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Visual Response Latencies in Striate Cortex of the Macaque Monkey

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SUMMARY AND CONCLUSIONS

1. Many lines of evidence suggest that signals relayed by the magnocellular and parvocellular subdivisions of the primate lateral geniculate nucleus (LGN) maintain their segregation in cortical processing. We have examined two response properties of units in the striate cortex of macaque monkeys, latency and transience, with the goal of assessing whether they might be used to infer specific geniculate contributions. Recordings were made from 298 isolated units and 1,129 multiunit sites in the striate cortex in four monkeys. Excitotoxin lesions that selectively affected one or the other LGN subdivision were made in three animals to demonstrate directly the magnocellular and parvocellular contributions. An additional 435 single units and 551 multiunit sites were recorded after the ablations.

2. Most units in striate cortex had visual response latencies in the range of 30–50 ms under the stimulus conditions used. The earliest neuronal responses in striate cortex differed appreciably between individuals. The shortest latency recorded in the four animals ranged from 20 to 31 ms. Comparable values were obtained from both single unit and multiunit sites. After lesions were made in the magnocellular subdivision of the LGN in two animals, the shortest response latencies were 7 and 10 ms later than before the ablations. A larger lesion in the parvocellular subdivision of another animal produced no such shift. Thus it appears that the first 7–10 ms of cortical activation can be attributed to activation relayed by the magnocellular layers of the LGN.

3. The units with the shortest latencies were all found in layers 4C or 6 and their responses were among the most transient in striate cortex. Furthermore, their responses all showed a pronounced periodicity at a frequency of 50–100 Hz. This periodicity was stimulus locked, and the responses of all short-latency units oscillated in phase.

4. An index of response transience was computed for the units recorded in striate cortex. The distribution of this index was unimodal and gave no suggestion of distinct contributions from the geniculate subdivisions. Magnocellular and the parvocellular lesions affected the overall transience of responses in striate cortex. The changes, however, were very small; extremely transient responses and extremely sustained responses survived both types of lesions.

5. A characteristic profile was observed in the response latencies in superficial layers. Latencies appeared to increase monotonically from layer 4 toward the surface of cortex, with the most superficial neurons not becoming active until 15 ms after responses were observed in layer 4C. This large delay is likely to reflect the anatomic organization of the superficial layers and could have important implications for the information processing that occurs in striate cortex.

INTRODUCTION

The physiological differences between the magnocellular and parvocellular subdivisions of the primate lateral geniculate nucleus (LGN) have been extensively studied (reviewed by Lennie 1980; Shapley 1990; Stone 1983). Comparatively little is known about how these subdivisions contribute to differences in neuronal response properties in cortex. Several lines of research suggest that differential magnocellular and parvocellular contributions could account for many of the pronounced differences in response properties that exist between extrastriate visual areas (see DeYoe and Van Essen 1988; Hubel and Livingstone 1987; Maunsell 1987). Unfortunately, direct evidence for the relative contributions of the magnocellular and parvocellular subdivisions to responses of cortical neurons has proven difficult to obtain (see Maunsell et al. 1990). In one of the most direct tests of LGN contributions, Malpeli and colleagues (1981) demonstrated that neither orientation selectivity nor direction selectivity in striate cortex depends exclusively on magnocellular or parvocellular contributions. The relative importance of magnocellular and parvocellular contributions to other response properties in striate and extrastriate cortex remains an important question.

The experiments described here were directed at examining magnocellular and parvocellular contributions to two response properties in monkey striate cortex: the latency and transience of visual responses. We were particularly interested in whether these properties could be used to identify a difference in LGN contributions in cortex. Latency and transience were selected because they distinguish magnocellular and parvocellular responses in the LGN and because they are relatively straightforward to measure. We were also encouraged to examine response latencies because large differences (10–20 ms) had been observed between the earliest activity in striate cortex and that in the middle temporal visual area (MT) and area V4 (Maunsell and Schiller 1984). Two factors are likely to contribute to magnocellular signals arriving at cortex earlier than those relayed by the parvocellular subdivision of the LGN. The axons of magnocellular neurons and the retinal ganglion cells that innervate them conduct impulses faster than their parvocellular counterparts (Dreher et al. 1976; Gouras 1969; Kaplan and Shapley 1982; Schiller and Malpeli 1977, 1978). Retinal processing is also likely to introduce differences. Stages before the level of the retinal ganglion cells account for a large portion of cortical response latencies, and it is likely that the retinal ganglion cells that project to the magnocellular layers are activated before parvocellular-projecting neurons when stimulus contrast is moderate or high (Shapley and Victor 1978). The greater transience of the responses to sustained stimuli in the magnocellular pathway also distinguishes them from parvocellular neurons (Marrocco 1976; Schiller and Malpeli 1977, 1978).

Visual responses were recorded from populations of striate neurons in behaving macaque monkeys before and

after making ibotenic acid lesions of either the magnocellular or parvocellular subdivision of the LGN. We found that the earliest 7–10 ms of visual responses can be attributed entirely to excitatory drive provided by the magnocellular layers of the LGN. However, most neurons become active after this period, so their responses could be mediated by excitatory drive from either LGN subdivision. The transience of cortical responses changed after selective LGN lesions, but this measure also failed to provide a conclusive indicator of LGN contributions. Extremely sustained responses and extremely transient responses remained after either type of LGN lesion.

During these studies we also found large and consistent differences in the latency of visual responses between different layers in striate cortex. In particular, latencies increase progressively from layer 4 toward the surface of cortex, so that activation of the most superficial neurons in striate cortex occurs ~10 ms after the initial arrival of geniculate signals in cortex. This difference is likely to reflect a pattern of microcircuitry that may be important for the computations performed by cortex.

METHODS

Visual stimulation and data collection

Visual responses were recorded from striate cortex in four behaving macaque monkeys (3 *Macaca fascicularis* and 1 *M. mulatta*) while they performed a fixation task for a water or juice reward. Each animal was implanted with a head-restraining post and a scleral search coil in an initial surgery (Judge et al. 1980; Robinson 1963). In most cases the restraining post was secured with orthopedic plates and screws (Synthes). All surgeries were aseptic and performed under gaseous anesthesia. After recovery from the surgery, the animal was trained to maintain fixation on a small spot for periods that lasted up to 4 s. The peak-to-peak noise in the eye position signal from the search coil was ~0.1°, and trials were aborted without reward if gaze drifted more than ~0.5° from the center of the fixation target.

Once the animal had learned the fixation task, a recording chamber was implanted over striate cortex in another surgery. A 6-mm craniotomy was made within this chamber, leaving the dura mater intact. The craniotomy provided access to cortex for 3–4 wk, and several craniotomies were made in the recording chamber. On each day of recording a microdrive was mounted over the chamber, which was then filled with sterile mineral oil and sealed. The microdrive advanced a Pt/Ir microelectrode (Wolbarsht et al. 1960) through the dura mater and into striate cortex on the surface of the hemisphere. The signal from the electrode was amplified, filtered, and monitored on an oscilloscope and audio monitor. Recordings were made from isolated units and from small multiunit clusters that had receptive fields in the lower visual quadrant between 2 and 4° eccentricity. Efforts were made to ensure that the entire thickness of striate cortex was sampled on every penetration.

The visual stimulus was a high-contrast, bright, stationary, black-and-white square-wave grating with a spatial frequency of 3.0 cycles/deg and a diameter of 3.25°. The bright bars of the grating had a luminance of 50 cd/m^2, and it was projected on a Polacoat screen that had a background luminance of 1 cd/m^2. This stimulus was selected in part because it was an adequate stimulus for neurons in both the magnocellular and parvocellular subdivisions of the LGN. When a unit was isolated, the grating was centered on the receptive field and the preferred orientation was determined to the nearest 22.5° by listening to the audio monitor or examining response rasters. The animal was given no incentive to attend to the stimulus. Quantitative data were collected under computer control, using a window discriminator to digitize action potentials. The time of occurrence for each action potential was recorded to the nearest millisecond. During data collection the stimulus was presented in 1-s cycles, during which it was on for 250 ms. Usually three stimulus cycles were presented in succession during each fixation period. Stimulus presentation was controlled with a mechanical shutter, which opened fully within 2 ms.

Data analysis

Responses to 100 or more stimulus presentations were collected at each site. These were compiled into an average response histogram with a binwidth of 1 ms, which was then used to compute response latency. Latency was determined by first finding a Poisson distribution that described values in the 250 bins immediately before stimulus onset. Latency was taken to be the time corresponding to the first bin after stimulus onset that *1*) exceeded a level corresponding to a probability of $P = 0.01$ for the background distribution and *2*) was immediately followed by a bin that also reached this criterion and a third that exceeded a level corresponding to a probability of $P = 0.05$. This method was found empirically to provide a reliable indication of the beginning of visual responses for the data set. Because the calculated latency is based on a statistical criterion, adding stimulus presentations to the histogram could affect the latency determination by reducing the variance of the baseline activity. For this reason we consistently used responses to 100 presentations for determining response latency. In some figures data are presented as impulse density functions that were produced by smoothing the 1-ms peristimulus histograms with a discrete-time Gaussian pulse with a standard deviation of 3 ms.

Selective LGN lesions

The individual contributions of the magnocellular and parvocellular subdivisions of the LGN to cortical response latencies were examined by recording from striate cortex after selectively ablating one or the other LGN subdivision with ibotenic acid (Schwarcz et al. 1979). For making these lesions, the animal was premedicated with ketamine (15 mg/kg im), diazepam (0.5 mg/kg im), and atropine (40 μg/kg im), and the trachea and a leg vein were cannulated. A craniotomy and durotomy were made dorsal to the LGN under sufentanyl anesthesia (20 μg/ml iv), and a guide-tube containing a Pt/Ir electrode was advanced to a site ~6 mm dorsal to the LGN. The animal was then paralyzed (vecuronium 20 μg/kg), respired on room air, and maintained on an infusion of paralytic that contained a dose of anesthetic that was shown to be adequate during the initial surgery (vecuronium 100 $\mu g \cdot kg^{-1} \cdot h^{-1}$ and sufentanyl 4–10 $\mu g \cdot kg^{-1} \cdot h^{-1}$; Van Sluyters and Oberdorfer 1991). Expired CO_2, electrocardiogram (ECG), and heart rate were monitored throughout the procedure, and the respiration rate adjusted to maintain expired CO_2 near 4.5%. A thermostatically controlled heating pad maintained body temperature. The eyelids were opened and covered with gas-permeable contact lenses after applying ophthalmic atropine (1%) for mydriasis and cycloplegia.

Microelectrode recordings and visual stimulation with a handheld projector were used to locate receptive fields in either the magnocellular or parvocellular layers of the LGN that were centered within ~0.5° of the receptive fields that had been recorded from striate cortex. When the target had been located, the microelectrode was removed and was replaced with a recording/injection probe (Malpeli and Schiller 1979; Maunsell et al. 1990) that contained a solution of ibotenic acid in phosphate buffer (5 μg/μl, 7.4 pH). Recordings from the probe were used to relocate the

target representation. Once the tip of the probe was positioned, ibotenic acid was slowly injected under pressure. For magnocellular lesions, a single 750-nl injection was made centered between layers 1 and 2. For parvocellular lesions, two injections were made: 750 nl in layer 4 and 1,200 nl in layer 6. These lesions ablated representations of the visual field that were appreciably larger than the receptive fields of the neurons being recorded in striate cortex (see Maunsell et al. 1990).

When the injections were completed, infusion of the paralytic was stopped and the probe and guide were withdrawn. The surgical site was closed using aseptic technique. Recovery from paralysis was facilitated with an anticholinesterase (neostigmine 25 mg/kg im, preceded by atropine 15 mg/kg im). The animal was usually breathing normally within 90 min.

Histology

When the recordings were completed, the animal was killed with barbiturates and perfused with phosphate buffer rinse followed by paraformaldehyde fixative. In most cases a second rinse was used to remove excess fixative. The brain was then removed, blocked, and equilibrated with 30% sucrose in phosphate buffer. Forty-micrometer sections were cut on a freezing microtome and series of sections were stained with cresyl violet or reacted to show cytochrome oxidase activity (Wong-Riley 1979).

Some of the analysis involved examining laminar differences in response properties. Because the penetrations spanned only one thickness of cortex and recordings were made over a period of many months, it was not possible to reconstruct individual electrode penetrations. Depth within cortex was established physiologically on each penetration. The electrode's entry into cortex was usually obvious, and layer 4C could be identified unambiguously on the basis of its distinctive response properties (high spontaneous rate of firing, lack of orientation selectivity, and monocular responses). Electrolytic lesions made shortly before death in one animal confirmed the physiological criteria. The accuracy of the physiological determinations of depth in cortex is also confirmed by the consistent changes in response properties that were seen at different depths in cortex described below.

RESULTS

Responses were collected from 298 single units and 1,129 multiunit sites in striate cortex of four macaque monkeys. In the three animals in which excitotoxin lesions were made in the LGN, a further 435 single units and 551 multiunit sites were recorded in striate cortex after the ablations were made.

Determination of visual response latencies of neurons in striate cortex

For latency measurements to be meaningful, the responses must have a clear onset. Most neurons in the striate cortex of alert macaques responded strongly to the visual stimulus, and average response histograms usually showed a clear onset of activity. In most cases it was possible to determine a well-defined and unambiguous latency. Figure 1 shows responses recorded from two representative units in striate cortex. Each plot is the average of responses to 100 stimulus presentations. The stimulus was on during the first 250 ms of the period (thick portion of the x-axis). The calculated latency (see METHODS) for the response in Fig. 1A was 27 ms, whereas that for Fig. 1B was 99 ms. Although the responses differed greatly in their latency and

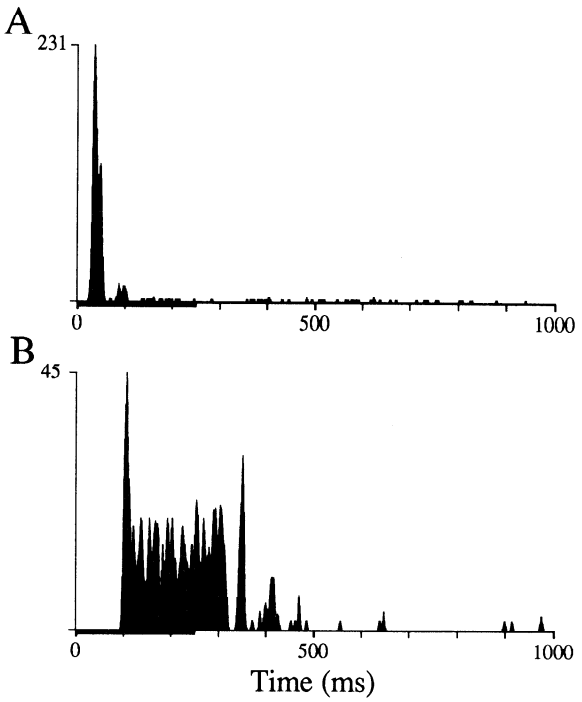

FIG. 1. Visual responses recorded from 2 isolated units in striate cortex. Each plot shows average response to 100 stimulus presentations. Plot represents 1 stimulus cycle in which the stimulus was on during the 1st 250 ms (thick portion of x axis). Response latency for the plot in A is 27 ms, whereas that for the plot in B is 99 ms. Although these units differed considerably in latency and strength of response, the onset of response was clear in both cases. Both plots have been smoothed as described in METHODS. Vertical axes of responses in this and other response plots are scaled in units of impulses per second.

maximum rate of firing, both had sharp onsets and rose quickly to a peak. It was our experience that even responses with long latencies usually had sharp onsets. Low spontaneous activity was common among the units recorded and contributed to the clarity of the onset.

An overall picture of responsiveness in striate cortex can be obtained by averaging the responses of different units. Figure 2A shows an average response histogram that combines the responses of 100 neurons that were recorded individually in striate cortex in one cerebral hemisphere. The units that contributed to the average were not selected for strength or even responsivity: data were collected from every neuron that was isolated and held long enough to sample 100 stimulus presentations. Of the 100 units, only 9 did not reach the response criterion described in METHODS. The average response was robust, with a peak that was 10 times the background probability of firing (72:7 imp/s).

Although the onset of activity in Fig. 2A is reasonably sharp (details in its rising edge are discussed below), it does not accurately represent the response profile of a typical neuron. Averaging across units smears the response because different neurons became active at different times (see Fig. 1). This temporal smearing can be reduced by shifting the response histograms from individual units left or right to align their response onsets before they are averaged together. This was done in making the plot in Fig. 2B, which replots the same data that appear in A. Before averaging, each unit's response histogram was shifted to bring its calculated latency to 50 ms. The number of impulses in

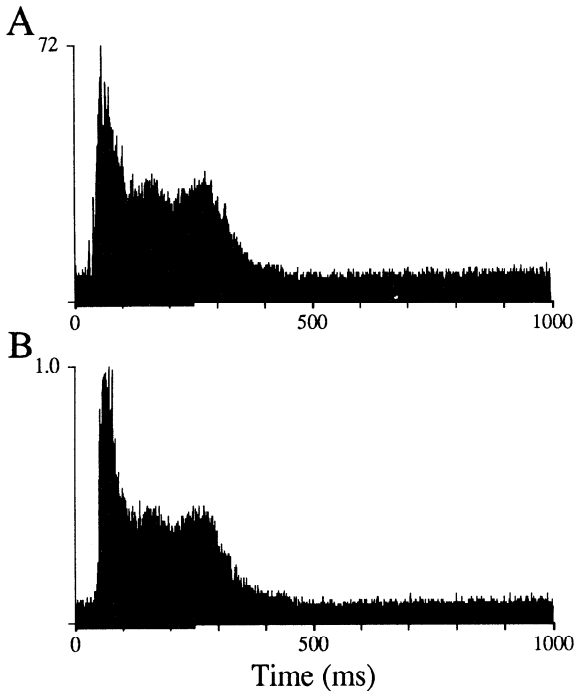

FIG. 2. Average response of individual striate neurons. *A*: average of responses from 100 striate neurons that were recorded individually. *B*: same data replotted, representing the average response profile for striate neurons. Plot *B* was constructed by first normalizing all individual histograms to same total number of impulses (to avoid emphasis of more active units). Each histogram was then shifted horizontally so that the response began at 50 ms (responses from units lacking a detected response were not shifted before averaging). Individual histograms were then averaged.

each unit's response histogram was also normalized to an arbitrary value before averaging, to avoid emphasizing the more active units. This histogram therefore represents the average response profile for the units recorded in striate cortex. The response rises rapidly to a peak, and the sustained response is about one-third the peak level. Thus the latencies of visual responses described in the following sections were typically clearly defined. Because the average activity before the alignment time in Fig. 2*B* (50 ms) is fairly flat, this plot also shows that the method used to compute latency does not miss major portions of the responses.

Although we could determine a well-defined visual response latency at most sites, neurons do not have a single, fixed latency. It is well established that stimulus parameters such as contrast and luminance have pronounced effects on visual response latencies (Lennie 1981; Levick 1973; Sestokas and Lehmkuhle 1986; Shapley and Victor 1978). Figure 3 shows responses from a site where responses were collected from a cluster of units while the stimulus was attenuated with neutral-density filters (Kodak Wratten filter #96) of different strengths. Attenuating the stimulus decreased responses and shifted the latency from 21 ms in the unattenuated condition to 69 ms with 1,000-fold attenuation. No response was detected with 10,000-fold attenuation. Visual response latencies invariably lengthened with decreasing stimulus intensity. Thus all visual response latencies described here must be considered specific to the stimulus conditions used and cannot be compared directly with those from other studies. We used a fixed, intense stimulus with a fast onset because it provided the clearest responses. It is probable that some of the observations reported here would not be seen using other stimuli. For example, less sharp response onset would be expected using a video display, which scans the stimulus across the screen and can achieve only limited contrast and brightness.

It should also be noted that trial-by-trial variability in latency and strength of response is not addressed here. The visual response latency of neurons varies considerably from one stimulus presentation to the next (Sestokas and Lehmkuhle 1986, 1988). The analyses presented here are based on compiled responses from many stimulus presentations and therefore measure the latency of a neuron's average response.

Distribution of visual response latencies in striate cortex

The distributions of visual response latencies for individual neurons and multiunit sites in striate cortex are plotted

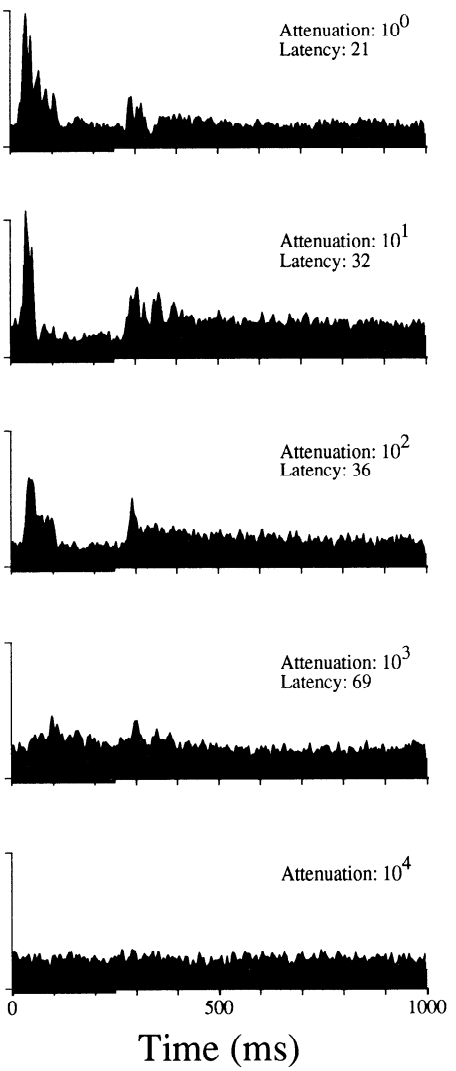

FIG. 3. Effects of stimulus intensity on response latency. Each panel shows responses of a single multiunit site near layer 4Cα to stimuli of different intensity. Stimuli were attenuated with neutral-density filters. Reducing intensity increased response latency and eventually abolished responses. The *y* axis in all panels is scaled to 750 imp/s. Each plot has been smoothed.

in the *top* and *bottom* rows of Fig. 4. The *left* two columns are data from two different animals, whereas the *right* column includes data from all animals. The single unit latency distributions had long tails (latencies beyond 75 ms are truncated). The distributions for individual animals had different short-latency cutoffs. The shortest latency recorded from a single unit in *case 1* was 27 ms, whereas that from *case 2* was 20 ms. Multiunit recordings were made to examine whether the single-unit recordings were adequately sampling the shortest response latencies. We reasoned that multiunit recording would sample from more neurons and would be less likely to miss significant populations of cells that might be difficult to isolate. For multiunit recording, responses were sampled every 100 μm from the surface of cortex to white matter. The shortest latencies in the multiunit latency distributions (*bottom* row) were always similar to those in the single-unit distributions. The most salient difference is that multiunit sites have fewer long response latencies, presumably because most clusters of units included some with short latencies. For this reason, multiunit recordings are likely to underestimate the number of units with long latencies.

Several factors could contribute to the differences in neuronal response latencies between animals. There may be systematic differences with size and age. *Case 2* was a young animal (3.3 kg), whereas *case 1* was a mature male (~7 kg). The other young animal used in this study (4.5 kg) had a minimum latency of 20 ms, whereas that for the other large animal (which was the *M. mulatta*) was 31 ms. Variation might also arise from individual differences in the optics of the eyes. In humans, pupil area can vary between individuals by as much as a factor of 4 (Spring and Stiles 1948), which would greatly affect retinal illuminance.

Because the latency distributions of different animals had markedly different cutoffs, combined distributions in the *rightmost* column in Fig. 4 were made plotting visual response latencies relative to the shortest latency recorded from each animal. In addition to the expected shorter median latency in the multiunit distribution, the combined single-unit distribution has a small peak at the shortest latencies. This peak can also be seen in the single-unit latency distributions for *cases 1* and *2*, albeit less convincingly. The comparative weakness of this early peak in the multiunit data probably stems from the early units having a relatively weak signal (see below), so that their contribution tends to be lost in multiunit recordings. The overall shift toward earlier responses in the multiunit data may also act to obscure the clarity of an early peak in the distributions.

The units with the shortest response latencies were encountered in layers 4C and 6. We suspect that these responses were recorded from afferent LGN axons because they typically had action potentials of very short duration and lacked an inner segment/soma-dendritic break; additionally, the amplitude of the signal recorded was very sensitive to small movements of the electrode (Lemon 1984). The following section presents data that show that the shortest response latencies in striate cortex depend on magnocellular contributions.

Magnocellular and parvocellular contributions to visual response latencies

The dependence of the earliest response latencies on excitatory drive relayed by the magnocellular layers of the LGN could be shown directly by eliminating magnocellular contributions with selective lesions in the LGN. In two animals a lesion was placed in the portion of the magnocellular LGN that represented the part of the visual field that was also represented under the striate cortex recording chamber. In another animal a corresponding lesion was made in the parvocellular layers. An attempt to make a second parvocellular ablation in another animal failed to

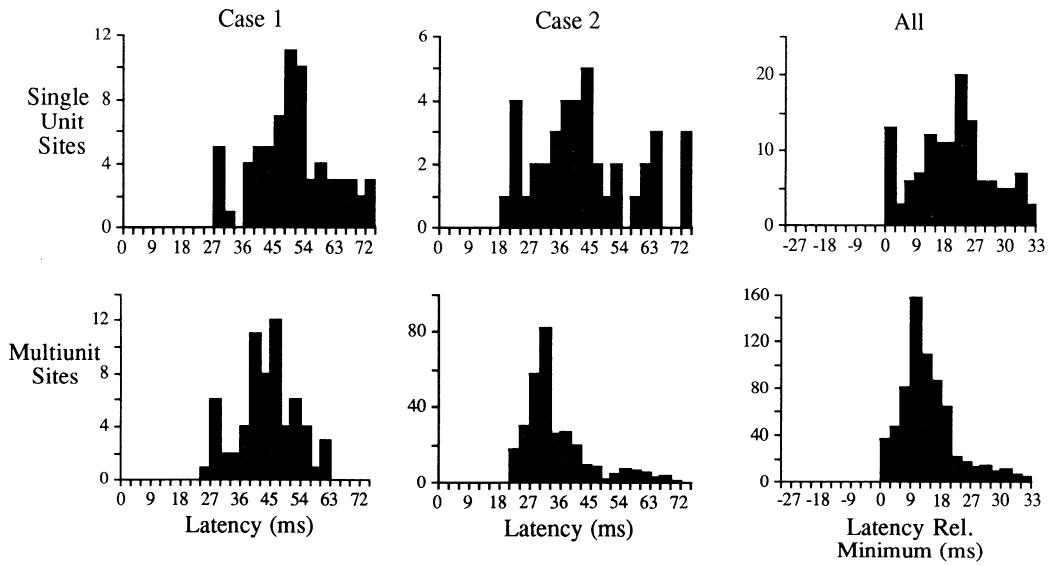

FIG. 4. Distributions of visual response latencies in striate cortex. *Upper* and *Lower* rows are data from single and multiunit recording sites, respectively. Data from 2 individual animals are plotted in the *left 2* columns. *Right* column: average from all animals. Different animals had appreciably different minimum response latencies. Because latency distributions from different animals were offset in time, latencies in the *right* column were plotted relative to the shortest latency recorded in each animal.

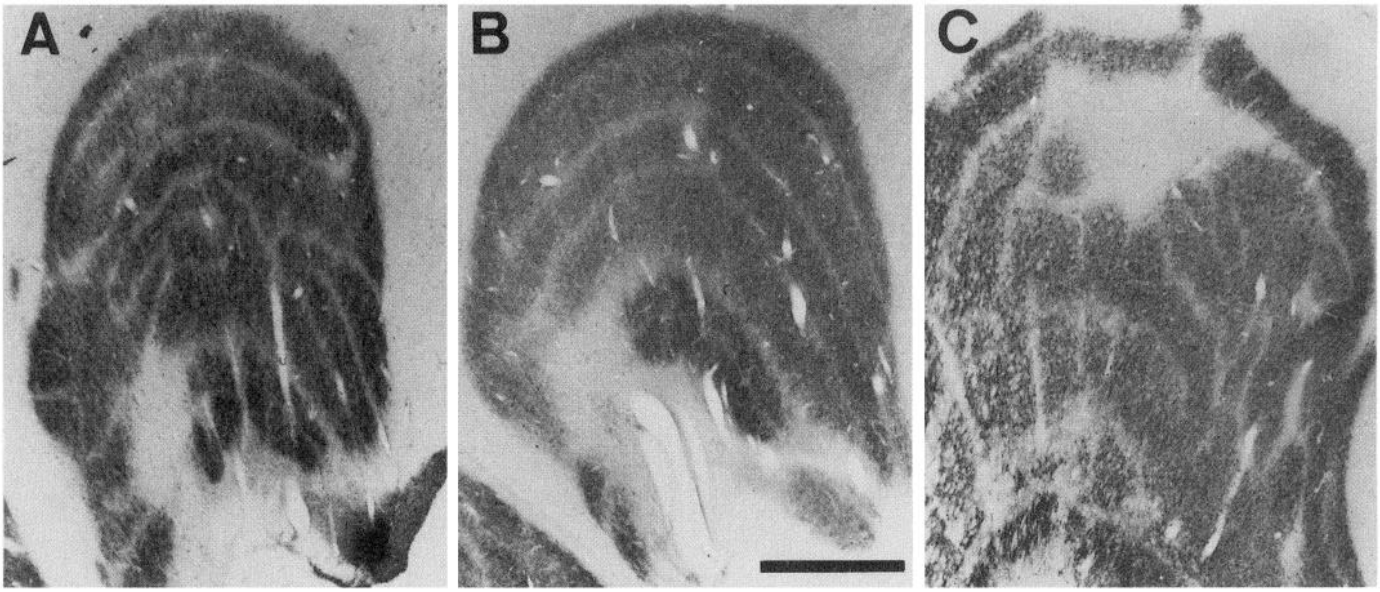

FIG. 5. Ibotenic acid lesions of the LGN. *A*: coronal histological section through a magnocellular lesion that has been stained for cytochrome oxidase. Lesion is evident as a small, lightly staining region at the bottom of the LGN in the magnocellular layers. *B*: magnocellular lesion in a 2nd animal. Large hole in the lesion is a blood vessel. *C*: histological section through a parvocellular lesion that has been stained for cytochrome oxidase. Parvocellular lesion was somewhat larger than is apparent on this section, extending further into layers 6 and 4 on other sections. All photomicrographs are at the same magnification (scale 1 mm).

produce a histologically identifiable lesion for unknown reasons. Figure 5,*A* and *B*, shows cytochrome oxidase–stained sections that were taken from the animals with magnocellular lesions. Each lesion is visible as a lightly staining region in the magnocellular layers. A section through the parvocellular lesion in the other animal is shown in Fig. 5*C*.

Data were collected from striate cortex before and after the lesions were made. The distributions of postlesion multiunit visual response latencies are plotted in Fig. 6. The three panels in this figure show data associated with the lesions in the corresponding panels in Fig. 5. Vertical lines mark the latency of the earliest multiunit response that was recorded in each animal in prelesion recording. The earliest responses were appreciably later after the magnocellular ablations (10 and 7 ms). The slower response cannot be attributed to a nonselective reduction of the number of LGN neurons innervating the region, because the parvocellular lesion—which was larger and involved a greater number of neurons (Fig. 5C)—did not eliminate the earliest visual response latencies. The failure of the parvocellular lesion to eliminate the earliest responses also shows that the ibotenic acid lesion did not affect fibers of passage.

The data in Fig. 6 suggest that the earliest parvocellular contributions are evident in striate units ~7–10 ms after the earliest magnocellular activity (under these stimulus conditions). While about the first 7–10 ms of activity in normal striate cortex can be assigned to magnocellular contributions, later responses might depend on either LGN subdivision. For this reason visual response latencies appear to be of limited value in determining the magnocellular and parvocellular dependencies of the majority of neurons in visual cortex.

Magnocellular and parvocellular contributions to the transience of visual response

Neurons in the magnocellular and parvocellular layers of the LGN differ in the transience of their responses to sus-

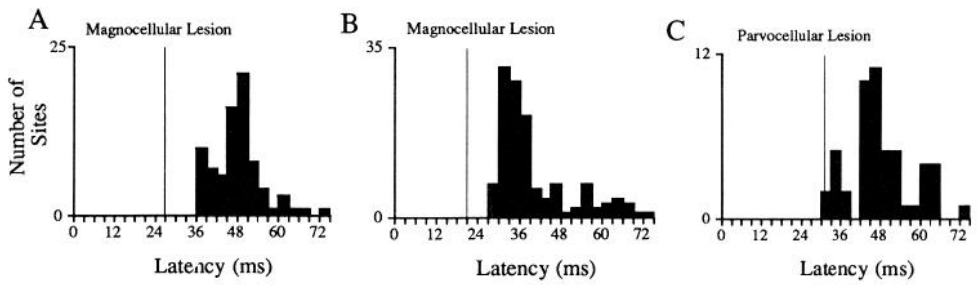

FIG. 6. Distributions of multiunit visual response latencies in striate cortex after selective LGN lesions. *A–C*: data collected after lesions in corresponding panels in Fig. 5: *A* and *B*, responses after magnocellular lesions; *C*, distribution after a parvocellular lesion. Vertical lines mark earliest multiunit responses that were recorded from striate cortex in each animal before the lesion was made. Both magnocellular lesions eliminated the earliest 7–10 ms of activity. Failure of the parvocellular lesion to produce this effect suggests that this change cannot be attributed to a nonspecific reduction in LGN contributions to cortex.

tained stimuli (Schiller and Malpeli 1978). A corresponding difference is seen between the M- and P-cells in the retina that provide the input to the LGN. Figure 7 reproduces data that show this difference, as published by Schiller and Malpeli (1977, 1978). These investigators computed an index of response transience that was defined as 100·[early response/(early response + late response)]. A unit with no late response would have an index of 100, and a response that did not diminish with time would have an index of 50. M- and P-cells in the retina and neurons in the magnocellular and parvocellular layers of the LGN differ greatly in their transience. The mean indexes for P-cells and parvocellular neurons are 69 and 68; those for M-cells and magnocellular neurons are 84 and 88. The responses of cortical neurons might be expected to reflect this property of their inputs.

The transience of cortical responses to the 250-ms stimulus was examined by computing the index of transience used by Schiller and Malpeli (1977, 1978). The number of impulses in the first 35 ms after the onset of response was compared with the number in a 35-ms period starting 125 ms after onset. The distribution of transience indexes for single units in normal striate cortex in one animal is shown in Fig. 8*A*. It is not overtly bimodal. The mean value in striate cortex (69) is similar to the means for parvocellular neurons and retinal P-cells, but the distribution is much broader than in earlier stages. A larger portion of the population had extremely transient responses (near 100), and values below 50 were also encountered, indicating that the activity of some neurons increased between the early and late periods.

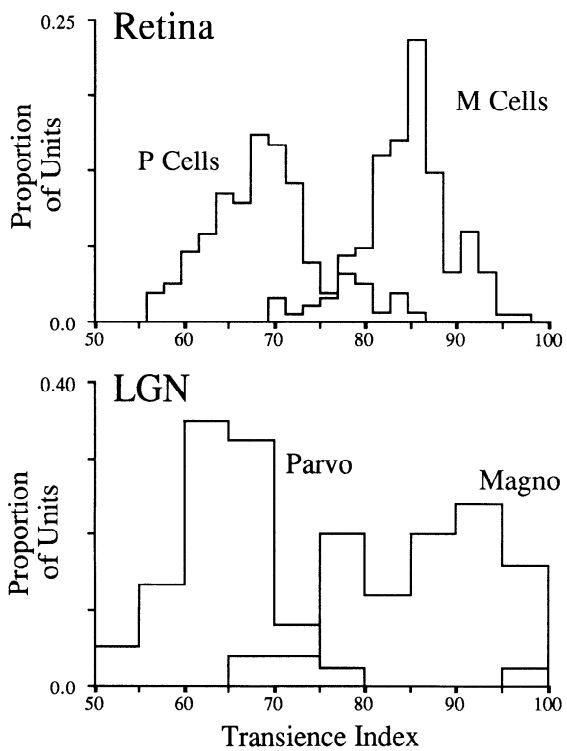

FIG. 7. Transience of striate responses in monkey retina and LGN. Data from studies by Schiller and Malpeli (1977, 1978) have been replotted for comparison with this study. Schiller and Malpeli computed an index of transience by comparing average rate of firing during early and late stages of the response. Index was 100·[early response/(early response + late response)]. Retinal distributions are based on 122 P-cells and 147 M-cells. Those for the LGN are from 37 parvocellular and 26 magnocellular neurons. Response transience distinguishes populations of neurons in both structures.

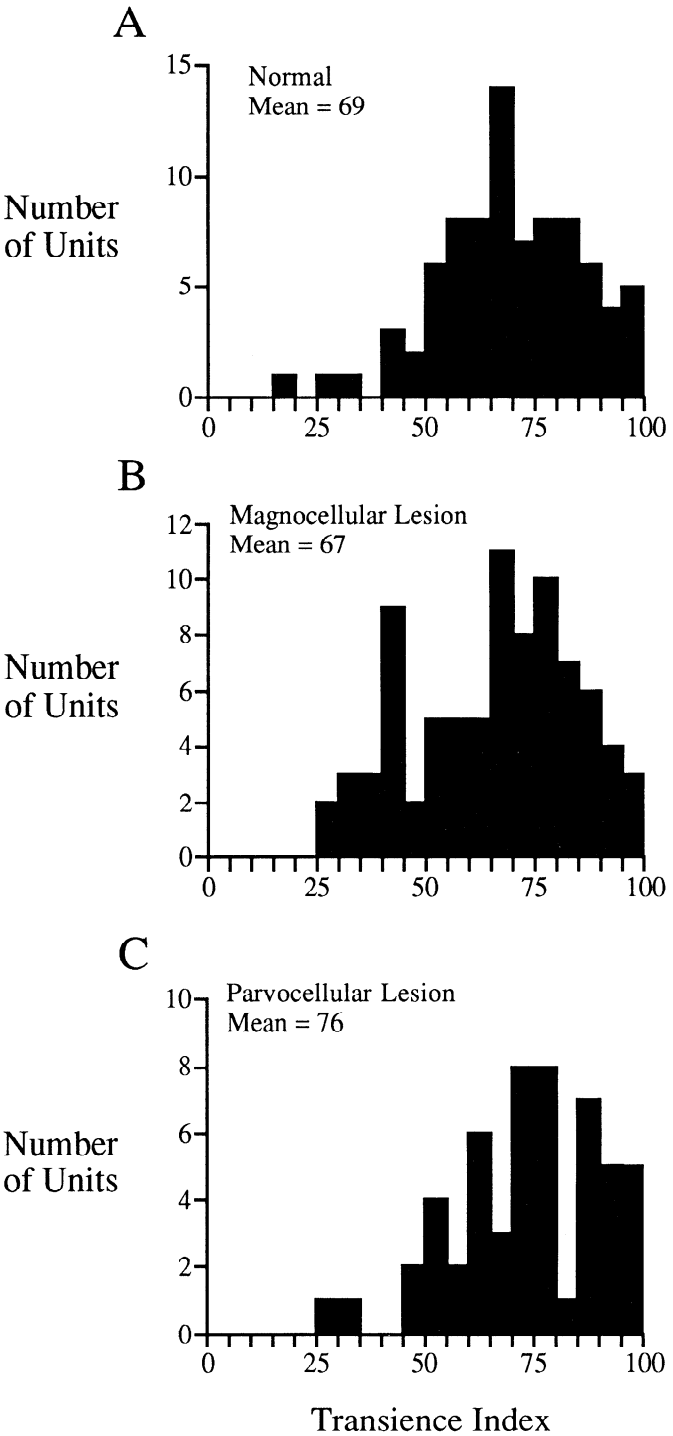

FIG. 8. Transience of striate cortex responses in normal and LGN-lesioned animals. Transience index of 100 corresponds to an extremely transient response, 50 indicates a response that does not change with time, and numbers below 50 are associated with responses that increase over time. *A*: distribution of transience indexes for single units in normal striate cortex. *B*: distribution of striate transience after a magnocellular lesion. Mean is little affected. *C*: transience in striate cortex after a parvocellular lesion. Responses are appreciably more transient overall, but some very sustained responses survive.

After the magnocellular layers were ablated, the mean transience index in striate cortex was slightly reduced (Fig. 8*B*), indicating that responses were more sustained. However, the shift was very small and extremely transient responses were supported by parvocellular inputs alone. Ablating the parvocellular layers in another animal had a more obvious effect, suggesting that many sustained responses depended on parvocellular input. However, some very sustained responses remained. Neither postlesion distribution was significantly different from the prelesion distribution (Mann-Whitney test). The parvocellular lesion, unlike the magnocellular lesions, did not destroy the relevant visual field representation in all layers in that subdivision of the LGN. It is therefore probable that the shift toward more transient responses would have been larger had the parvocellular lesion been complete. Nevertheless, the extensive overlap of the distributions in Fig. 8 suggests there is little basis for using response transience to distinguish magnocellular and parvocellular contributions to individual neurons in striate cortex.

Perhaps the most striking change after the LGN ablations was an increase in the number of neurons with indexes below 50 after the magnocellular lesion. Some of these had poor, slowly rising responses, but others had very strong, sustained responses. It is possible that the magnocellular ablation removed a suppressive input from these neurons.

Characteristics of magnocellularly derived responses in striate cortex

The units in striate cortex with the earliest latency shared two other characteristics: they were very transient, and their responses oscillated at a frequency in the range of 50–100 Hz. Figure 9 shows responses of three representative isolated units from this group. Each response is plotted twice.

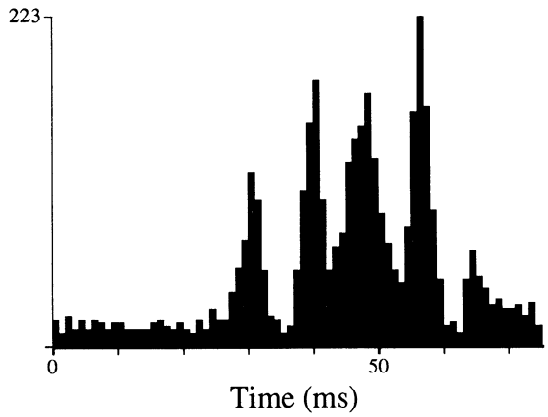

FIG. 10. Average response histogram from neurons with high-frequency oscillations. Responses from all 6 units with early response latencies from *case 1* were included in the average. Prominent peaks in the average plot show that oscillations in different units were in nearly the same phase.

The *left* column shows the full stimulus period, and the *right* column shows an expanded representation of the response during the first 75 ms after stimulus onset. Each of these units gave a transient response to the onset of the stimulus, and then quickly returned to a low or suppressed rate of firing for the remainder of the stimulus presentation.

The expanded plots in the *right* column of Fig. 9 show that the transient responses contained a series of short bursts of response. Examining responses to individual stimulus presentations revealed that these neurons frequently fired multiple action potentials within the individual peaks of the oscillations. Virtually all the single units that had a response latency within 5 ms of the earliest observed latency had this type of periodicity. Oscillations of this sort have been described previously in the visual responses of

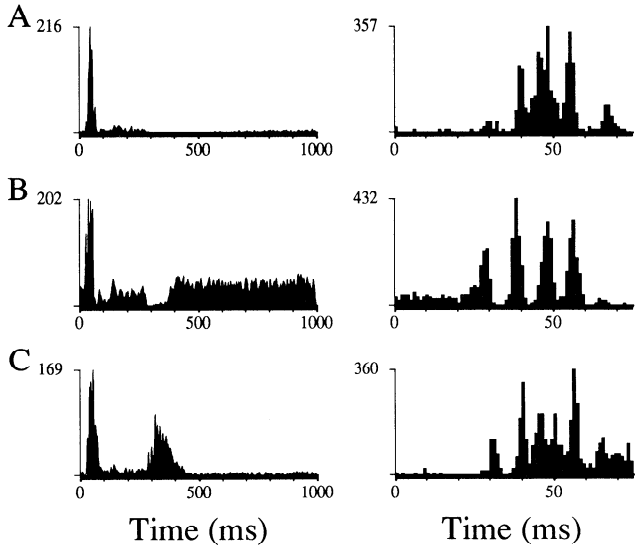

FIG. 9. Representative response profiles from 3 neurons that were among those with shortest response latencies. *Left*: smoothed impulse density functions for the whole stimulus cycle. *Right*: unsmoothed histograms of 1st 75 ms. One hundred stimulus presentations were averaged for each unit. All 3 had very transient responses (transience indexes: 93, 86, and 96). The *right* column shows that the transient response to stimulus onset comprised several cycles of a high-frequency oscillation. This oscillation was characteristic of units with earliest response latencies.

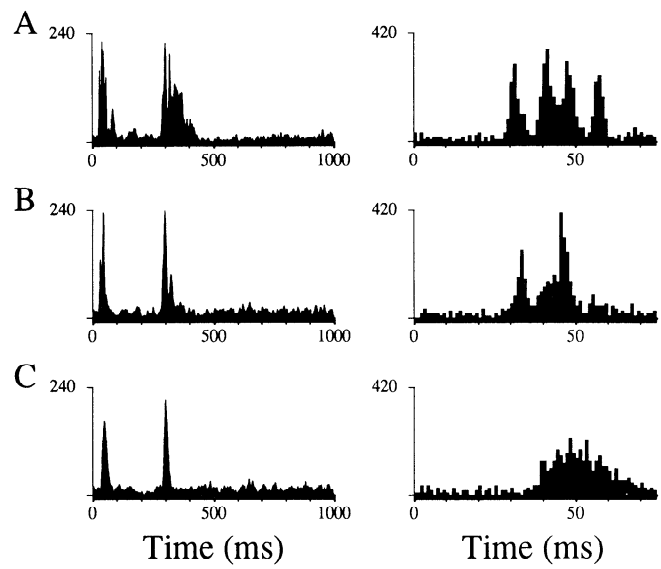

FIG. 11. Effect of lowering contrast on high-frequency oscillations. One unit that had high-frequency oscillations was tested with different stimulus intensities. *Left*: smoothed impulse density plots of responses to the standard stimulus (*A*), 10-fold attenuation (*B*), and 100-fold attenuation (*C*). One hundred stimulus presentations are averaged in each. *Right*: unsmoothed histograms of 1st 75 ms of response. Attenuating the stimulus reduced the response and eliminated high-frequency oscillations.

retinal ganglion cells in unanesthetized or lightly anesthetized mammals of many different species (Steinberg 1965). The majority of retinal ganglion cells of all classes have oscillations in their responses to strong stimuli, and these oscillations are thought to stem from interactions between the center and surround of receptive fields of bipolar cells (Ariel et al. 1983; Doty and Kimura 1963).

In our data, the oscillations of different units had not only the same period, but also the same phase (within animals). This can be seen in the *right* half of Fig. 9. Figure 10 shows an average response histogram that was produced by combining the activity of all the short-latency single units collected from one animal ($n = 6$). The prominent periodicity in the combined histogram shows that the oscillations of the individual response profiles were aligned, although each unit was recorded on a different penetration on a different day. The synchronous firing of these units can also be seen in the average response histograms in Fig. 2*A*, where small peaks are superimposed on the earliest portion of the response. Figure 2*A* also makes clear that units of this sort contribute a small fraction of driven activity in striate cortex.

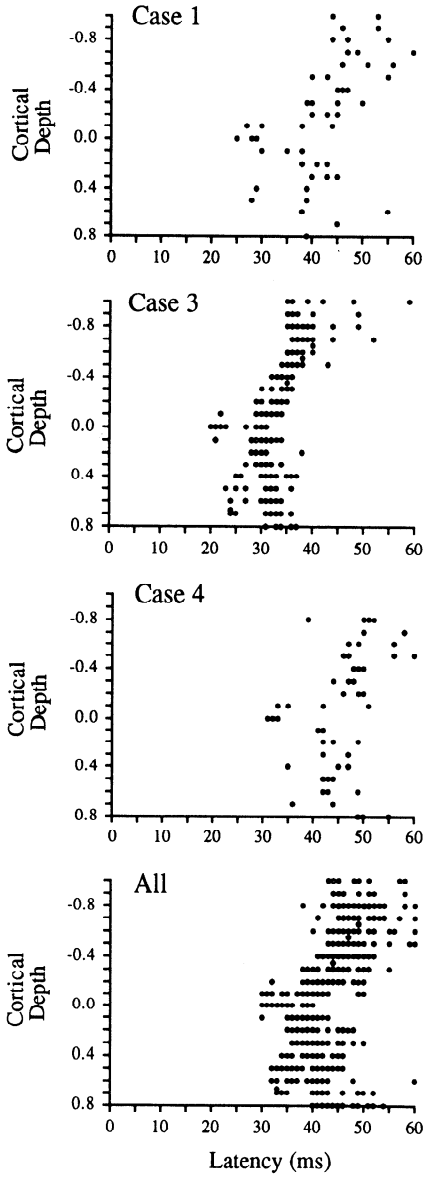

FIG. 12. Scatterplots of latency as a function of depth within striate cortex. Data from multiunit recording sites in 3 cases are plotted. Zero depth was physiologically determined layer 4C. Negative values are toward the surface, positive values toward white matter. Combined plot at the *bottom* was made by first shifting the latency values to bring the earliest latency in each animal to 30 ms. A consistent pattern is seen across animals. Shortest latencies are found in geniculate-recipient layers, and latencies become much longer in more superficial layers.

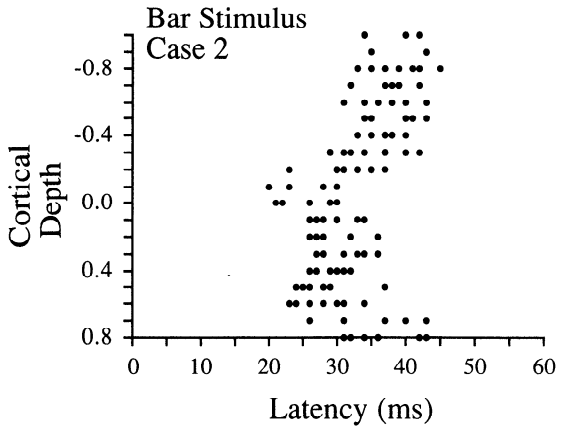

FIG. 13. Latency as a function of depth within striate cortex. These data were collected from multiunit sites using a bar stimulus. Distribution is similar to that found using the grating pattern, showing that it is not unique to the grating stimulus.

These oscillations were highly dependent on stimulus intensity. Figure 11 shows responses from one unit that was tested with stimuli of different intensities. The *left* and *right* columns plot the complete stimulus cycle and an expanded representation of the first 75 ms. Decreasing stimulus intensity reduced the number of driven action potentials, increased the response latency, and reduced the oscillations.

Response latencies in different cortical layers

Characteristic differences in visual response latencies were seen between different layers in striate cortex. The latency of multiunit recording sites is plotted against depth in cortex in Fig. 12. Data from three animals and the composite from all cases are shown. A depth of 0 is physiologically identified layer 4C for each traverse (see METHODS), negative numbers correspond to supragranular sites, and positive numbers correspond to sites in infragranular cortex. The surface of cortex was usually encountered ~0.9–1.0 mm above layer 4C. Multiunit recordings were made every 100 μm in near-radial traverses of cortex. *Case 3* was the most intensely sampled animal. Consistent patterns of laminar differences were found, although individual animals had different absolute latencies. To make the combined distribution in the *bottom* panel, the distribution from each animal was shifted to bring its shortest latency to 30 ms.

Unsurprisingly, the shortest latencies were consistently found in the top half of layer 4C (depths near 0.0). Responses with only slightly longer latency were encountered in layer 6 (+0.5–0.6 mm), which also receives direct input from the LGN (Blasdel and Lund 1983; Bullier and Henry 1980). Latencies were appreciably longer for sites closer to

the surface of cortex. Measurable responses near the surface of cortex were rarely seen earlier than 15 ms after layer 4Cα became active, or ~7 ms after layer 4Cβ. This difference was also seen in data from single-unit recordings.

To test whether the laminar differences in latency might depend on the particular stimulus used, we collected multiunit responses in the fourth animal using a small bar stimulus ($0.6° \times 0.06°$). Background and stimulus luminance were the same. Figure 13 shows that the same pattern of differences in latency exists with this stimulus as well, suggesting that it may reflect a general feature of processing in striate cortex.

DISCUSSION

Physiological signatures of parvocellular and magnocellular signals in cortex

The primary objective of this study was to examine whether magnocellular and parvocellular contributions to cortical responses could be identified in the response properties of cortical neurons. By recording responses before and after selective LGN ablations, we were able to assess the extent to which cortical response characteristics depended on each subdivision. The results suggest that response latency and transience are of limited value in determining magnocellular or parvocellular contributions. Only the first 5–10 ms of activity in striate cortex could be unequivocally assigned to magnocellular contributions. Activity that occurs later might be dependent on either subdivision for excitatory drive. Although selective LGN lesions affected response transience in the expected ways, either extremely transient or extremely sustained responses can arise from either magnocellular or parvocellular contributions.

It is possible that some reorganization occurred after the LGN lesions and that better correlations of response properties exist in normal animals. The current data cannot address this possibility. Experiments using reversible inactivation of individual LGN layers might resolve this question. We think the most likely explanation for the absence of distinct populations of response properties in striate cortex is that the inputs from the magnocellular and parvocellular subdivisions converge extensively in this area. Although the terminations of axons from the LGN subdivisions are cleanly segregated in layer 4C (Fitzpatrick et al. 1985), anatomic studies suggest extensive intermixing in other layers of striate cortex (Blasdel et al. 1985; Fitzpatrick et al. 1985; Lachica et al. 1992), and physiological recordings have demonstrated that a large fraction of neurons in striate cortex receive excitatory drive from both subcortical pathways (Malpeli et al. 1981).

Hubel and Livingstone (1990) measured color and contrast sensitivity in striate cortex to examine correspondences with magnocellular and parvocellular characteristics. They also found that these indexes did not unambiguously characterize the inputs to striate neurons. At present there is little basis for asserting that a particular cortical response property arises from one subdivision or the other.

Previous studies have attempted to relate response latency and transience to magnocellular and parvocellular contributions. Petersen and co-workers (1988) examined the latency and transience of average response profiles derived from single unit recordings in four extrastriate visual areas in the owl monkey (*Aotus*). They found that the MT and the medial area (M) had responses that were of shorter latency and less sustained than those in the dorsolateral area (DL) or the dorsomedial area (DM). Although these observations are consistent with the expectation that MT and M should receive more magnocellular input and DL and DM more parvocellular input, the current study makes clear that transient and sustained portions of visual response do not depend exclusively on magnocellular and parvocellular inputs.

Another report described response latency and transience in striate cortex and area V4 (Maunsell 1987) using a stimulus similar to that used in this study. Among the findings were the observations that the earliest response latencies in V4 were 22 ms after those in striate cortex and that, on average, responses in V4 were as sustained as the most sustained quartile of striate cortex units. These observations suggested that responses in V4 might depend exclusively on excitatory drive relayed by the parvocellular layers. However, recent experiments using selective reversible inactivation of LGN layers has shown that many V4 neurons are driven in part by the magnocellular neurons (Ferrera et al. 1991). The sustained, long-latency responses in V4 that exist despite appreciable excitatory drive from the magnocellular layers further emphasize the limited value of latency and transience as indicators of LGN contributions.

The apparently low reliability of physiological signatures of magnocellular and parvocellular contributions in cortex is readily explained. Many forms of processing can alter the appearance and nature of neuronal signals. Relatively simple manipulations can transform a sustained signal into a transient one, or vice versa. The absence of an early magnocellularly driven response in V4 might be explained if V4 neurons sum over such large populations of inputs that the earliest magnocellular inputs cannot generate a detectable response alone. Cortical processing of LGN response characteristics is also documented by the spatial contrast sensitivity of neurons in MT. Many neurons in MT have higher contrast sensitivity than any magnocellular or parvocellular neurons (Sclar et al. 1990), presumably reflecting the effects of converging inputs. Because LGN neurons have no true threshold and give graded responses even to very low contrasts (Derrington and Lennie 1984), the high sensitivity of MT neurons might arise from convergence of either magnocellular or parvocellular inputs.

Collectively, the available data suggest that measurements of response properties are unlikely to provide unequivocal support for magnocellular or parvocellular inputs. Because none of the established physiological differences between magnocellular and parvocellular neurons involves an absolute threshold, below which one channel is active and the other completely silent, the prospect of convergence and other cortical processing makes it impossible to associate a sensitivity in cortex with a particular LGN subdivision. Although it is possible that some index that involved a combination of multiple measurements might provide a valid indication of LGN contributions (see Ro-

dieck and Brening 1983), the weakness of individual measures makes this seem unlikely.

Laminar differences in responses

We observed striking differences in latency between different layers in striate cortex. Although there is little basis for assigning functional significance to these differences, they may provide valuable insights into mechanisms of cortical processing.

Activation in monkey striate cortex appears to spread from layer 4 toward the surface in a monotonic fashion. A similar pattern of activation by visual stimuli has been reported in striate cortex of anesthetized cats (Best et al. 1986). The consistency of this pattern across species, behavioral states, and different stimuli suggests that it may reflect an important feature of striate processing and potentially of neocortex in general. The current data cannot distinguish between a continuous, wavelike propagation toward the surface and a process in which signals are relayed in a few discrete steps. In either case, neither single-unit nor multiunit recordings revealed driven activity among the most superficial cell bodies until ~15 ms after layer 4 became active. This delay is long compared with the times normally associated with cortical processing. For example, a visual stimulus may activate neurons in V2 before those in the superficial parts of layer 2 in striate cortex: electrical stimulation of the optic chiasm activates V2 neurons only 7 ms after the earliest responses in striate cortex (Mitzdorf and Singer 1979). Because projections from V2 to striate cortex terminate primarily around the border between layers 1 and 2 (Rockland and Virga 1989), it is also possible that activation of superficial striate cortex may depend in part on extrastriate inputs.

The delay in the response of the superficial layers would seem less pronounced if neurons in those layers depended exclusively on parvocellular contributions, which arrive at layer 4C much later than the magnocellular contributions. This is unlikely to be the case. Anatomic studies show direct projections from magnocellularly dominated layers 4Cα and 4B to the superficial layers (Blasdel et al. 1985; Fitzpatrick et al. 1985; Lachica et al. 1992), and selective inactivation of the magnocellular layers reduces responses in most neurons in the superficial layers (Nealey and Maunsell 1991). Thus the latencies in the superficial layers are appropriately compared with the earliest activation in layer 4C.

A 10- to 15-ms delay may seem difficult to reconcile with axonal conduction speeds that are in the range of meters per second and synaptic delays that are ~1 ms. This delay could be explained if excitatory signals reached superficial neurons only after being relayed by many successive cortical neurons. However, a chainlike propagation of activation from layer 4 toward the surface is not immediately predicted from the anatomy of striate cortex. For example, some LGN afferents terminate in layer 1 (Blasdel and Lund 1983). On the other hand, these axons and terminations are described as extremely fine, and it is possible that they have slow conduction speeds. They are also so few that they do not label appreciably when the LGN is injected with radioactive amino acids (Hendrickson et al. 1978). Other anatomic observations are consistent with the idea that information is relayed toward the surface in a series of steps. Neurons in layer 4C do not send axons to superficial cortex, but instead terminate in layers 4B, 4A, and, to a lesser extent, in 3B (Fitzpatrick et al. 1985; Lachica et al. 1992). These layers in turn send axonal projections to more superficial cortex (Blasdel et al. 1985; Lachica et al. 1992). The axon collaterals of pyramidal neurons in layers 2 and 3 usually terminate immediately superficial to the soma (Lund and Boothe 1975). Thus it is possible that neurons in the superficial layers feed activation toward the surface in a series of many successive steps.

An equally plausible explanation for the delay is that the time for activation to spread from one level of neurons to the next is appreciably longer than typical 1- to 1.5-ms synaptic delays (Bullier and Henry 1980; Eccles et al. 1966). Cortical neurons can receive inputs from thousands of synapses (O'Kusky and Colonnier 1982), presumably from hundreds of neurons each making a small contribution to postsynaptic activation. Because action potentials arrive asynchronously, inputs must be integrated for appropriate intervals. The integration time for the postsynaptic neuron may make the delay in passing the signal to the next level of neurons much longer than a synaptic delay. Postsynaptic responses that are delayed by $\leq$1 ms may be restricted to neurons with extremely low convergence, such as those in the LGN (Cleland et al. 1971; Hamos et al. 1987). A neuron-to-neuron delay of 5 ms could account for much of the difference in activation times between the geniculate-recipient layers and the surface of striate cortex, even if only two or three levels of neurons were involved. The shorter cell-to-cell transmission times that have been seen in cortex with electrical stimulation (Bullier and Henry 1980) could be explained by the abnormal synchronization of the action potentials arriving at cortex after a shock. This might also explain why upper layer 2 is activated with less delay after electrical stimulation than it is after visual stimulation (Best et al. 1986; Komatsu et al. 1985).

A slow transmission of signals from one level of cortical neurons to the next could have important implications for understanding cortical processing. The slowness of neurons is frequently cited as an important limitation for the nervous system (e.g., Marr 1982; Sejnowski 1986). It has been suggested that the speed of neuronal processing requires that the nervous system carry out complex behaviors in the equivalent of "100 steps" of processing (Feldman 1985). Such views, however, usually estimate that steps occurred ~2 ms apart, a value based on a synaptic delay and a short axonal conduction time. If the effective step size for neuronal processing is several times longer, the constraints on neuron processing become even more severe.

Regardless of its origin, the late activation of superficial neurons raises many questions about its implications for cortical processing. Unfortunately, little is known about laminar differences in neuronal response properties that might be associated with the sequential activation in the superficial layers. Very few studies have examined functional differences within the superficial layers, and no striking differences have been described. Thus the functional implications of this pattern of activation remain obscure. It may have none. If cortical computations are based on average rates of firing determined over moderate periods, the exact time of activation may be irrelevant. Similarly, the

timing of the peak rate of firing may have more consequence than the onset. Whether or not the pattern of activation has direct functional consequences, it must be a consequence of the organization in the underlying microcircuitry. As such, it could provide valuable insights for understanding cortical processing.

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