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No Binocular Rivalry in the LGN of Alert Macaque Monkeys

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Orthogonal drifting gratings were presented binocularly to alert macaque monkeys in an attempt to find neural correlates of binocular rivalry. Gratings were centered over lateral geniculate nucleus (LGN) receptive fields and the corresponding points for the opposite eye. The only task of the monkey was to fixate. We found no difference between the responses of LGN neurons under rivalrous and nonrivalrous conditions, as determined by examining the ratios of their respective power spectra. There was, however, a curious "temporal afterimage" effect in which cell responses continued to be modulated at the drift frequency of the grating for several seconds after the grating disappeared.

Binocular vision Rivalry Macaque monkey Lateral geniculate nucleus

INTRODUCTION

Despite numerous investigations which have established a detailed knowledge about many aspects of the anatomy and physiology of the lateral geniculate nucleus, the function of that structure is still unknown. In this study we shall investigate the possibility of lateral geniculate nucleus (LGN) involvement in binocular vision. In particular, we are interested in examining the LGN of alert monkeys for neural correlates of binocular rivalry, a psychophysical effect that has been extensively studied in humans.

Binocular rivalry occurs when nonmatching stimuli are presented to the two eyes, such as a vertical grating to the left eye and a horizontal grating to the right eye. Under this condition, the stimuli to the two eyes do not fuse to form a plaid. Rather, the visual system is thrown into oscillations, so that the percept switches back and forth between the inputs to the two eyes, with a mean period of several seconds. The phenomenon has been reviewed by Lehky (1988) and Blake (1989), and there is a substantial body of quantitative human psychophysical data related to it. The work of Levelt (1965) is seminal. It is known that monkeys, as well as humans, do indeed experience rivalry, as indicated by their perceptual choices in a motion discrimination task (Logothetis & Schall, 1990). By stressing the LGN with rivalrous stimuli, we hoped to elicit responses that might establish whether it contributes to binocular processing. Aside from serving as a probe for LGN function, the physiological mechanisms underlying rivalry are relevant to those interested in the perceptual phenomenon in its own right, and have also attracted the attention of people involved in issues related to visual awareness (Crick & Koch, 1992).

Several investigators have suggested an LGN locus for binocular rivalry (Blakemore *et al.*, 1972; Lehky, 1988; Lehky & Blake, 1991; Singer, 1977). It is attractive as the site for rivalry for two reasons:

1. Inputs from the two eyes are segregated in separate laminae, which allows the signal from one eye to be selectively suppressed; and
2. It receives a substantial feedback from striate cortex (V1), which could provide a control signal indicating whether stimuli are binocularly fused or not.

These two features have been combined into a model of rivalry (Lehky, 1988), and the argument for an LGN locus is set forth in greater detail in Lehky and Blake (1991).

Binocular interactions, predominantly inhibitory, have been widely reported in cat LGN (Sengpiel *et al.*, 1995; Guido *et al.*, 1989; Moore *et al.*, 1992; Murphy & Sillito, 1989; Pape & Eysel, 1986; Rodieck & Dreher, 1979; Sanderson *et al.*, 1971; Schmielau & Singer, 1977; Singer, 1970; Suzuki & Kato, 1966; Tong *et al.*, 1992; Xue *et al.*, 1987). Such binocular inhibition in the LGN could play a role in producing rivalry. Possible pathways for the interactions are cortical feedback, perigeniculate feedback, or dendrites of interneurons which extend between laminae (Singer, 1977). Binocular interactions have also been reported in monkey LGN (Marrocco & McClurkin, 1979; Rodieck & Dreher, 1979; Schroeder *et*

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al., 1990), though there is disagreement about how widespread they are.

Moving to a consideration of the cortical feedback to LGN, studies of its anatomical organization include those by Gilbert & Kelly (1975), Holländer & Martinez-Millan (1975), Lin & Kaas (1977), Robson (1983) and Spatz *et al.* (1970). In cats, this feedback seems to be numerically the dominant input, exceeding retinal afferents by an estimated factor of ten (Sherman & Koch, 1986). In monkeys, the feedback is relatively smaller (Wilson, 1989), but probably still larger than the retinal input.

The functional role of cortical feedback on LGN activity has been assessed by both cortical ablation and cooling, generally showing weak and inconclusive effects. This seems surprising given the size of the cortical feedback, although Koch (1987) speculates why this may be the case. Functional studies in cats include those by Geisert *et al.* (1981), Gulyas *et al.* (1990), Kalil & Chase (1970), Richard *et al.* (1975), Schmielau & Singer (1977), Tsumoto *et al.* (1978) and Vidyasagar & Urbas (1982). Work from Sillito and colleagues (Murphy & Sillito, 1987; Sillito *et al.*, 1993, 1994) provide data showing more clear-cut corticofugal effects in cat LGN. Monkey studies include Baker & Malpeli (1977), Hull (1968), Marrocco *et al.*, (1982), McClurkin & Marrocco (1984), and McClurkin *et al.*, (1994).

Speculations concerning the function of cortical feedback to the LGN center on the notion that it is performing a gating or gain control function in the transmission of information from retina to cortex (see, for example, Ahlsén *et al.*, 1985; Sherman & Koch, 1986; Singer, 1977). An LGN involvement in binocular rivalry would be compatible with such a gating function. An interesting and important variant of the 'gating' hypothesis is the idea that the feedback is involved in selective, location-based attention [the searchlight of attention, as Crick (1984) describes it]. Others have emphasized feature-based rather than location-based selection of information. Along these lines is the suggestion that corticofugal feedback provides top-down information about models, hypotheses, or constraints concerning the external world generated at higher levels (Harth *et al.*, 1987; Mumford, 1991; Sillito *et al.*, 1994). This leads to synthesis of the visual world by mutual enhancement between sensory inputs and higher-level hypotheses that support each other, and attenuation of irrelevant or incompatible features.

There have been a number of previous studies searching for the physiological basis of binocular rivalry. Varela and Singer (1987) reported a neural correlate of rivalry in the LGN of anesthetized cat, but this could not be replicated by Sengpiel *et al.*(1995) working with a similar preparation. Dobbins *et al.* (1994), Logothetis and Schall (1989), as well as Sengpiel *et al.* (1995) have all reported what may be neural correlates of rivalry in various areas of cortex, though cortical work is still in its early stages and it is still too early to form firm conclusions. In any case, rivalry in cortex could reflect responses generated at the level of the LGN. There have been no investigations of binocular rivalry in the monkey LGN, nor in the LGN of alert animals of any species.

METHODS

Animal preparation and recording procedure

Recordings were made from the dorsal LGN of two alert monkeys (a female *Macaca nemestrina* and a male *M. fascicularis*). Initial surgery implanted a stainless steel headpost, and also a scleral eye coil for monitoring eye position (Judge *et al.*, 1980; Robinson, 1963). After the monkeys learned their task, a second surgery was performed to open a 2 cm craniotomy and implant a stainless steel recording chamber around it. The craniotomy was directly dorsal to the LGN. All surgery was conducted under aseptic conditions while the animals were under deep isoflurane anesthesia.

Tungsten microelectrodes with paralene insulation and a polyimide outer sheath were used (Micro Probe Inc., Clarksburg, MD, U.S.A.). The electrodes were positioned using a plastic grid inserted in the recording chamber (Crist *et al.*, 1988). A guide tube was pushed through the grid at the desired location, until its tip was located ~7 mm above the LGN. The electrode was then lowered through the guide tube. Visual responsiveness of neural activity as the electrode descended through brain tissue was monitored using a hand-held light source. When LGN layer 6 was reached, the receptive field location was mapped while the monkey viewed a fixation spot on the computer monitor. At this point, the stimulation was switched to the grating stimuli described below. Recording sites could be assigned unambiguously to individual layers within the LGN based on physiological criteria, including the characteristic alternation of the dominant eye from layer to layer and differences between the temporal frequency selectivities of magnocellular and parvocellular cells (Schiller & Malpeli, 1978). Electrode penetrations through the LGN were not reconstructed histologically.

Stimulus conditions and behavioral procedure

The only task of the monkeys was to maintain fixation while binocular rivalry stimuli or other control stimuli were presented within a unit's receptive field, usually 5–10 deg from fixation. Human psychophysical studies indicate that rivalry occurs at those eccentricities (Blake *et al.*, 1992). The monkeys had restricted access to water and worked for a juice reward. Body weight was monitored daily to insure that liquid intake was adequate, and the animals received free water at weekends.

The simple task we chose had the advantage of being quick to train, and the disadvantage of offering no positive behavioral support connecting any putative neural rivalry activity with the psychological phenomenon (sharing this disadvantage with anesthetized preparations). However, if there was neural activity that could plausibly be related to rivalry, we had the option of moving to a more complex task to confirm that.

TABLE 1. Description of the seven stimulus conditions presented to each unit

Cond.	Name	Dominant orientation	Nondominant orientation	Dominant contrast	Nondominant contrast
1	Rivalry	45	135	1.0	1.0
2	Matching	45	45	1.0	1.0
3	Dominant mono.	45	—	1.0	0.0
4	Rivalry (low contrast)	45	135	0.5/0.2*	1.0
5	Dominant mono. (low contrast)	45	—	0.5/0.2*	0.0
6	Nondominant mono.	—	135	0.0	1.0
7	Blank	—	—	0.0	0.0

"Dominant" and "nondominant" refer to the ability of each eye to physiologically drive an individual neuron under monocular conditions, and not the perceptual state of the monkey during binocular rivalry.
*Low contrast: 0.5 for parvocellular units, 0.2 for magnocellular units.

The monkeys viewed the computer display monitor through a Wheatstone stereoscope, arranged so that half the screen was devoted to the stimulus for each eye. The stereoscope mirrors were aligned for each animal so that the visual fields of the two eyes were in register. This was done by switching display of a fixation spot back and forth between the two eyes, and adjusting the mirror angle until there was no change in eye position when the fixation spot switched eye. This was repeated with the fixation spot located at three noncolinear points in the visual field. During data collection, fixation spots were always visible to both eyes.

The stimuli were drifting sinusoidal gratings, confined within a circular aperture 6 deg in diameter. Their spatial frequency was usually 1.0 c/deg, and temporal frequency was 2.0 c/sec. No great effort was made to optimize the stimulus for each unit. The gratings were usually grayscale, but color stimuli were sometimes used if they were preferred by the neuron. In either case, stimuli were displayed on a background with the same mean luminance. Color look-up tables for the display monitor were calibrated to provide a linear luminance response. Mean luminance of the screen was 40 cd/m^2. Whenever we found a candidate neuron for recording, a grating was centered over its receptive field. During binocular stimulus conditions, a second grating was displayed at the corresponding position in the visual field of the nondominant eye.

Stimulus duration was 5 sec. The monkey was required to maintain fixation for 500 msec before and after the stimulus presentation, so that each trial lasted a total of 6 sec. The inter-trial interval was 1 sec. A trial was aborted if the monkey's eye position moved further than 0.75 deg from the center of the fixation window at any time.

Seven stimulus conditions were presented to each neuron, as listed in Table 1. The rivalry condition used orthogonal drifting gratings, the matching condition had gratings at the same orientation, and there were monocular and blank controls. "Blank" means the screen was held at mean luminance and not darkened. There were also "low contrast" rivalry and monocular conditions. Low contrast was defined as 0.5 for parvocellular units and 0.2 for magnocellular units, a lesser contrast for magnocellular units because they have greater contrast sensitivity (Derrington & Lennie, 1984; Kaplan & Shapley, 1982; Shapley *et al.*, 1981). Reducing the contrast of the dominant eye stimulus was an attempt to increase the chances that the LGN response would be suppressed by a high contrast grating presented to the nondominant eye. ("Dominant" and "nondominant" eye refer here to the physiological ability to drive an LGN cell, and not the perceptual state of the animal.) From human psychophysics (Levelt, 1965), it is known that reducing contrast to one eye during rivalry increases the fraction of time that eye is suppressed. The entire set of seven conditions was repeated twenty times for each neuron, with the seven conditions presented in random order within each repetition. In some cases it was not possible to hold a unit long enough for all 20 repetitions, and any data set with at least 15 repetitions was accepted for analysis.

Data analysis

Analysis focused on power spectra of neural responses rather than peristimulus time histograms. This was because the timing of binocular rivalry effects within each trial was expected to be random with respect to stimulus onset time. Therefore, pooling data from multiple trials in a PSTH would tend to obscure rivalry effects rather than enhance them. Since the power spectrum throws away phase information, it is possible to usefully average power spectra of nonphase-locked responses over multiple stimulus repetitions. Binocular rivalry oscillations would be expected to be at very low frequencies, in the range of 0.2–0.4 Hz.

In calculating single-unit power spectra, there were two possible ways of pooling data from individual trials:

1. Calculate the power spectrum for each trial, and average the spectra together.
2. Average the PSTHs first, and then calculate a single power spectrum from that.

We used the first method, because it enhances responses which are not phase-locked to stimulus onset. The second method would have emphasized responses which are phase-locked.

For each unit, after power spectra for the seven stimulus conditions were calculated, they were normalized so that the tallest peak (over all seven conditions)

TABLE 2. Pooled responses for 41 parvocellular and 23 magnocellular units

Cond.	Name	Parvocellular		Magnocellular	
		Relative peak response peak	Mean response (spikes/sec)	Relative peak response	Mean response (spikes/sec)
1	Rivalry	0.99	44	1.00	51
2	Matching	0.96	45	1.00	51
3	Dominant mono.	1.00	44	1.00	51
4	Rivalry (low contrast)	0.43	40	0.50	49
5	Dominant mono. (low contrast)	0.44	42	0.46	49
6	Nondom. mono.	0.04	39	0.03	44
7	Blank	0.03	37	0.04	45

"Peak response" indicates relative peak heights of the power spectra at the 2.0 Hz stimulation frequency. "Mean response" indicates mean firing rate over the entire 5 sec stimulus presentation. Data for the "low contrast" parvocellular and magnocellular conditions are not directly comparable because different low contrasts were used for the two groups. Magnocellular and parvocellular data were independently normalized.

had a height equal to 1.0. Then, all the normalized single-unit spectra from different units were averaged together to give a population power spectrum for each stimulus condition. Since responses for the different stimulus conditions were not normalized separately, relative peak height across different conditions can be compared.

Although each single-unit power spectrum had a maximum peak of 1.0, the population power spectrum formed by averaging them had a peak of <1.0. This happened because the maximum response was not at the same frequency for every unit. (For example, maximum response could occur at the stimulus frequency for one unit, and at the video frame rate for another unit. Averaging the spectra of these two units leads to peaks of <1.0 at both frequencies.) The data in Table 2 have been renormalized to account for this. The spectra plotted in Fig. 2 have not been renormalized, and therefore all have peaks <1.0.

It is interesting to note that a high frequency signal, such as the video frame rate, can be visible in the power spectrum of a spike train even when the spike rate appears too low to transmit such high frequencies. Contributing here in some cases is the existence of short pockets of high spikes rates, which do not appear in PSTHs because of various smoothing procedures typically used when calculating spike rate. Also, pooling data from multiple trials allows one to see high-frequency modulations in a low-frequency spike train caused by probabilistic tendencies for increased or decreased firing during certain periods.

RESULTS

We recorded from 41 parvocellular units and 23 magnocellular units from the dLGN of one monkey, and 11 parvocellular and 4 magnocellular units from the second. Results from both animals were essentially the same. No differences in binocular responsiveness were seen with units tested at different spatial frequencies and colors, and those data are pooled in the power spectra shown below.

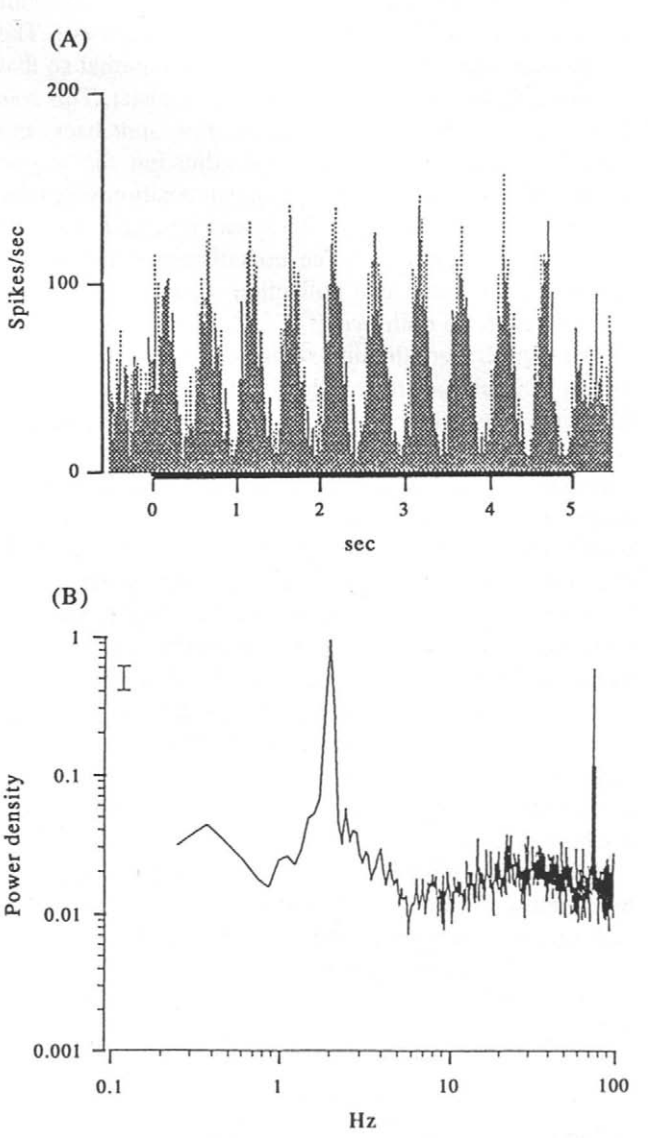

FIGURE 1. (A) Peristimulus time histogram (PSTH) showing response of a parvocellular unit to a grating drifting at 2.0 Hz. The stimulus was presented during the time between 0 and 5 sec. The histogram is based on 20 repetitions of a binocular grating stimulus, with identical orientations presented to the two eyes (Condition 2). (B) Power spectrum calculated from the data in the PSTH in part A of this figure. Notable are peaks at 2.0 Hz in response to the grating, and at 75 Hz, which is the frame rate of the display monitor. The small marker near the y-axis represents one standard error in this and subsequent diagrams of power spectra.

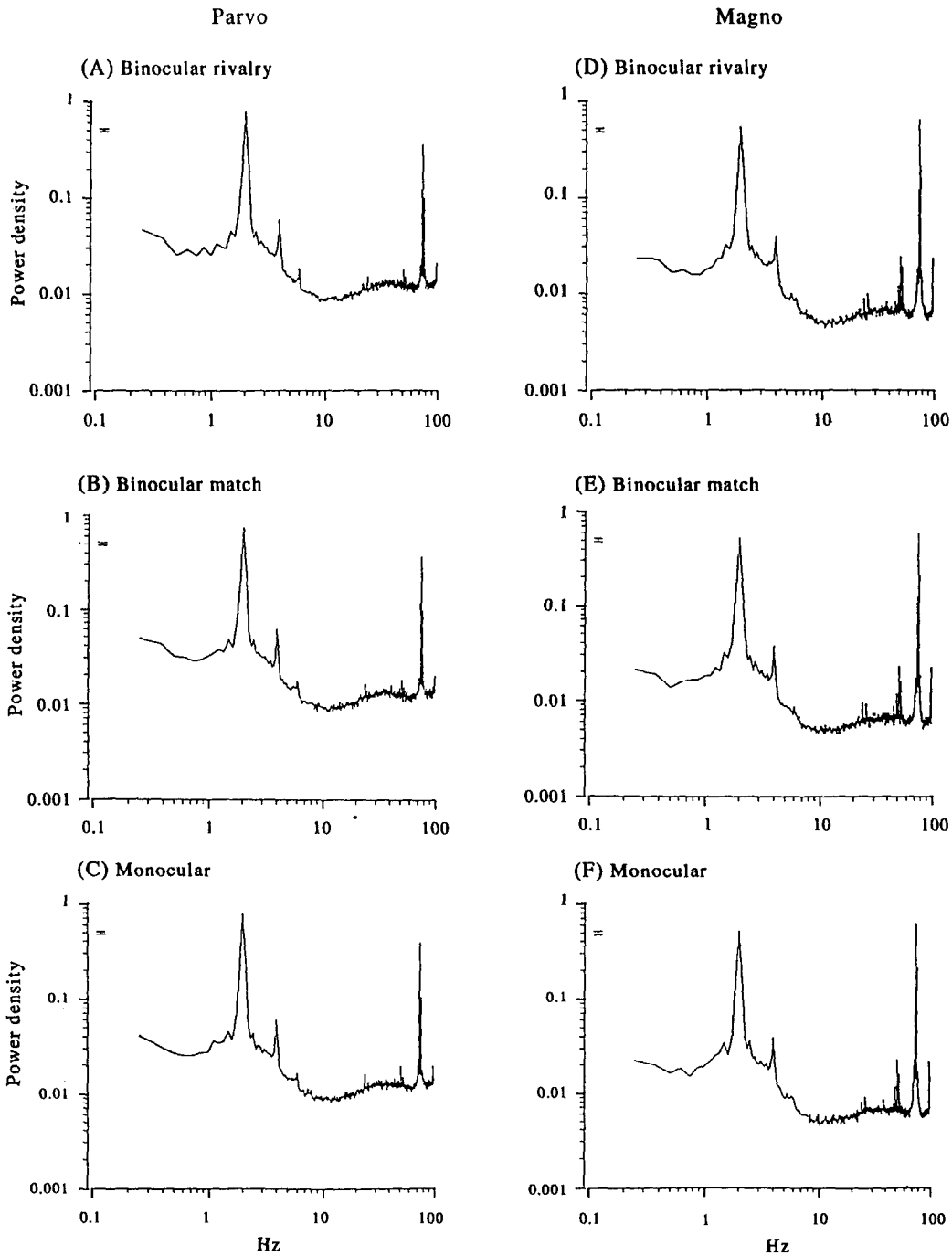

FIGURE 2. Average power spectra under three stimulus conditions: (A) binocular rivalry, (B) binocular matching, and (C) monocular stimulation to the dominant eye. Left column shows spectra for parvocellular units ($n = 41$), and right column shows spectra for magnocellular units ($n = 23$). There is no apparent difference among the three conditions.

Figure 1(A) is a PSTH from an example parvocellular unit in response to a drifting sine-wave grating. It shows a 2.0 Hz sinusoidal temporal modulation corresponding to the drift frequency of the grating. Figure 1(B) shows the power spectrum calculated from the data in the PSTH of Fig. 1(A). As expected, a major peak occurs at 2.0 Hz. Another peak occurs at 75 Hz, which is the frame rate of the display monitor. If the PSTH in Fig. 1(A) is plotted with an expanded time scale, this 75 Hz modulation clearly shows up superimposed on the 2.0 Hz modulation. Frame rate modulation was always observed in the responses of parvocellular and magnocellular LGN neurons, though there was a great degree of variability in its size.

Table 2 shows average responses from parvocellular and magnocellular units in one monkey, for all seven stimulus conditions. The table indicates relative peak heights of the power spectra at the 2.0 Hz stimulation frequency, as well as mean spike rates over the entire 5 sec stimulus period. It can be seen that signal power of

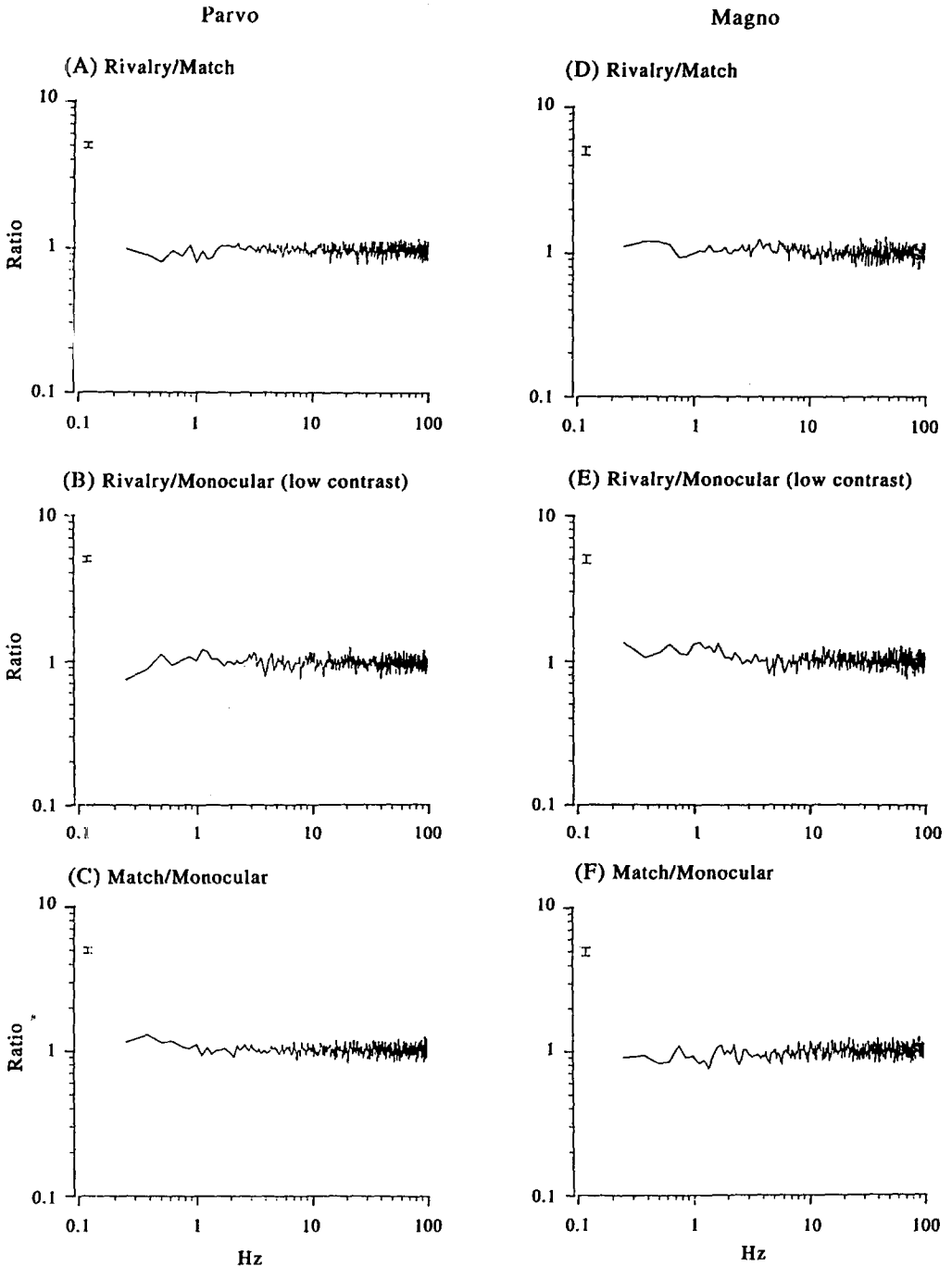

FIGURE 3. Power spectrum ratios calculated from power spectrum curves such as those shown in Fig. 2. The left column (A–C) shows parvocellular data: (A) Ratio of binocular rivalry/binocular matching (Condition 1/Condition 2). (B) Ratio of binocular rivalry/dominant monocular (Condition 4/Condition 5). (C) Ratio of binocular match/dominant monocular (Condition 2/Condition 3). [In (B), grating contrast to the dominant eye was low (0.5). During rivalry, low contrast to one eye increases the probability of suppression by the high contrast grating to the other eye.] The right column (D–F) shows the corresponding power spectrum ratios for magnocellular data. Markers near the y-axis indicate one standard error of the power spectrum ratio. For those ratios involving rivalry, Student t-tests were performed at frequencies where a rivalry effect would be expected to be strongest, at 0.25 and 2.0 Hz. These showed no significant differences ($P > 0.01$) from a ratio of 1.0 (i.e., no effect). Given the size of the error bars, there is no suggestion of statistically significant differences at other frequencies either.

neural activity at the stimulus frequency increases greatly as a function of contrast to the dominant eye, by a factor of about 30. On the other hand, mean activities increase only slightly (~15%) going from zero to full contrast. This indicates that the response signal is primarily carried by modulation of activity about a level close to the spontaneous firing rate, an aspect of the response which may be apparent upon inspection of the PSTH of Fig. 1(A).

Figure 2 shows average power spectra for all magnocellular and parvocellular units from one monkey under three different stimulus conditions. The three

parvocellular plots have the same vertical scale, as do the three magnocellular plots. In both sets of plots there is prominent power at 2.0 Hz and at the video frame rate. Far less power (<10%) can be seen at harmonics of the grating frequency, 4.0 and 6.0 Hz. The small size of these harmonics is in accord with earlier reports that LGN cells have very linear responses (Derrington & Lennie, 1984; Kaplan & Shapley, 1982). Figure 2 also shows that parvocellular units are less responsive than magnocellular ones at the 75 Hz frame rate, again in accord with a previous observation indicating a lower temporal cutoff frequency for parvocellular units.

A small peak of activity is visible at 50 Hz in the spectra for magnocellular units. This activity was virtually eliminated when low contrast or blank (unpatterned mean luminance) stimuli were presented to the dominant eye (not shown in Fig. 2). Another feature of the 50 Hz response was that it was phase-locked to the 75 Hz video frame rate. We suspect the 50 Hz signal is a subharmonic of the frame rate because their frequencies form a ratio of small integers and because they are phase-locked. Possibly, this subharmonic artifact becomes prominent only against a background of high spike rates produced by a strong stimulus. In addition, the signal might be more visible in magnocellular units than parvocellular ones because magnocellular units respond more vigorously at the frame rate. Ghose and Freeman (1992) report a prominent oscillation at ~50 Hz in the LGN of anesthetized cat, which might be related to the one observed here. They found that the strongest oscillations were predominantly in Y cells rather than X cells. Cat Y cells are thought to be analogous to the monkey magnocellular units.

One additional noteworthy feature of the spectra in Fig. 2 is the broad, shallow hump of activity centered at 40 Hz and ranging from about 20–60 Hz. This hump may reflect small, intrinsic neural oscillations of the sort that have recently been of interest in connection with global aspects of visual processing, as reviewed by Singer *et al.*, (1990) as well as Llinás and Ribary (1994). The present data do not influence any of these theories one way or the other.

Moving on to the central concern of this study, a comparison of power spectra for the three conditions in Fig. 2 (binocular rivalry, binocular matching, and monocular stimulation) shows no appreciable differences among them, either for parvocellular or magnocellular units. This can be examined more closely by plotting power spectrum ratios for different stimulus conditions (Fig. 3). The ratio of power spectra binocular rivalry/binocular matching [Fig. 3(A) for parvocellular and Fig. 3(D) for magnocellular] stays flat at close to 1.0 for all frequencies (deviations not significant at $P = 0.01$, under a Student t-test), indicating no difference between the two conditions. If there had been a neuronal correlate of rivalry, we would have expected the ratio to be depressed at around 2.0 Hz, since the grating stimulus would have been suppressed a substantial fraction of the time. Also, the ratio would have been elevated in the range 0.2–0.4 Hz, the band in which rivalry oscillations occur. There was also no effect when the grating to the dominant eye had low contrast (as defined in Table 1) and was therefore more likely to be suppressed by the high contrast grating to the other eye [see Fig. 3(B) and (E)]. Finally, Fig. 3(C) and (F) show no difference in the responses between binocular matching and monocular conditions. These are pooled data for multiple units, but examination of data from individual units did not reveal anything different. From these results we conclude that there is no evidence for a neural correlate of binocular rivalry in the LGN.

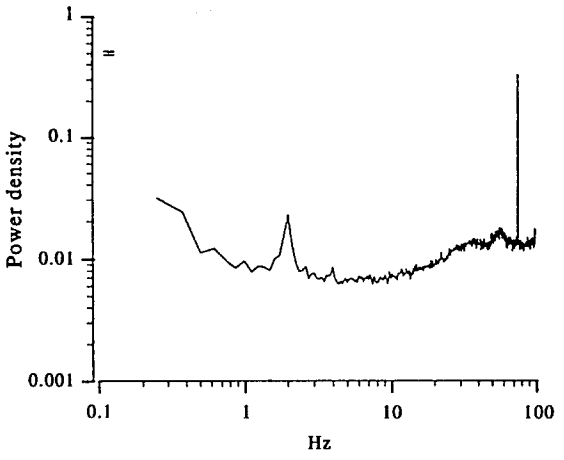

FIGURE 4. Demonstration of a "temporal afterimage" effect. This is the average power spectrum for 41 parvocellular units, calculated for a binocular blank screen condition which was randomly interspersed among other conditions in which a grating drifting at 2.0 Hz was presented. The peak at 2.0 Hz indicates that the unit continued to oscillate weakly at the stimulus frequency for several seconds even after the stimulus was removed. Magnocellular units showed the same effect.

An unexpected observation, unrelated to binocular rivalry, was something we call the "temporal afterimage", which appeared in both magnocellular and parvocellular units. As a control, a blank screen condition (blank to both eyes) was randomly interspersed among the grating stimuli during the experiment. Oddly, the power spectra of the neuronal responses to a blank screen showed a peak at 2.0 Hz (Fig. 4), which was the temporal frequency of the grating used in the other trials. The peak is small, only a few percent of the activity produced when the stimulus was present, but nevertheless clearly visible. When examined on a trial-by-trial basis, the phase of this spontaneous 2.0 Hz oscillation was randomly scattered over the range of all possible values [that is, it was not phase-locked to the (blank) "stimulus" onset, nor to the grating presented in the previous trial]. This is in contrast to the 2.0 Hz response produced by having a grating present, which was phase-locked to stimulus onset and therefore had the same phase every trial.

To demonstrate that this effect was not an artifact of our equipment or computer programs, we tested them using an "artificial eye" device. It consisted of a photocell connected to a voltage controlled oscillator, which produced a series of pulses ("spikes") at a

frequency proportional to luminance. This device was held against the monitor running the stimulus display program, and the resulting pulses were run through the data acquisition hardware and software as well as the data analysis software, as if an actual experiment were being run. This test invariably showed 2.0 Hz power when a stimulus was present, and none during the blank control trials.

The amplitude of the spontaneous 2.0 Hz oscillations decayed linearly over the course of the 5 sec "blank screen" stimulus period, taking about 3 sec to drop by half. This was determined by breaking the stimulus period into three time segments and calculating the power spectrum for each segment. Recall that our "blank screen" stimulus period, during which data were collected, was preceded by a blank intertrial period of 1.0 sec and blank prestimulus period of 0.5 sec, so the spontaneous oscillations observed already had some time to decay after the end of the grating presentation from the previous trial. We tested whether it was just a coincidence that the spontaneous oscillations and the stimulus were both at 2.0 Hz by changing the frequency of the stimulus from 2.0 to 4.0 Hz when recording from one unit. In this case, the peak of spontaneous activity appeared at that new frequency. Finally, there was virtually no difference in the spontaneous oscillations resulting from a binocular blank "stimulus" and a monocular stimulus to the nondominant eye (and therefore blank to the dominant eye). This was true with respect to both their amplitudes and lack of phase-locking. If we had not known about the responses in the binocular blank condition, those observed during nondominant monocular stimulation might have been mistaken for binocular crosstalk. Figure 4 shows data pooled from 41 units. When one examines data from individual units, the "temporal afterimage" effect is apparent in only about one third of the cases.

DISCUSSION

We found no evidence for a neural correlate of binocular rivalry in the LGN of awake monkeys. This is in agreement with the findings of Sengpiel *et al.* (1995) in the LGN of anesthetized cat, and contrary to the findings of Varela and Singer (1987), also in anesthetized cat. There was no support for conjectures based on psychophysical evidence of a LGN locus for rivalry, as set forth by Lehky and Blake (1991), among others. These findings do not affect the general idea that rivalry involves reciprocal feedback inhibition between left and right signals (Lehky, 1988), but discredits one possible anatomical locus for such a circuit.

That leaves the cortex as the site of rivalry. There have been several studies suggesting rivalry in various parts of cortex. A neural correlate of rivalry has been reported in MT (V5) of behaving monkey to motion stimuli (Logothetis & Schall, 1989) in about 20% of units, although the latency of onset of the putative rivalry was shorter than human psychophysics would indicate. However, any effects observed in MT may be a reflection of rivalry in V1. To selectively suppress the motion signal from one eye would seem to require monocular circuitry of some sort (or at least units that have a strong ocular dominance, even if not pure monocular), and V1 has a much higher incidence of ocular dominance than MT. There is a report of suppression in V1 units under rivalrous stimulus conditions (Sengpiel *et al.*, 1995), but this was done in anesthetized cats and therefore offers no behavioral support connecting this suppression with the psychological phenomenon. In another, preliminary, report, Dobbins *et al.* (1994) have examined V1, V2, and V4 for rivalry in awake monkey and found only a small fraction of units in which suppression correlated with behavioral reports of the monkey. No one has reported any oscillatory behavior, which is one of the hallmarks of rivalry. Overall, neural correlates of rivalry appear far less conspicuous than one might have expected from the dramatic psychological percept, and it may be that direct involvement of only a small fraction of units in any one area is sufficient to produce the perceptual effect.

Leaving aside rivalry, we did not observe binocular interactions of any sort in the LGN [Fig. 3(C) and (F)]. This is different from the results of both Marrocco and McClurkin (1979) and Rodieck and Dreher (1979), who have reported binocular inhibition or excitation in a small fraction of units (around 10–15%) in anesthetized monkey. However, our experimental design was less sensitive than theirs for picking up small effects. We had the stimulus to the nondominant eye either continuously present or continuously absent within a single trial, and thus could only do between-trial comparisons for binocular interactions. They had the nondominant stimulus present intermittently during a trial (a procedure which would not have been suitable for our purposes), and could do more sensitive within-trial comparisons. In addition, their design may have led to more noticeable binocular effects because of transients caused by switching the nondominant eye stimulus on and off within a trial. In other experiments, Schroeder *et al.* (1990) report large and widespread binocular interactions in awake monkey LGN, observing field potentials rather than single unit activity. Possibly their observations were the result of using very brief, structureless, full field flashes as stimuli (again likely to cause transients) rather than the sustained, patterned stimuli we used.

The "temporal afterimage" effect we observed (continued oscillation at the stimulus frequency for several seconds after the stimulus was removed) is of unknown significance, though potentially interesting. A similar effect has been seen in the LGN of cats (Ohzawa, personal communication). Steriade *et al.* (1990, pp. 235–236) also report some examples of this class of behavior in thalamic nuclei. As was mentioned earlier, the aftereffect activity in response to a blank screen could be misinterpreted as a binocular interaction, when using a nondominant stimulus condition.

It would be interesting to know whether the "temporal afterimage" is generated within the LGN, or whether cortical feedback plays an important role. The latter

opens up a broader range of functional possibilities. To give one speculation, if the afteraffect were cortex-dependent, perhaps it represents a signal indicating what the cortex is "looking for" in the sensory input (i.e., the cortex is imposing on the LGN selective filtering based on a match or "resonance" between sensory inputs and higher level expectations). A somewhat related idea is that the affereffect we observed is a short term memory store of the stimulus, perhaps held by reverberating activity between the cortex and LGN along the lines suggested by Koch and Crick (1994). Another question of interest is whether the affereffect mimics the spatial, as well as the temporal aspects of the stimulus. That is to say, do the afteraffect oscillations sweep across the LGN in coherent waves, in the manner of a drifting grating, or are they the product of random, spatially uncoordinated bursts of firing?

For those interested in understanding the microcircuitry involved in generating rivalry, the lack of rivalry in the LGN is unfortunate, because the laminar structure, synaptic glomeruli and feedback loops offer a lot to work with. An understanding of the low-level circuitry underlying neural correlates of psychological phenomena becomes more difficult as one moves up the visual system. On the other hand, for those interested in rivalry as a probe for visual awareness, no rivalry in the LGN may be taken as good news, for they would generally prefer the effect to occur late in the visual system.

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