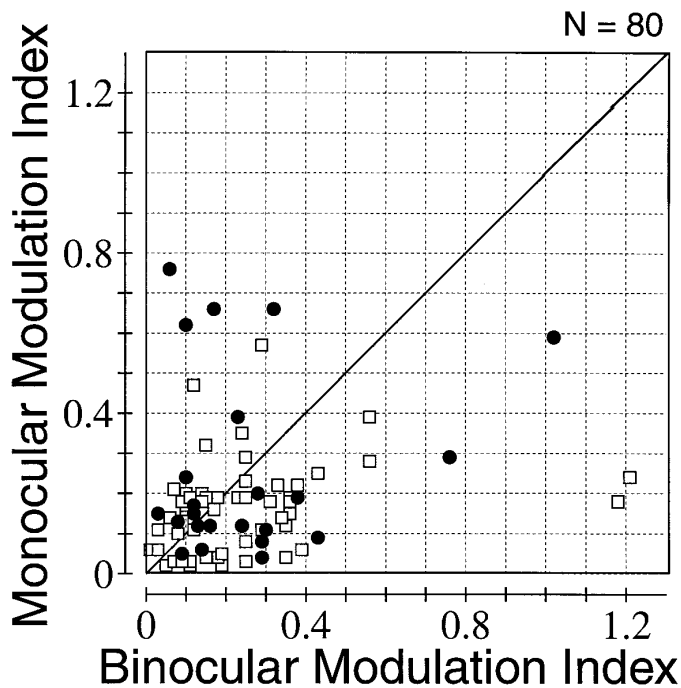


FIG. 4. Modulation index (MI) of suppression for our population of cells in the binocular experiment. This figure shows that little modulation occurs in either binocular or monocular COS. MI for monocular masking (Fig. 1D) is plotted on the y axis, and the MI for binocular masking (i.e., Fig. 1B) is plotted on the x axis. ●, simple cells; □, complex cells. These same symbols are used in remaining graphs. A line of slope 1 denotes an equal degree of modulation in the 2 conditions. Monocular data are paired with binocular data such that the mask is phase shifted for the same eye in the 2 conditions. Thus, for most cells, there are 2 data points and usually the 2 binocular MI values are very similar. For example, the complex cell of Fig. 3B is represented by the 2 rightmost squares of the plot. Cells which were lost before the second binocular run was completed have only 1 point on the plot.

excite the neuron differently, depending on the ocular dominance. SI values of monocular and binocular suppression for dominant and nondominant eye suppression are shown in Fig. 5, A and B, respectively. In these plots, all points lie either near or below the line of slope = 1. This confirms that suppression is generally stronger in the binocular condition. Qualitatively, the points tend to lie closer to the diagonal line and are distributed over a broader monocular range in Fig. 5A, implying that the dominant eye contributes most of the suppression. Also, the large cluster of points lying along the abscissa in Fig. 5B indicate that for these cells, the nondominant eye contributes no monocular suppression. Quantitatively, with binocular excitation, the mean monocular SI for the dominant eye is 0.34 ± 0.27 (SD), which is significantly ($P < 0.01$) stronger than the suppression index of 0.20 ± 0.22 (SD) obtained from the nondominant eye. Both monocular measures of SI were significantly lower than the binocular SI ($P < 0.001$), while the repeated measures of the binocular SI were equivalent ($P > 0.75$).

In summary, the disparity of an orthogonal mask grating does not affect the strength of binocular COS. Typically, a binocular cross-orientation stimulus suppresses the response of a cell to a certain level, and varying the disparity usually causes a slight fluctuation of the suppression around that level. One strongly suppressed complex cell was an exception to this trend. For this cell, a disparity was found for which suppression was minimal and another for which suppression was complete (Fig. 3B).

Interocular suppression

The goal of this experiment is to evaluate the degree to which a mask in one eye can suppress the response of an

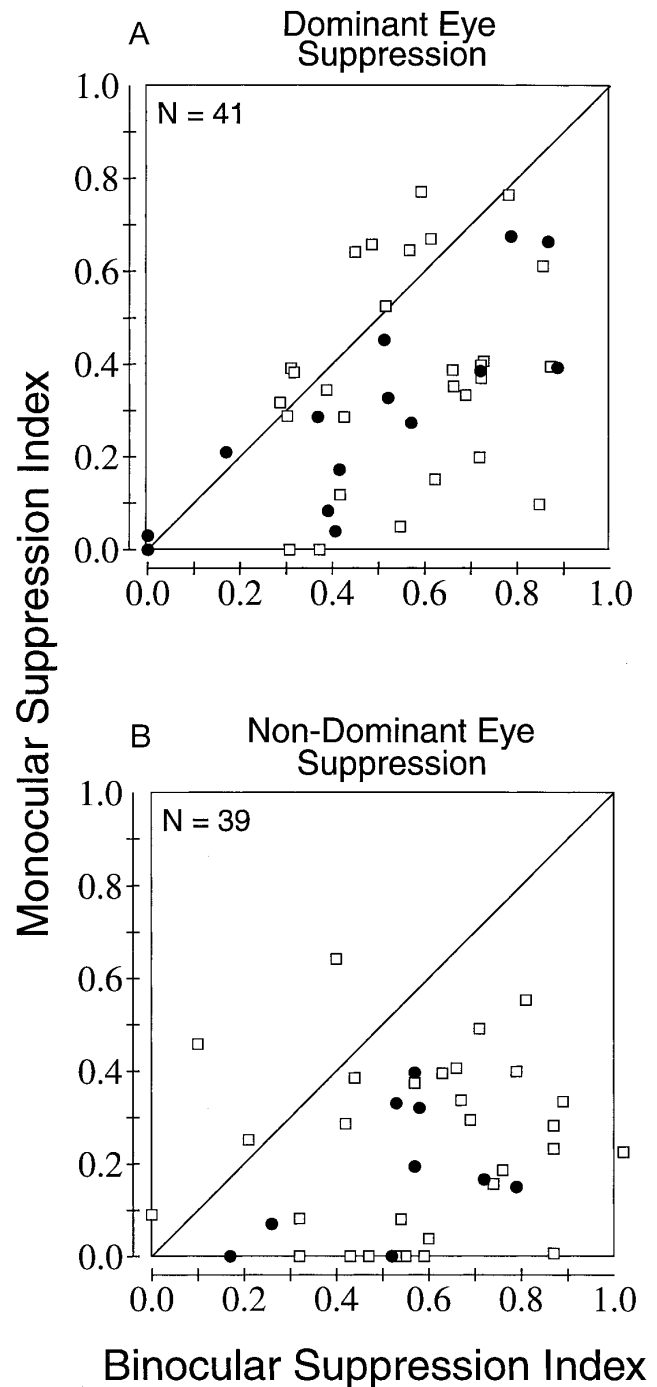


FIG. 5. The strength of suppression for the population of cells. The suppression index (SI) for the monocular condition is plotted on the y axis, and the SI for the binocular condition is plotted on the x axis. The identity relationship is drawn as a reference (—). Points on this line signify an equal amount of suppression in both the binocular and monocular COS conditions. A: data from the runs in which the mask was presented to the dominant eye in the monocular COS conditions. B: data from runs in which the mask was presented to the nondominant eye in the monocular COS conditions. Points with negative values (indicating facilitation) were placed at zero on the appropriate axis. A single point in B has binocular suppression to a level below the spontaneous response and thus has an SI greater than one.

excitatory stimulus in the other eye. As in the previous experiment, the mask is an orthogonally oriented, drifting grating. In this experiment, we measure the mean spike response during 4 s of interocular stimulation in which one eye views an optimal grating and the other eye views the orthogonal mask (Fig. 1E). The optimal orientation for the two eyes is generally slightly different because of ocular cyclo-rotation associated with anesthesia and paralysis. Thus, orthogonality is defined as 90° from optimal for the eye to which the mask is presented. The optimal and mask gratings were either presented simultaneously or the optimal grating preceded the mask by 1–5 s. Every condition ends with 4 s of interocular cross-orientation stimulation. Thus, the duration of the longest condition is 9 s (5 s optimal only plus 4 s interocular stimulus). We therefore included a control condition in which the optimal grating was presented alone for 9 s to estimate the time course of intrinsic adaptation.

Examples of results from two complex cells tested in this manner are shown in Figs. 6 and 7. Figures 6A and 7A show runs in which the optimal grating was presented to the dominant eye and the mask was presented to the nondominant eye. Figures 6B and 7B are runs for which the mask was presented to the dominant eye and the optimal grating to the nondominant eye. Eye dominance was determined in the conventional way from earlier runs for orientation and spatial frequency tuning. Note that for the cell shown in Fig. 7, the distinction between dominant and nondominant eye is somewhat arbitrary since nearly equal responses were elicited through either eye. We quantified the suppression as the ratio of responses during interocular stimulation (open circles in Figs. 6 and 7) to that of the corresponding 4-s time period of the control (filled circles in Figs. 6 and 7). For example, to measure the effect of the 5-s onset asynchrony condition, we compare the interocular cross-orientation response to the response in the control condition beginning at 5 s and ending at 9 s [see peristimulus time histograms (PSTHs) in Fig. 6]. This method assures that we do not mistakenly measure adaptation instead of suppression. When possible, the above procedure was run twice; once with the dominant eye viewing the optimal grating, and again with the dominant eye viewing the orthogonal mask grating. For each stimulus configuration, the individual trials were randomly interleaved and the entire set was repeated between 4 and 15 times. Each presentation was separated by 3 s during which a blank screen was presented.

Responses to interocular cross-orientation stimuli spanned the range from strong suppression to no suppression or even weak facilitation. Onset asynchrony between the optimal and the orthogonal grating had no effect. Notice that for the cells in Figs. 6 and 7, the amount of interocular COS depended on which eye viewed the orthogonal grating. The cell in Fig. 6 displayed almost no interocular COS when the orthogonal grating was viewed by the nondominant eye but exhibited consistently strong suppression at all onset asynchronies from the dominant eye. A similar result was observed for the cell in Fig. 7, which is somewhat unexpected since this cell had nearly balanced monocular input. This may be an indication that the ocular dominance of suppression is independent of the ocular dominance of excitation. Additionally, it is clear that the onset asynchrony does not alter the amount of suppression.

In Fig. 8A, we plot the percentage change between the

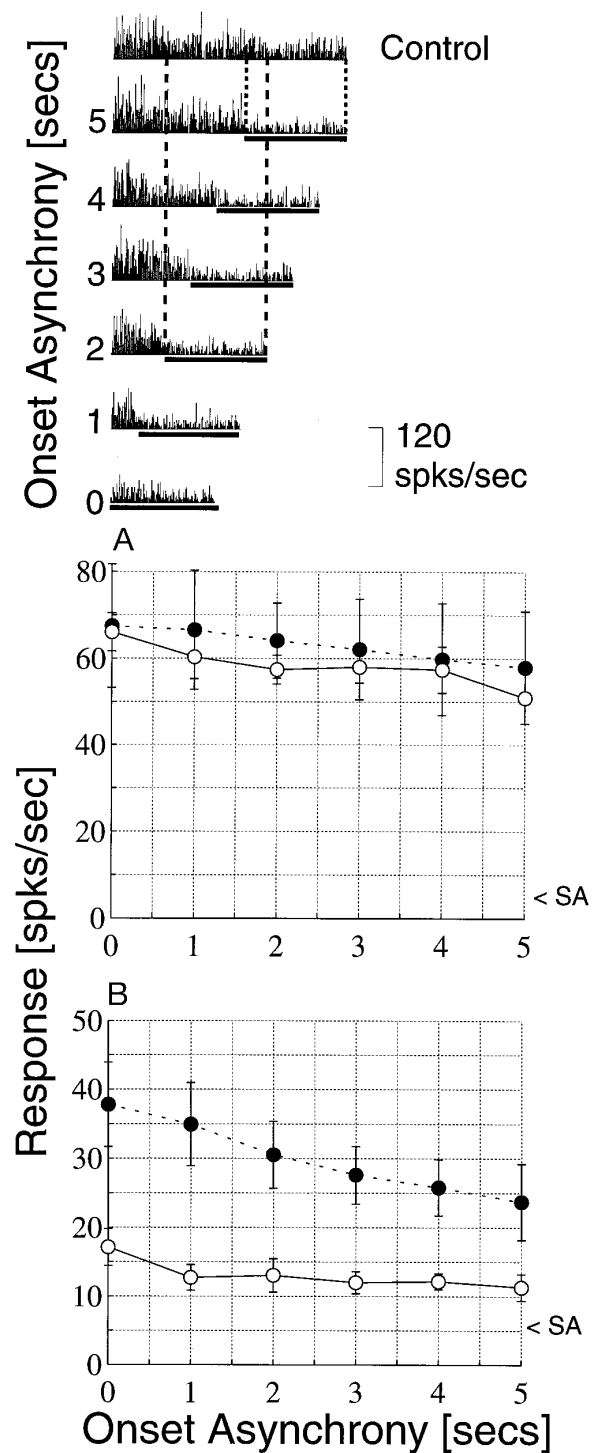


FIG. 6. Data from a complex cell in the interocular cross-orientation suppression experiment. Peri-stimulus time histograms (PSTH) illustrate responses to stimuli with various temporal onset asynchronies. The optimal grating is visible to one eye for the entire duration of each presentation, and the orthogonal grating is visible to the other eye during the last 4 s of each presentation, indicated by the thick lines under the PSTHs. Dashed lines extending upward from the 2-s and 5-s asynchrony conditions illustrate how the data are analyzed. The interocular response (thick underline) is compared directly with the temporally corresponding response from the control. A: the response when the nondominant eye viewed the mask grating. ○, the responses during interocular cross-orientation stimulation. ●, the responses from the portion of the control run which temporally matches the cross-orientation presentation. Error bars denote ± 1 SE of the mean. B: response when the dominant eye viewed the orthogonal mask grating. The PSTHs for these data are shown at the top of the figure.

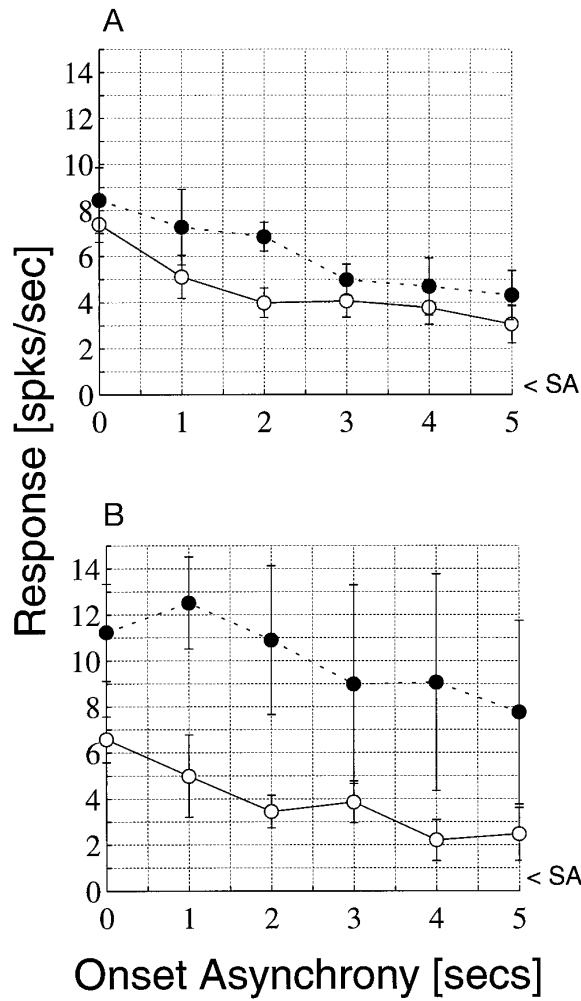


FIG. 7. Interoocular COS data from another complex cell. *A* and *B*: same conditions as in Fig. 6 (*A* and *B*).

baseline response and the interocular responses as a function of onset asynchrony for all cells. A negative percentage indicates suppression and a positive percentage denotes facilitation relative to the response to the optimal stimulus in the control condition. Most curves are essentially flat, indicating that the degree of suppression does not vary with onset asynchrony. In addition, there is a wide range of interactions, from strong suppression to weak facilitation. To quantify this, a linear regression was performed on each trace in Fig. 8*A*. The distribution of the slopes from the regression, shown in Fig. 8*B*, demonstrates that onset asynchrony did not change the suppression level significantly [mean = $-0.11 \pm 4.24\%/s$ (SD)]. If suppression becomes stronger with increased onset asynchrony, there should be a larger negative slope. Figure 8*B* also shows that the slopes are clustered near zero, and there are roughly equal numbers of positive and negative slopes. A plot of the fitted *Y* values at onset delays of 2.5 s (the midpoint of the linear regression) in Fig. 8*C* shows a mean of $-19.92 \pm 29.03\%$ which is significantly different from zero (2-tailed *t*-test, $t = -5.31$ with 59 df $P < 0.0001$). This value compares well with the distribution of suppression observed at each onset asynchrony (see Table 1). The mean suppression of all cells at all onset delays was -19.95% . Note that the uppermost trace in Fig. 8*A* was quite variable in response

which accounts for its erratic shape. This cell is the outlier in Figs. 8, *B* and *C*, and 9, *B* and *C*, as well.

For our entire cell population, the distribution of suppressive strength for each onset asynchrony yields no significant pairwise differences at the 5% significance level. In fact, the distribution of suppression for each onset asynchrony is quite uniform (Table 1). Using a nonparametric matched sign-test for all pairwise comparisons, only the (simultaneous, 1-s delay) pair yields a significant difference ($P = 0.003$). A Wilcoxon signed-rank test confirms that of all the pairs, only the (simultaneous, 1-s delay) pair is marginally different ($z = 2.13$, $P < 0.04$).

RELATION BETWEEN SUPPRESSION, BINOCULARITY, AND EYE DOMINANCE. What is the relationship between the degree of binocularity and the amount of interocular COS? Specifi-

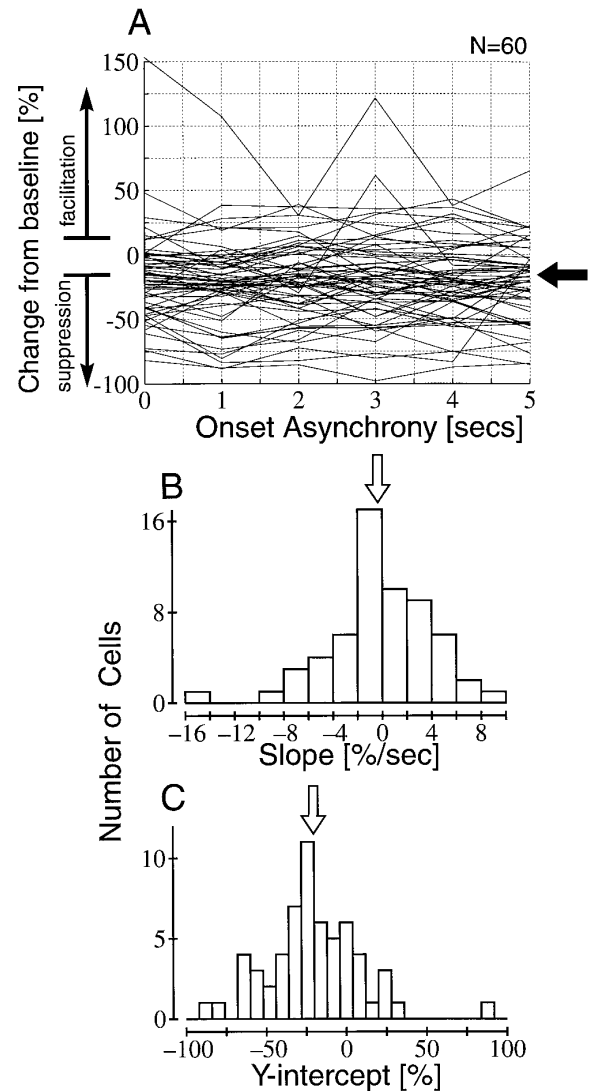


FIG. 8. Summary of all cells in the interocular COS experiment. *A*: the difference in response between the optimal monocular stimulus and the interocular mask conditions was quantified as the percent change in response with COS stimulation: $[(R_{\text{mask}}/R_{\text{opt}}) - 1] \cdot 100\%$. A negative percentage means there was interocular suppression relative to R_{opt} . A positive percentage indicates facilitation with respect to R_{opt} . The *x* axis is the onset asynchrony, as in Figs. 6 and 7. All the data are superimposed to show the macroscopic trend. *B*: histogram of the slopes of the linear regression of each line in *A*. Mean = $-0.11 \pm 4.24\%/s$ (SD). *C*: histogram of the *y* intercept of the linear regressions of data in *A*. Mean = $-19.9 \pm 29.0\%$ (SD).

TABLE 1. *Interocular suppression for each onset asynchrony*

| | Onset Asynchrony, (s) | | | | | |
|---------|-----------------------|------------------|------------------|------------------|------------------|------------------|
| | 0 | 1 | 2 | 3 | 4 | 5 |
| Mean, % | -18.0 ± 34.0 | -22.0 ± 33.2 | -20.5 ± 28.8 | -18.6 ± 35.3 | -20.4 ± 29.2 | -20.2 ± 29.0 |

Values are in means \pm SD.

cally, we want to know if a cell with equal excitation from both eyes is more likely to exhibit interocular COS than a cell which is driven primarily through one eye. The suppression for each cell was calculated as the average percent change from all onset asynchronies, and an ocular balance index (OBI) was computed to describe the degree of binocularity of each cell (Anzai et al. 1995). An OBI of 1 means the cell receives equal input from the two eyes. An OBI of 0 corresponds to strictly monocular input. Values between 0 and 1 represent varying degrees of binocular input. For example, the cells of Figs. 6 and 7 have OBIs of 0.53 and 0.97, respectively. The results of this analysis are shown in Fig. 9. Our sample is biased with respect to the OBI in that most of the points lie in the right half of Fig. 9A. This is because some excitatory response was required to identify the location of the RF for each eye. Figure 9A shows that the degree of interocular COS is not related to the binocularity. For the nearly monocular cells in this study, suppression is close to that of the mean suppression for the population.

Recall that both cells in Figs. 6 and 7 show no suppression when the mask is in the nondominant eye but strong suppression when the mask is in the dominant eye. Figure 9B highlights the difference between these two stimulus configurations and indicates that there is a slight tendency over the population for stronger suppression when the mask is presented to the dominant eye, although the difference is not significant (two-tailed t -test, $t = 1.94$ with 58 df, $P = 0.06$). We then performed a pairwise comparison of this data, discarding cells for which only one configuration was completed. This analysis yields a significant difference (2-tailed t -test, $t = 2.67$ with 22 df, $P = 0.014$).

Finally, we note that simple and complex cells appear to exhibit different levels of suppression (Fig. 9C). In particular, simple cells averaged 10.9% suppression, significantly less than the 28.3% averaged by complex cells (2-tailed t -test, $t = -2.44$ with 28 df, $P = 0.02$).

Monocular suppression

To complete this study of cross-orientation suppression, we compare the suppressive characteristics of binocular, interocular, and monocular suppression.³ We measured monocular COS as the change in response when an orthogonal grating is superimposed with an optimal grating in one eye only (Bonds 1989; DeAngelis et al. 1992; Morrone et al. 1982). We varied the spatial frequency (SF) of the orthogonal grating to obtain the SF tuning of the suppression. Figure

³ In this section we use "monocular suppression" to describe the condition in which the optimal and masking stimuli are presented to one eye while the other eye views a blank screen (Fig. 1F). This should be distinguished from the condition of binocular stimulation with an optimal grating combined with a masking stimulus in one eye (Fig. 1D).

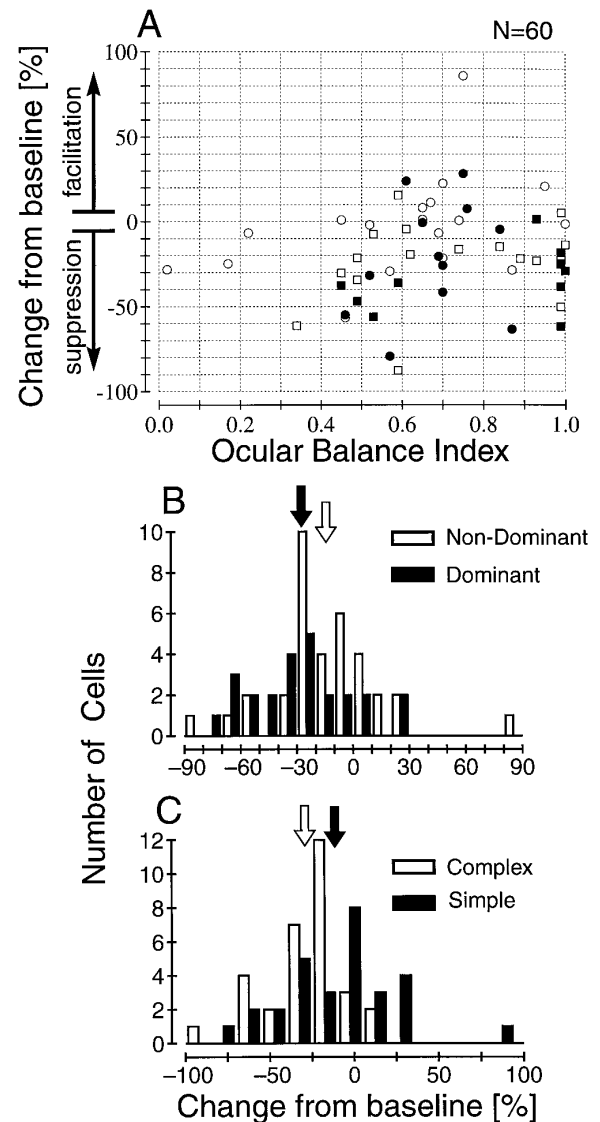


FIG. 9. Comparison between degree of suppression and binocularity. The ocular balance index (OBI) is calculated as: $1 - 2 \cdot |R_I / (R_I + R_C) - 0.5|$; where R_I and R_C are the responses of the ipsi- and contralateral eyes. The "change from baseline" is the average ratio between the interocular response and the corresponding control for all onset asynchronies [0–5 s]. A: filled and open symbols denote that the mask was presented to the dominant or nondominant eye, respectively. Squares are complex cells. Circles are simple cells. There are 2 data points for each cell, unless the cell was lost prematurely or was monocular. Balanced binocular cells have an OBI of 1 and purely monocular cells have an OBI of 0. B: histogram of data in A, obtained by collapsing the x axis, to eliminate the OBI distinction. This histogram shows slightly stronger suppression from the dominant eye (■: mean = -28.3%) than from the nondominant eye (□: mean = -13.9%). C: histogram obtained in same way as B but differentiating between simple and complex cells. This histogram shows that simple cells in our sample (■: mean = -10.9%) tend to be less suppressed than complex cells (□: mean = -28.3%).

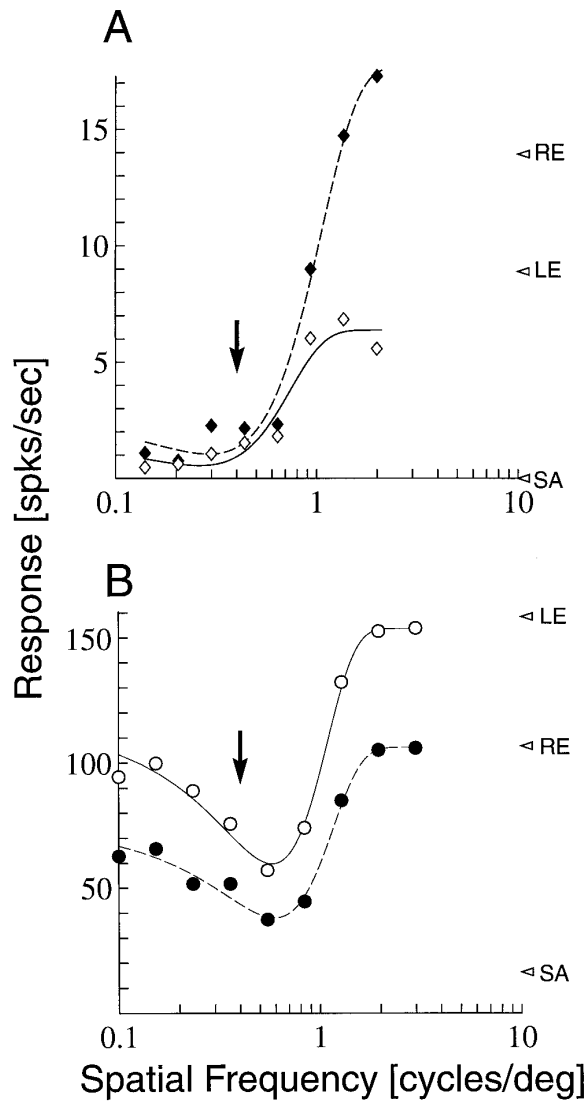


FIG. 10. Two examples of monocular excitation and COS in the same eye. Open and filled symbols denote left and right eye data, respectively. The smooth curves are the least square fits of Eq. 1. Responses to optimal stimulation alone are indicated (right). SA, spontaneous activity. The arrows indicate the optimal SF, which was 0.4 cycles/° for both cells in this figure. A: simple cell. Data points are the first harmonic of the response. $SF_{opt} = 0.4$ cycles/°; contrast for excitation and mask was 40 and 60%, respectively. B: complex cell. Data points are the DC response. $SF_{opt} = 0.3$ cycles/°; Contrast for excitation and mask was 10 and 35%, respectively.

10 shows the response for a simple and a complex cell. The data are fit by a Gaussian function subtracted from a constant

$$R = k - A \cdot \exp[-0.5 \cdot (sf - sf_{opt})^2 / \sigma^2] \quad (1)$$

where R is the response; k is an estimate of the response evoked by the optimal stimulus alone (R_{opt}); A is the amplitude of the Gaussian; sf is the variable spatial frequency; sf_{opt} is the optimal SF of the suppression, and σ is the standard deviation of the Gaussian. Strong suppression and good fits were obtained for most cells, usually with a clear minimum near the preferred spatial frequency for the cell, as indicated by the downward arrows in Fig. 10. For the population, the Spearman correlation coefficient between the optimal excitatory and inhibitory SF is 0.31 and neither a paired

t -test ($P = 0.94$) nor a nonparametric Wilcoxon test ($P = 0.59$) revealed a significant difference between the two distributions. Also, in previous studies, COS was measured only in the dominant eye (Bonds 1989; DeAngelis et al. 1992; Morrone et al. 1982), while in our study, monocular COS is examined in both eyes which allows us to compare suppression in the two eyes. We find that the strength of COS is usually correlated with the strength of the excitatory input, such that stronger suppression is typically observed through the dominant eye.

Figure 11 shows the summary for the four suppressive types studied in this paper: 1) monocular suppression with binocular excitation (Fig. 11A, unfilled bars); 2) binocular suppression with binocular excitation (Fig. 11A, filled bars); 3) interocular suppression with monocular excitation (Fig. 11B, unfilled bars); 4) monocular suppression with monocular excitation (Fig. 11B, filled bars). We find that monocular suppression with monocular excitation produces the strongest suppression (mean SI = 0.62), although it is not significantly different from binocular suppression (mean SI = 0.55). The strength of these suppressive conditions contrasts with the weak suppression observed in monocular suppression with binocular excitation (mean SI = 0.26) and interocular suppression (mean SI = 0.22), which were not significantly different.

Why is monocular COS with binocular excitation so weak with respect to purely monocular COS? Strong monocular suppression is expected for the eye viewing the mask, and we expect weak suppression of the other eye via interocular COS. However, the overall suppression observed is as weak

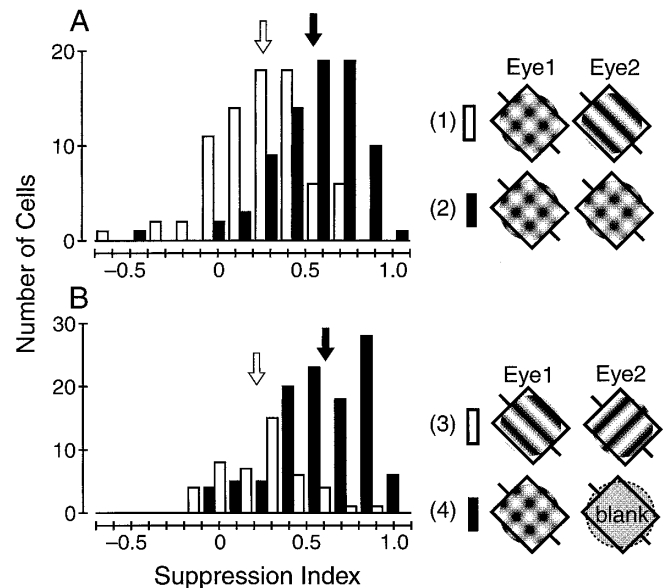


FIG. 11. Histograms of the SI obtained in 4 different COS stimulus configurations: A: from the binocular COS experiment, these values were obtained for conditions in which the cell was stimulated binocularly by an optimal grating and masked in one eye [\square : SI = 0.26 ± 0.26 (SD); $n = 80$] or in both eyes [\blacksquare : mean SI = $0.55, \pm 0.25$ (SD); $n = 80$]. B: unfilled bars are from the interocular suppression experiment; masking and excitatory gratings presented to different eyes [mean SI = 0.22 ± 0.25 (SD); $n = 46$]. Filled bars represent SI obtained from monocular COS in which the excitatory and masking gratings are presented to the same eye [mean SI = 0.62 ± 0.26 (SD); $n = 109$]. Arrows above the histogram denote the mean of each condition.

as interocular suppression alone. Perhaps the extra excitation due to binocular stimulation is enough to swamp most of the suppression in the monocular channel.

DISCUSSION

Binocular suppression

Previous work on COS focused primarily on monocular properties and showed that monocular COS is a robust and ubiquitous property of primary visual cortex (Bonds 1989; DeAngelis et al. 1992; Green et al. 1996; Morrone et al. 1982; Petrov et al. 1980). Our current study demonstrates that this also applies to binocular COS. Contrary to a reasonable expectation, we find that binocular COS is generally not affected by the relative disparity of the orthogonal grating. Given that binocularly stimulated cells in the cat's striate cortex can exhibit strong disparity tuning (Barlow et al. 1967; Ferster 1981; Ohzawa et al. 1986a,b) and that disparity can modulate suppression in area MT of awake behaving monkeys (Bradley et al. 1995), it is somewhat surprising that disparity is not a factor in determining the strength of COS.

Psychophysically, it has been shown that a drifting plaid pattern can be made to appear transparent by introducing disparity between the two components (Adelson and Movshon 1984; von Granau et al. 1993). If striate cells were suppressed by different amounts, depending on the relative disparity of the optimal and mask gratings, they could play a role in this percept. Since this did not turn out to be the case, it appears that there is no interaction between the orientation and disparity channels at the level of the striate cortex. The response of the complex cell in Fig. 3B is intriguing though. This cell could be part of a small population which receives inhibition from disparity sensitive cells, and maybe only a small number of such neurons are needed to facilitate the percept of transparent gratings in different depth planes. It is interesting that this particular cell also exhibited a great deal of binocular facilitation with excitatory stimuli. The monocular responses were only slightly above spontaneous levels (see Fig. 3B), but when stimulated binocularly at the appropriate disparity, the optimal response (R_{opt}) was greatly enhanced. Perhaps cells such as this one, whose response is readily enhanced by binocular stimulation are apt to be more sensitive to other binocular-disparity factors. To account for our observation that disparity does not mediate binocular COS in the remainder of our cell population, the suppressive mechanism must be either insensitive to disparity or it must pool a large number of cells which span the entire disparity range.

Interocular suppression

Results from previous studies of interocular COS (DeAngelis et al. 1992; Ferster 1981; Freeman et al. 1987; Sengpiel et al. 1994, 1995) are somewhat mixed, but it has been generally observed that interocular suppression is weak, if present at all. We confirm here that interocular COS is generally modest but present for nearly all cells (average suppression = $19.9 \pm 29\%$). The actual degree of interocular COS depends on factors such as the contrast of the masking grating. The eye (dominant or nondominant) which is presented with the masking grating is also relevant. For example, in the cells shown in Figs. 6 and 7, when the orthogonal

grating is presented to the nondominant eye, there is no suppression. However, there is considerable suppression when it is presented to the dominant eye. The fact that earlier studies of interocular COS typically placed the suppressive stimulus in the nondominant eye may account for the slightly weaker suppression previously reported.

Another result from this experiment is that temporal onset asynchrony between the optimal and orthogonal gratings does not affect the strength of suppression. With respect to the data of Sengpiel and colleagues (1994, 1995), it should be noted that the distribution of percent suppression observed in our study (Fig. 8A) is similar to the distribution of their data (see Fig. 1a in Sengpiel and Blakemore 1994, and Fig. 6 in Sengpiel et al. 1995). However, it is not clear that they tested all of their cells with simultaneous onsets, and the fact that we found the same suppression at simultaneous onset as with 5-s delays suggests that if they tested all their cells with simultaneous onset, they would have found the same results. Additionally, they did not restrict the stimulus size to the classical excitatory RF center, and it has been shown that surround inhibition can be mediated interocularly (DeAngelis et al. 1994), although surround inhibition is typically weaker at orthogonal orientations. Furthermore, in the reports of Sengpiel et al. (1994, 1995), firing rates are compared for two consecutive 5-s periods. If these cells were adapted by the excitatory stimulus during the first 5-s, the decreased response in the second 5-s period might erroneously be attributed to interocular suppression. Concordantly, suppression would appear weaker for the simultaneous onsets of optimal and mask stimuli because the cell was not adapted yet. In our study, we always compared the interocular response with the cell's response to the optimal stimulus after the same duration of prior excitatory stimulation. We also restricted the stimuli to the central excitatory RF.

Neural circuitry and the origin of suppression

One of the characteristics of cross-orientation suppression that we have attempted to determine is whether the suppression is a monocular or binocular phenomenon. From our data, we conclude that it is predominantly a monocular mechanism. If the mechanism were binocular, equal suppression should be obtained when the mask is presented to either eye. Figure 11B shows that the suppression is weaker when the mask and optimal grating are presented to separate eyes. Furthermore, with binocular excitation, a monocular mechanism should produce stronger suppression when both eyes are masked compared with a monocular mask. Indeed, Fig. 11A shows that there is an increase in overall suppression with binocular masking.

One possible interpretation of our results involves a contrast normalization mechanism (Carandini et al. 1998; Heeger 1992), which acts prior to the combining of the two input signals from the left and right eyes. But what is the actual neural substrate for this mechanism? Our data are compatible with previous work which suggests that monocular COS arises from within the receptive field, is not tuned for orientation (DeAngelis et al. 1992; Ferster 1987), and is broadly tuned for spatial frequency (Bonds 1989; DeAngelis et al. 1992; Morrone et al. 1982). Considering these properties, one is tempted to infer that lat-

eral geniculate afferents mediate suppression, since they form an ideal substrate with respect to the monocular, nonoriented nature of suppression. In particular, one can ask whether geniculate afferents provide the cortex with a raw excitatory signal that is subsequently normalized in the visual cortex or if the LGN output is already normalized. Several points of evidence suggest that the bulk of the suppression does occur within visual cortex. First, it appears that geniculate afferents make only excitatory synapses (Garey and Powell 1971; Stone 1972) which would require at least one intermediate cortical neuron. Second, Bonds (1989) did not find COS in geniculate afferents measured in cortex. It must be noted though, that only five LGN fibers were recorded and there are technical difficulties associated with measuring COS in LGN afferents. This is because any stimulus designed to produce suppression will also excite the cell since center/surround RFs respond to bars or gratings of any orientation. Furthermore, the relative phase differences between the orthogonally oriented stimuli will affect the response. Third, Bonds (1989) also found that the temporal frequency band-pass of suppression was more closely matched to striate neurons as opposed to geniculate cells. Fourth, Morrone et al. (1982) found that for both simple and complex cells, presentation of a phase-reversing orthogonal grating caused a frequency doubled modulation of response to an excitatory stimulus. The inference is that cortical complex cells must be providing the suppression, although a pool of LGN cells could provide frequency-doubled responses to counter-phase gratings as well. Fifth, Morrone et al. (1987) were able to extinguish the visual evoked potential (VEP) signal of COS with cortical application of the γ -aminobutyric acid antagonist, bicuculline. Finally, a normalized signal should be accompanied by a saturating contrast response function, and the LGN responds roughly linearly to contrast increases (Ohzawa et al. 1985; Sclar et al. 1985), implying that lateral geniculate afferents do not carry a normalized input to the cortex.

Collectively, the evidence described above establishes strong evidence that the suppressive mechanism resides in visual cortex. Still, it seems inefficient to pool cortical units to arrive at a source of suppression which so closely resembles the initial LGN input. Additionally, if the mechanism is truly monocular, as our data suggest, it creates a puzzling dilemma since the mechanism is limited to the cortical input layers IV and VI. There is anatomic and physiological support for inhibitory neurons acting in layer IV (Kisvarday et al. 1983, 1985; Martin, 1988; Martin et al. 1983; Somogyi et al. 1983, 1986), but the paucity of monocular neurons is troubling. Estimates of the number of monocular cells in layer IV of area 17 range from 20 to 40% (Blakemore and Pettigrew 1970; Hubel and Wiesel 1962; Macy et al. 1982; Shatz and Stryker 1978). However, it is not necessary for the excitatory units to go through a monocular stage, as long as the inhibitory units are monocular. Encouragingly, two clutch cells studied in layer IV by Martin et al. (1983) were predominantly monocular. Thus the clutch cells appear as ideal candidates to mediate not just COS but also the general suppression associated with a normalization mechanism. Therefore, we suggest that there may be an initial, monocular normalization acting in layer IV prior to the additional processing in other layers.

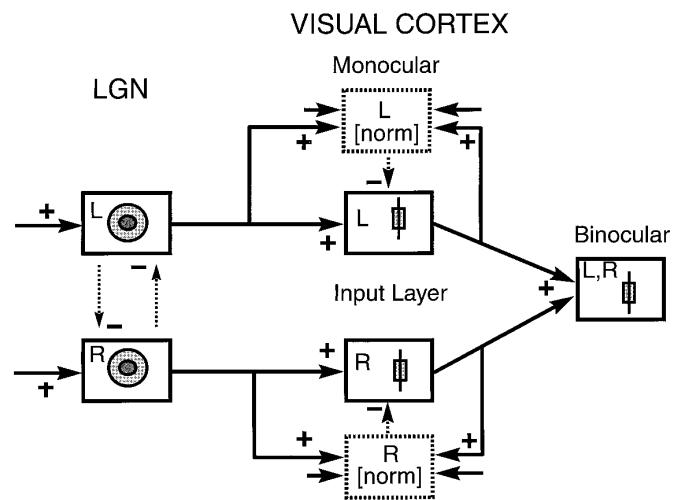


FIG. 12. A model of cortical processing which incorporates a monocular normalization mechanism to account for COS. Weak interocular suppression has been shown to exist in the LGN, and we propose that this may represent the main source of interocular COS observed in cortical units. Monocular signals arrive in the visual cortex and are subjected to a normalizing operation. We assume that this occurs in the input layers of the visual cortex. After normalization, the monocular signals converge to form binocular units. This diagram depicts all cortical units as monocular at the normalization stage, but an alternative model can be made in which only the normalizing units are monocular. This drastically reduces the number of monocular cells needed. However, it requires that the monocular normalizing units act on the monocular afferents from the LGN, or on the dendrites of cortical binocular cells prior to the convergence of the signals from the 2 eyes.

Regarding the interocular COS we observed, it could be demonstrated consistently for many neurons, yet it was usually weaker than monocular COS. One explanation for the weak strength of interocular COS is that it originates in the LGN and is simply propagated to visual cortex. Ferster (1987) suggested a geniculate origin for monocular COS, although given the strength of monocular COS and the reasons outlined above, it is unlikely that the LGN is the sole source. However, interocular suppression is weak in visual cortex and has been shown to exist in the LGN for stimuli of the same orientation (Moore et al. 1992; Varela and Singer 1987; Xue et al. 1987). Our current data show a striking resemblance to those obtained from the LGN. In particular, see Fig. 5 in Xue et al. (1987) and compare with Varela and Singer (1987) and Moore et al. (1992). Thus, it is possible that the interocular effect we have observed originates in the LGN, while the monocular effect has a cortical site of action. Alternatively, if the clutch cells of layer IV are not completely monocular, some of the suppression might transfer from the other eye. Note that the only way to avoid interocular COS while still retaining a cortical origin for monocular COS is for the site of suppression to be prior to the combination of the two monocular signals. If the suppressive mechanism is binocular (irrespective of disparity issues), it will confer some degree of interocular suppression. Additionally, if the mechanism is monocular, but acts after binocular combination, there will be interocular suppression.

Figure 12 illustrates a simple model to explain our results and offer a suggestion for how cross-orientation suppression

may arise in visual cortex. The main feature is monocular normalization in the input layers of visual cortex which can produce strong monocular COS. Figure 12 depicts excitatory units receiving inhibition from monocular normalization units, but we do not imply that the excitatory units are themselves monocular. The excitatory units can be binocular at the input stage, in which case the normalization would likely occur on dendrites postsynaptic to the lateral geniculate input, but prior to the convergence of monocular signals from the two eyes. If the excitatory units are monocular, the suppression can be mediated by axo-somatic synapses from smooth stellate cells. The model also includes weak binocular suppression in the LGN, which may account for the interocular COS we observed.

Summary

Cross-orientation suppression is a curious phenomenon that violates the linearity of the receptive field because the addition of an orthogonal mask, which does not elicit a response on its own, can suppress the response to an optimal grating. In this study, we have shown that strong suppression can be obtained if an optimal stimulus is masked with an orthogonal grating. This is true for both monocular and binocular presentations. However, if the mask is presented to one eye and the optimal stimulus is presented to the other eye or to both eyes, the suppression is much weaker. Furthermore, the strength of suppression is not affected by introducing disparity or temporal presentation asynchronies. Considered together, these results indicate that the suppressive mechanism acts prior to the combination of signals from the left and right eyes.

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