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Visual latencies in areas V1 and V2 of the macaque monkey

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Abstract

Latencies to small flashing spots of light were measured in different layers of areas V1 and V2 in anesthetized and paralyzed macaque monkeys. The shortest latencies were found in layers 4Cα and 4B of area V1. Latencies in layer 4Cβ were on average 20 ms longer than those in 4Cα and 4B. The shortest latencies in area V2 were observed in the infragranular layers and they did not differ significantly from those found in the infragranular layers in V1. Similarly, latencies in the supragranular layers of V2 were not significantly different from those measured in the supragranular layers of V1. These results show that, in area V1, neurons of the magnocellular pathway are activated on average 20 ms earlier than those of the parvocellular pathway. Our data also suggest that much processing begins simultaneously in areas V1 and V2.

Keywords: Visual cortex, Magnocellular, Parvocellular, Electrophysiology

Introduction

The visual cortex of mammals contains a number of cortical areas which are densely interconnected. Connections between these areas can be classified as feedforward, feedback, or lateral (Felleman & Van Essen, 1991). Feedforward connections arise from supragranular and infragranular layer neurons and project predominantly to layer 4 of the target area whereas feedback connections arise mostly from infragranular layer neurons and terminate outside layer 4. By analogy with the LGN projections, which project to layer 4 of area V1, it is assumed that feedforward connections carry the visual drive from lower to higher-order areas.

Few functional studies have tested the function of feedforward and feedback connections. It has been shown that inactivation of area V1 leads to a strong decrease of response or total silence in extrastriate areas of the macaque (Schiller & Malpeli, 1977; Girard & Bullier, 1989; Girard et al., 1991*a*,*b*, 1992). This is in keeping with the idea that lower-order areas such as V1 send essential information to higher-order areas through feedforward connections.

Even less is known concerning the temporal aspect of the processing between two areas. Most studies of processing within area 17 have emphasized the fact that information is passed through several stages in layer 4 and supragranular layers before being transferred to other cortical areas. This suggests that extrastriate cortical areas should be activated only after a certain time devoted to processing within V1. One would therefore expect that latencies of neurons to visual stimulation would be longer in extrastriate cortical areas than in V1. However, a study by Raiguel et al. (1989) showed that latencies of neurons in areas V1, V2, and MT cover the same ranges and that latencies of neurons in V2 are only slightly longer than those of neurons in V1.

The results of Raiguel et al. (1989) were obtained with moving bars of different velocities, using an extrapolation method to determine the latencies. However, this method is less precise than the measure of the latency to a flash which can be done with a precision of a few milliseconds. It is also difficult to draw firm conclusions from the results of Raiguel et al. on areas V1 and V2 given the small sample of recorded neurons in V2 (13 units).

To obtain information on the relative timing of responses in areas V1 and V2, we measured the latencies to flashing stimuli in these two areas. We also studied the laminar distribution of neurons with different latencies, since consideration of the laminar position enables some inferences to be made concerning the flow of information transfer within one area and between areas (Maunsell & Gibson, 1992).

Methods

Recordings were made in Cynomolgus monkeys anesthetized and paralyzed according to methods described previously (Girard et al., 1991*b*). Anesthesia was maintained with nitrous

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oxide/oxygen (70/30%) supplemented with small regular injections of fentanyl (5 μg/kg·h). For simultaneous recordings in both areas, tungsten-in-glass microelectrodes were positioned in corresponding retinotopic positions of areas V1 and V2 by penetrating the dura mater. Single and multi-unit signals were extracted from recordings with the same microelectrode with the help of a Schmitt trigger with three independent windows.

Visual stimuli consisted of small (0.5–2 deg) spots of light produced by an optic bench and covering the receptive fields of units in both areas. When recordings were made in area V1 or in V2 alone, small rectangular or square bars were used (0.25–1 deg). We verified the effect of stimulus size on response latency in 39 neurons by stimulating with a small bar (usually 0.5 by 1 deg) and a square of light (1–2 deg wide). We found that the latency did not differ significantly for these two stimuli (Wilcoxon ranked pair test; 95% confidence). Light was ON for 3 s and OFF for 3 s. There was a delay of 5 ms between the synchronizing pulse and the time at which the shutter was 90% open and a delay of 7 ms for the 90% closing time of the shutter. Opening and closing times were faster that 1 ms, as measured with a photodiode. The latencies reported in the paper have been corrected for the opening and closing delays. The spot and background luminances were respectively 15 and 0.8 cd/m^2. The usual stimulus was white, but we used red or green isoluminant stimuli to optimize the response of color-selective units. We verified that the color of the stimulus did not influence the latency (Wilcoxon test on paired recordings in 37 units; see also Creutzfeldt et al., 1987). Receptive-field properties were measured with qualitative methods. Orientation-selective units were defined as those for which a clear cutoff in the response could be observed when orientation of the moving bar was varied. Direction-selective cells were those for which the response to the nonoptimal direction was less than 30% of the response to the optimal direction. Color selectivity was assessed qualitatively by comparing the responses to isoluminant red, green, and white stimuli moved through the receptive field or flashed ON and OFF. In some cases, this was verified by a quantitative comparison.

Latencies were measured from the peristimulus-time histograms (PSTHs) computed from a large number of presentations of the stimulus (150 on the average). For 90% of the sample, the number of presentations was higher than 100. In most of the remaining 10% cases, PSTHs were calculated from a number of presentations between 80 and 100. For a few cases of strong responses, a smaller number of presentations was used. To assess the latency, we used the method of Maunsell and Gibson (1992) that consists in assuming a Poisson distribution for the spontaneous activity and determining a threshold value based on this distribution. We computed the mean value of the number of spikes falling in the bins contained in the period 250 ms before light onset or offset. This was used for computing the value corresponding to $P = 0.01$. The latency was taken as the bin number for which this threshold value was crossed, provided that the next bin stayed above this level and that the following bin did not fall below the $P = 0.05$ level (Maunsell & Gibson, 1992). Latencies were usually estimated with bin widths of 1 or 2 ms. When the resulting PSTHs were too noisy, a 5-ms bin width was used (30% of the cases). The influence of bin width and number of presentations was assessed by recalculating the latencies for a single bin width (5 ms) and for a fixed number of presentations (100). This was done on the sample of responses obtained with number of presentations higher than 100 ($N = 740$). The latencies thus obtained were highly correlated with the original values ($r = 0.95$) and the mean values calculated this way in the different layers of V1 and V2 were within 3 ms of the original values. Since variability in bin width and number of presentations can change the minimum values of the latencies, we verified that the difference between the minimum latencies in layers 4Cα and 4Cβ remains the same when the latencies are measured with fixed bin width and fixed number of presentations. The data presented below are those obtained with the original procedure to preserve the largest sample and the most precise measurements.

Table 1. *Latencies in area V1 in ms*[a]

	V1 Total	ON su	ON mu	OFF su	OFF mu
Mean (ms)	77.3	77.7	70.8	88.4	79
Std. Dev.	25.1	24.1	22.7	27.7	25.1
S.E.M.	1.2	2.6	1.9	3.5	2.3
Count	412	83	144	62	123
Minimum	27	36	27	31	31
Maximum	187	140	187	158	153
Median	77	79	70	88	74

[a]Std. Dev.: Standard deviation; S.E.M.: Standard error of the mean.

For histological reconstructions, lesions were placed either every millimeter or every 1.5 mm in the electrode track (7–10 μA for 7 s, electrode negative). After an overdose of nembutal, animals were perfused with normal saline followed by 4% paraformaldehyde in phosphate buffer. Two days before cutting, the brain was placed in a 30% sucrose solution for cryoprotection. Forty- or fifty-micron sections were cut on a freezing microtome. Every section of interest was stained with cresyl violet (see Fig. 4). In all cases, minimal distortion of the brain was observed and the shrinking factor was of the order of 10%. Layers were identified according to classical criteria (Lund, 1973; Bullier & Henry, 1980; Lund et al., 1981). Identification of the laminar position was not possible for four recording sites (ten latencies) in V1. Recording sites that were found to be located in layer 4A were counted as belonging to the supragranular layers. In V2, there were 120 latencies from neurons recorded in brains cut parallel to the surface to reveal cytochrome oxidase-rich bands and for which laminar identification was not possible. This explains the differences in sample sizes between Tables 1 and 3–4 and between Tables 2 and 5–6.

Table 2. *Latencies in area V2 in ms*[a]

	V2 Total	ON su	ON mu	OFF su	OFF mu
Mean (ms)	85.7	87	81.4	94.1	86.9
Std. Dev.	30.6	26.9	29.8	32.1	33
S.E.M.	1.5	2.8	2.3	4.4	3.1
Count	422	94	161	53	114
Minimum	37	43	37	42	37
Maximum	205	177	205	173	203
Median	79	85	75	88	79.5

[a]Std. Dev.: Standard deviation; S.E.M.: Standard error of the mean.

Table 3. *Latencies of multi-unit (mu) activity in layers of area V1*[a]

	Supragranular	4B	4Cα	4Cβ	Infragranular
Mean (ms)	83.3	61.2	55.4	77	70.1
Std. Dev.	24.7	17.7	17	15.4	23.1
S.E.M.	2.1	2.4	4.3	3.3	4.0
Count	137	53	16	22	33
Minimum	35	31	27	57	42
Maximum	187	124	79	115	153
Median	80	58	58.5	72	63

[a]Std. Dev.: Standard deviation; S.E.M.: Standard error of the mean.

Table 5. *Latencies of multi-unit activity (mu) in layers of area V2*[a]

	Supragranular	Layer 4	Infragranular
Mean (ms)	89.9	93.1	78.8
Std. Dev.	32.8	32.3	32.8
S.E.M.	3.3	5.0	4.5
Count	100	41	54
Minimum	43	55	37
Maximum	203	188	205
Median	81	89	70.5

[a]Std. Dev.: Standard deviation; S.E.M.: Standard error of the mean.

Results

Data were obtained from 29 penetrations in six monkeys. Latencies were measured for 166 sites in V1 and 184 sites in V2. For each recording site, latencies for flash onset and offset, and for single (su) and multi-units (mu) were measured. Therefore, each recording site could yield up to four latencies. Responses to onset of the flash were obtained in 227 cases for V1 (83 su and 144 mu) and 254 cases for V2 (94 su and 161 mu). Latencies to flash offset were measured in 185 cases in V1 (62 su and 123 mu) and 167 cases in V2 (53 su and 114 mu). Approximately half of the sample (three monkeys, 79 recording sites in V1 and V2) was obtained during paired recordings in areas V1 and V2. In addition, the first response to the flash could consist in a decrease of firing, after which an excitatory response could be observed or not. Such responses were obtained in 70 cases in V1 (ON su: 6; ON mu: 20; OFF su: 16; OFF mu: 28) and 75 cases in V2 (ON su: 6; ON mu: 10; OFF su: 21; OFF mu: 38).

In most cases, the excitatory responses to flash consisted of a transient increase in firing followed by a more or less sustained elevation of activity (Figs. 1B and D-F). More rarely, we observed sustained responses such as the one illustrated in Fig. 1C. In 24 instances of recordings in V1, we observed high-frequency oscillatory responses (Fig. 1A) similar to those reported by Maunsell and Gibson (1992). Such oscillations were almost exclusively (20/24 cases) found in layers 4B, 4Cα, and 4Cβ. Sixteen out of the 20 cases of layer 4 oscillatory responses were observed in layers 4B and 4Cα. Earlier recordings suggest a retinal origin for these oscillations (Doty & Kimura, 1963).

We sometimes observed a decrease of firing in response to light ON or OFF (Fig. 2). Two types of responses were observed. The first was a complete suppression of activity (Fig. 2A). This type of response was associated with a sustained response to the flash of opposite polarity. The distribution of the latencies of such suppressive responses is presented in Fig. 2E for areas V1 and V2. The second type consisted in a *transient* decrease in firing rate that was usually, but not always, followed by an excitatory response (Figs. 2B and 2C). The frequency distribution of the latencies of such inhibitory responses is presented in Fig. 2D for both areas.

Frequency-distribution histograms of the latencies of excitatory responses in V1 and V2 to stimulus onset and offset are presented in Fig. 3 and the average values are given in Tables 1 and 2. As observed earlier in the cat retina (Bolz et al., 1982), OFF latencies are slightly longer than ON latencies (Tables 1 and 2), although the difference is significant only in V1 ($P < 0.001$ in a Mann-Whitney U test) and not in V2 ($P = 0.1$). Note that the earliest latencies of inhibitory responses presented in Fig. 2 are almost as short as the first excitatory responses (Fig. 3).

V1 and V2 excitatory latencies are easily compared in Fig. 3 in which the gray histograms correspond to V1 values and the thick line histograms to V2 latencies. Latencies are shorter in V1 than in V2 ($P = 0.0006$ Mann-Whitney U (MWU) test on the total sample). This is especially the case of ON latencies which are shorter in V1 by 10 ms on the average ($P = 0.03$ for single units and $P = 0.002$ for multi-units). Latencies to stimulus offset also tend to be shorter in V1 than in V2, as shown by the histograms presented on the right of Fig. 3. However, the distributions are not significantly different ($P = 0.15$ in

Table 4. *Latencies of single-unit activity (su) in layers of area V1*[a]

	Supragranular	4B	4Cα	4Cβ	Infragranular
Mean (ms)	92.3	63.8	58.6	87.4	87.2
Std. Dev.	25.1	18.2	16.7	24.7	26.1
S.E.M.	3	3.2	5.6	7.1	6.1
Count	69	33	9	12	18
Minimum	36	31	33	53	51
Maximum	158	118	80	120	125
Median	91	61	65	85	86.5

[a]Std. Dev.: Standard deviation; S.E.M.: Standard error of the mean.

Table 6. *Latencies of single unit activity (su) in layers of area V2*[a]

	Supragranular	Layer 4	Infragranular
Mean (ms)	101	96.1	84
Std. Dev.	28.7	27.5	30.9
S.E.M.	4.2	5.2	5.4
Count	46	28	33
Minimum	55	58	42
Maximum	177	173	168
Median	94	93	78

[a]Std. Dev.: Standard deviation; S.E.M.: Standard error of the mean.

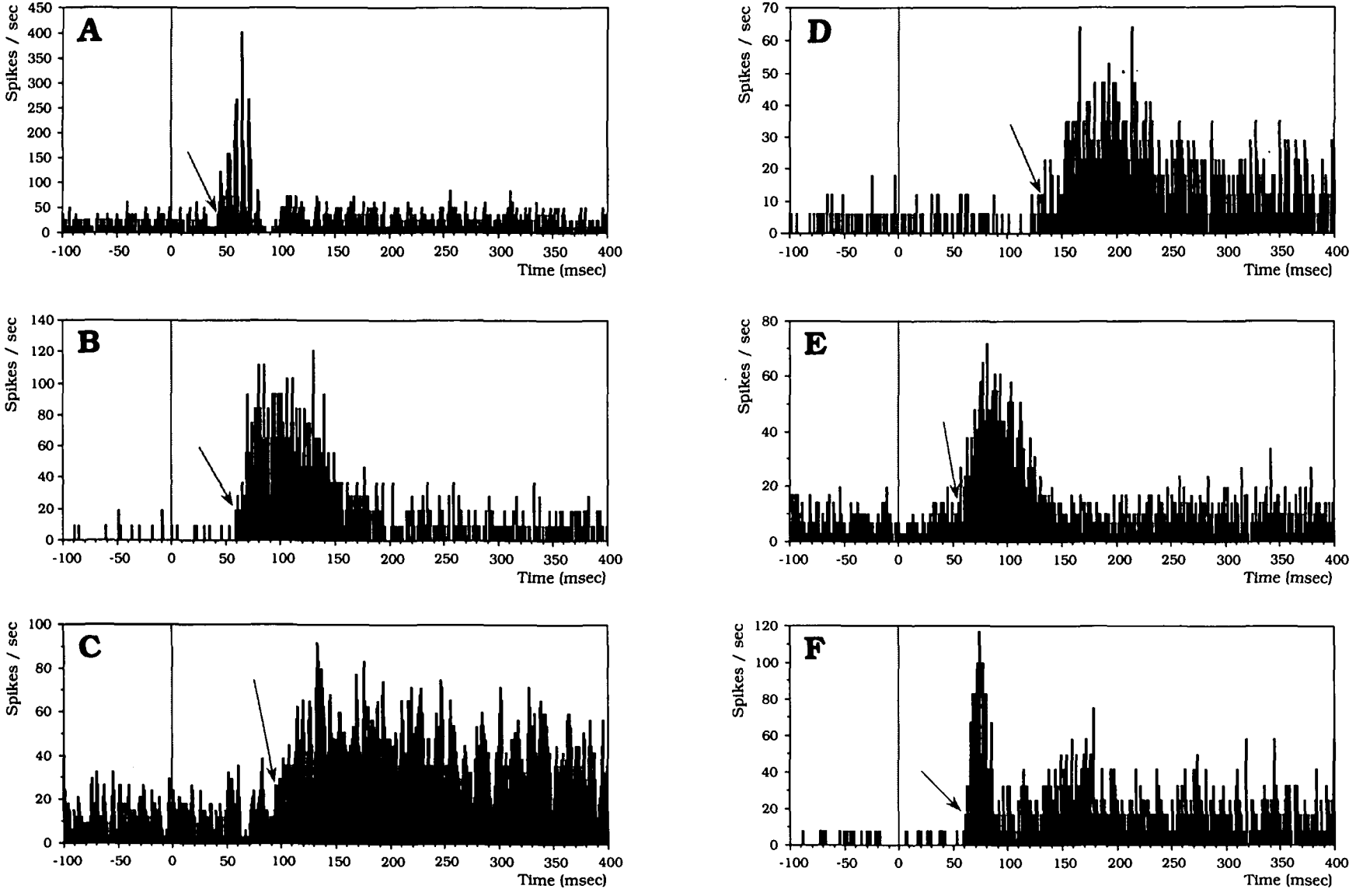

Fig. 1. Examples of responses to flashes of a small spot of light. Onset or offset trigger is at time zero. 1-ms bin width in all cases. Arrows indicate the bin corresponding to the measured latency (not corrected for opening and closing times of the shutter). A: Oscillatory multi-unit ON response in layer 4B of area V1 (MM5 14D24D02). B: Short-latency ON response in layer 4B single unit (MM10 12H). C: Long-latency ON response in multi-units in layer 4Cβ (MM10 11AV). D: Long-latency OFF response in supragranular layer multi-units of V1 (MM10 11A). E: Short-latency OFF response in multi-unit recording in supragranular layers of area V2 (MM8 25H). F: Short-latency single-unit ON response in infragranular layers of area V2 (MM5 14124102).

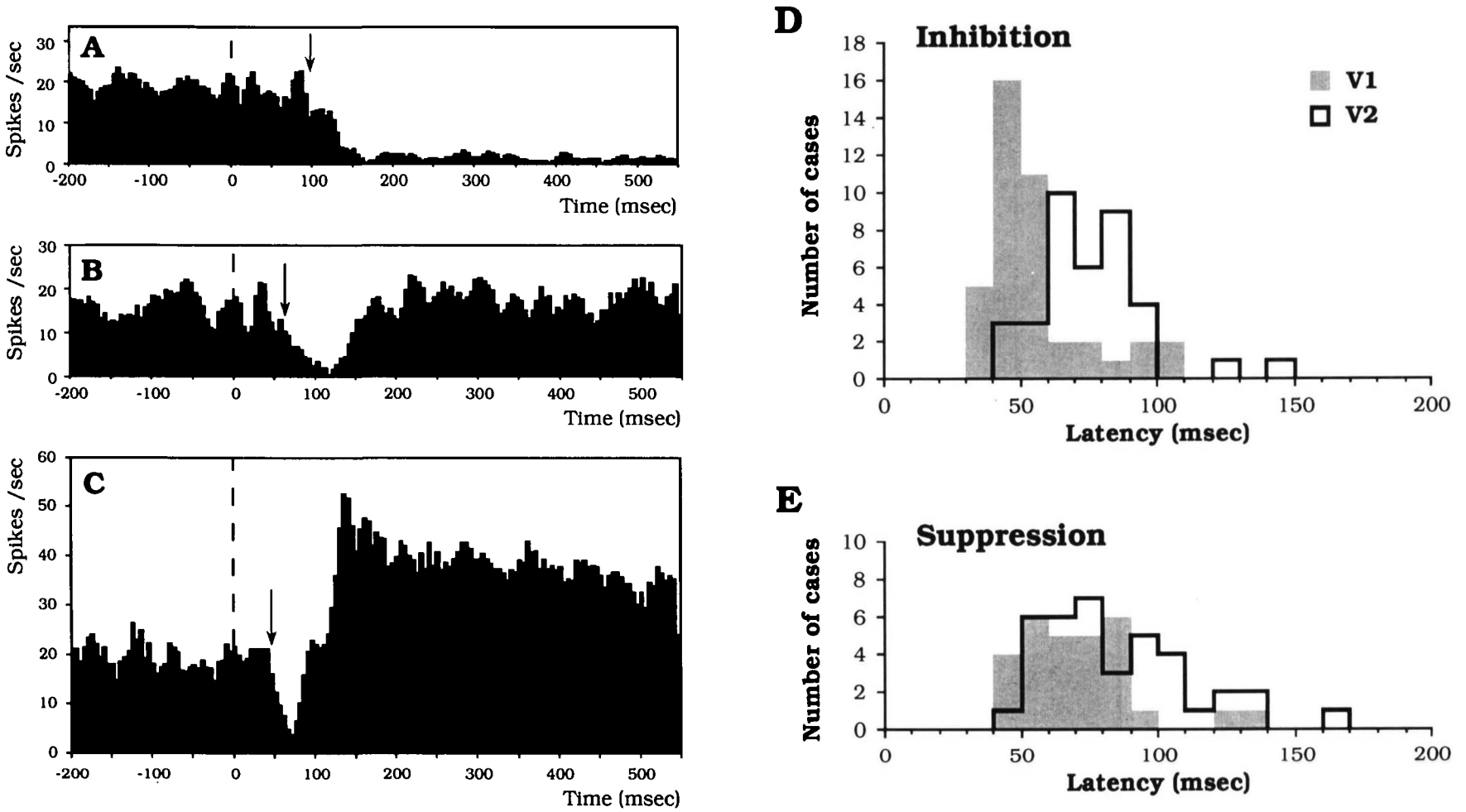

Fig. 2. Suppression and inhibitory responses. 5-ms bin width in A–C. Onset or offset trigger is at time zero. Arrows indicate the bin corresponding to the measured latency (not corrected for opening and closing times of the shutter). A: Example of a suppression type of response of multi-unit in V1 to OFF (MM7 12O21O02). B: Example of an inhibitory type of response not followed by an excitatory response. Multi-unit OFF response in V2 (MM5 14824702). C: Example of an inhibitory response followed by an excitatory response. ON response single unit in V1 (MM7 13522502). D: Frequency-distribution histogram of inhibitory responses in V1 and V2 (single- and multi-units pooled). The mean latency (±S.D.) for inhibitory responses in V1 is 57.7 ± 17.9 (n = 41) and 75.9 ± 20.2 in V2 (n = 37). E: Frequency-distribution histogram of latencies of suppression response in V1 and V2 (single- and multi-units pooled). The mean latency (±S.D.) for suppression type responses is 70.7 ± 21.7 (n = 29) in V1 and 85.3 ± 27.5 (n = 38) in V2.

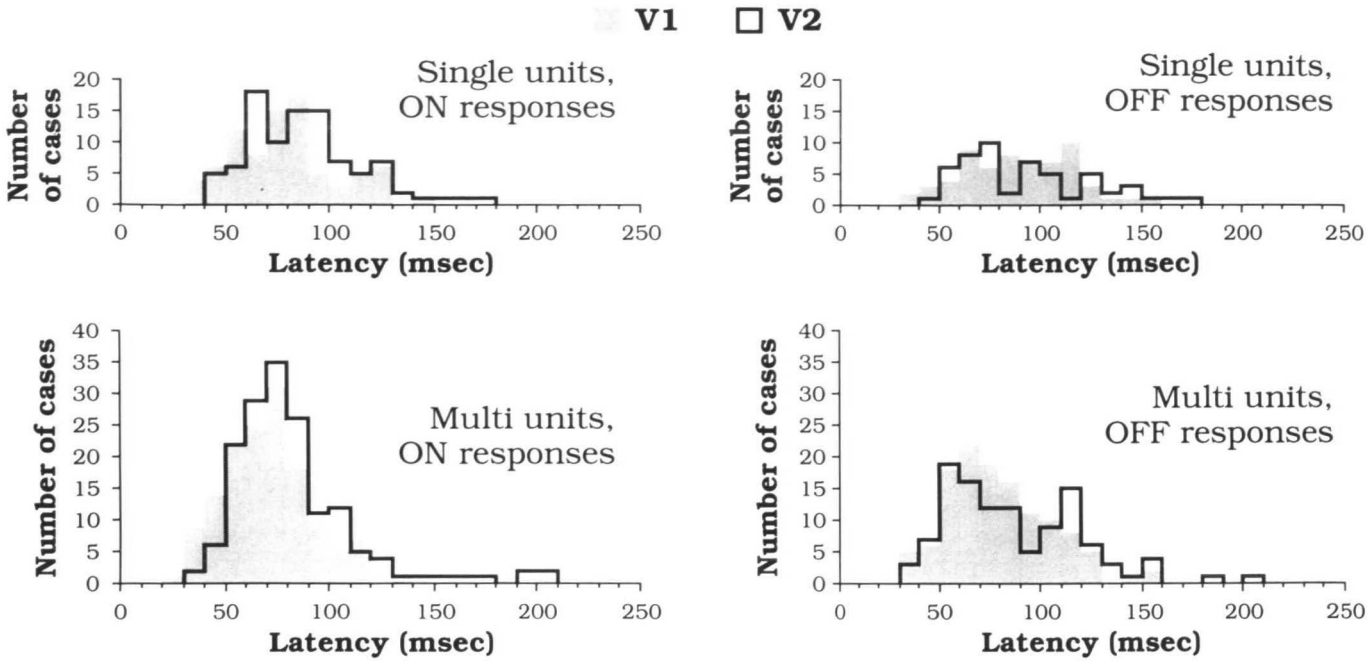

Fig. 3. Frequency distributions of latencies in V1 and V2 for excitatory responses of single units and multi-units to onset (ON) or offset (OFF) of a small spot of light.

MWU test). Significant differences between V1 and V2 OFF latencies were observed only for cases obtained from paired multi-unit recordings ($P = 0.005$ in Wilcoxon signed rank test).

Although, as a population, V2 neurons tend to respond later to visual stimulation than V1 neurons, there is a substantial overlap between the distributions of V1 and V2 latencies: for the whole population, when 50% of the V1 neurons have responded, 43% of the V2 neurons have already responded. Similarly, when 50% of the V2 neurons have responded, there are still 43% the V1 neurons that have not started responding. This means that, for a given delay after stimulation, a large proportion of neurons in V1 have not yet responded while activity is already being processed in V2. This suggests that information is rapidly sent to V2 while processing is carried on in V1. To gain a better understanding of the flow of information processing in the two areas, we made a laminar analysis of the latencies of excitatory responses to visual stimulation in areas V1 and V2. This was achieved by reconstructing the laminar positions of neurons encountered in the electrode penetrations from the locations of electrolytic lesions on Nissl-stained sections (Fig. 4).

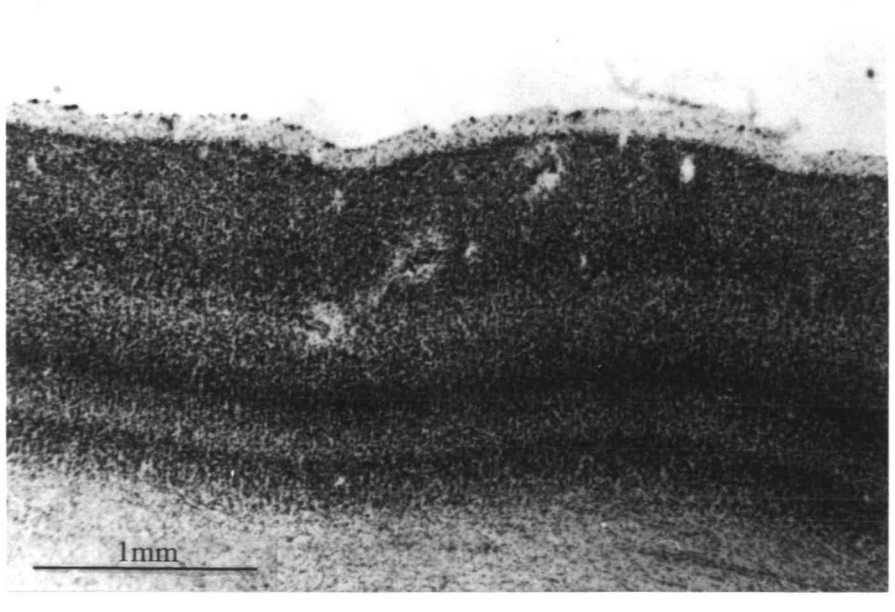

Fig. 4. Nissl-stained section of area V1 with lesions located in supragranular layers and in layer 4B. Scale bar = 1 mm.

Table 7. *Levels of significance (P values) in Mann-Whitney U tests for latencies of multi-unit activities (mu)*

	V1 Supragranular	V1 4B	V1 4Cα	V1 4Cβ	V1 Infragranular	V2 Supragranular	V2 Layer 4
V1 Supragranular	–	<0.0001	<0.0001	0.2	0.001	0.5	0.3
V1 4B	–	–	0.5	0.0002	0.09	<0.0001	<0.0001
V1 4Cα	–	–	–	0.001	0.07	<0.0001	<0.0001
V1 4Cβ	–	–	–	–	0.06	0.1	0.07
V1 Infragranular	–	–	–	–	–	0.0004	0.0004
V2 Supragranular	–	–	–	–	–	–	0.5
V2 Layer 4	–	–	–	–	–	–	–

To keep a sufficiently large sample in each lamina, we pooled together the results from ON and OFF latencies. These two populations are not statistically different in the different laminae of V1 and V2, with the exception of the supragranular layers of V1 ($P = 0.001$ for su and $P = 0.02$ for mu). The latency data are presented in Tables 3–6 for single and multi-units separately. Statistical comparisons are given in Tables 7–8. The latency differences between layers that are significant for the multi-unit recordings are also significant for the single-unit recordings, except when the comparisons involved the infragranular layers of V1.

Fig. 5 presents the frequency-distribution histograms of the latencies of single- and multi-unit recordings in the different layers of V1 (on the left) and V2 (on the right). It is clear that, in area V1, the shortest latencies are observed in layers 4Cα and 4B and are shorter than the latencies recorded in layer 4Cβ. Latencies in supragranular and infragranular layers have more widespread distributions, containing short as well as long latencies.

Contrary to what is observed in area V1, the shortest latencies in V2 are observed in the infragranular layers and not in layer 4 (Fig. 5, right). Latencies in layer 4 are longer than in the infragranular layers and similar to those recorded in the supragranular layers. That latencies in V2 are shorter in infragranular layers than in layer 4 was surprising given the fact that the visual information is supposed to be relayed through layer 4 which is the major recipient of the input from V1 (Rockland & Pandya, 1979; Lund et al, 1981; Weller & Kaas, 1983). Therefore, we checked that this was not due to small errors in the reconstructions of the electrode tracks. For this, we plotted the latencies of V2 neurons as a function of depth with respect to the middle of layer 4 (not shown). This scattergram showed that many short latencies were recorded at deep levels in cortex, presumably within layer 6. Thus, it is very unlikely that the short latencies observed in infragranular layers resulted from an accidental misplacement of the recorded sites in the electrode tracks. Also, short latencies in infragranular layers of V2 were observed in every animal.

Fig. 6 summarizes the data concerning the laminar distributions of latencies by presenting the cumulative distributions in different cortical laminae of areas V1 and V2. In area V1, two groups can be distinguished: layers 4B and 4Cα with short latencies and layers 4Cβ and supragranular layers with longer latencies. The infragranular layers occupy an intermediate position. Layers 4Cα and 4B have similar latencies which are significantly smaller than those in 4Cβ (Tables 7–8). Latencies in layers 4Cα and 4B are also significantly smaller than in supragranular layers and infragranular layers (Tables 7–8). The latencies of layer 4Cβ are not significantly different from those of supragranular and infragranular layers (Tables 7–8).

Comparison of the cumulative distributions of latencies in layers 4Cα and 4B of V1 (Fig. 6A) shows a clear similarity between these two distributions except for the earliest component in layer 4Cα. It is also interesting that most latencies in supragranular layers are longer by 5–10 ms than those in layer 4Cβ, with the exception of a small population of neurons (less than 10%) with latencies as short as in layer 4B. Latencies in infragranular layers appear to be composed of two components, one longer by about 5 ms than latencies in layer 4B and another with latencies slightly longer than in layer 4Cβ.

In area V2 (Fig. 6B), the latencies of infragranular layer neurons are shorter by about 10 ms than those of neurons in layer 4 and in supragranular layers (Tables 5–6 and 7–8). Latencies of neurons in layer 4 are not significantly different from those in supragranular layers (Tables 7–8). A small population of infragranular layer neurons in V2 (below the 10% level in Fig. 6B) have latencies that are only slightly longer than the earliest latencies in layer 4B of V1.

As expected from the presence of projections from both the supragranular layers and layer 4B of V1 to layer 4 of V2, neurons in layer 4 of V2 have on average longer latencies than in the supragranular layers and in layer 4B of V1 (Tables 3–5

Table 8. *Levels of significance (P values) in Mann-Whitney U tests for latencies of single-unit activities (su)*

	V1 Supragranular	V1 4B	V1 4Cα	V1 4Cβ	V1 Infragranular	V2 Supragranular	V2 Layer 4
V1 Supragranular	–	<0.0001	0.0001	0.5	0.5	0.2	0.7
V1 4B	–	–	0.7	0.003	0.003	<0.0001	<0.0001
V1 4Cα	–	–	–	0.01	0.02	<0.0001	0.0005
V1 4Cβ	–	–	–	–	0.9	0.2	0.4
V1 Infragranular	–	–	–	–	–	0.1	0.4
V2 Supragranular	–	–	–	–	–	–	0.5
V2 Layer 4	–	–	–	–	–	–	–

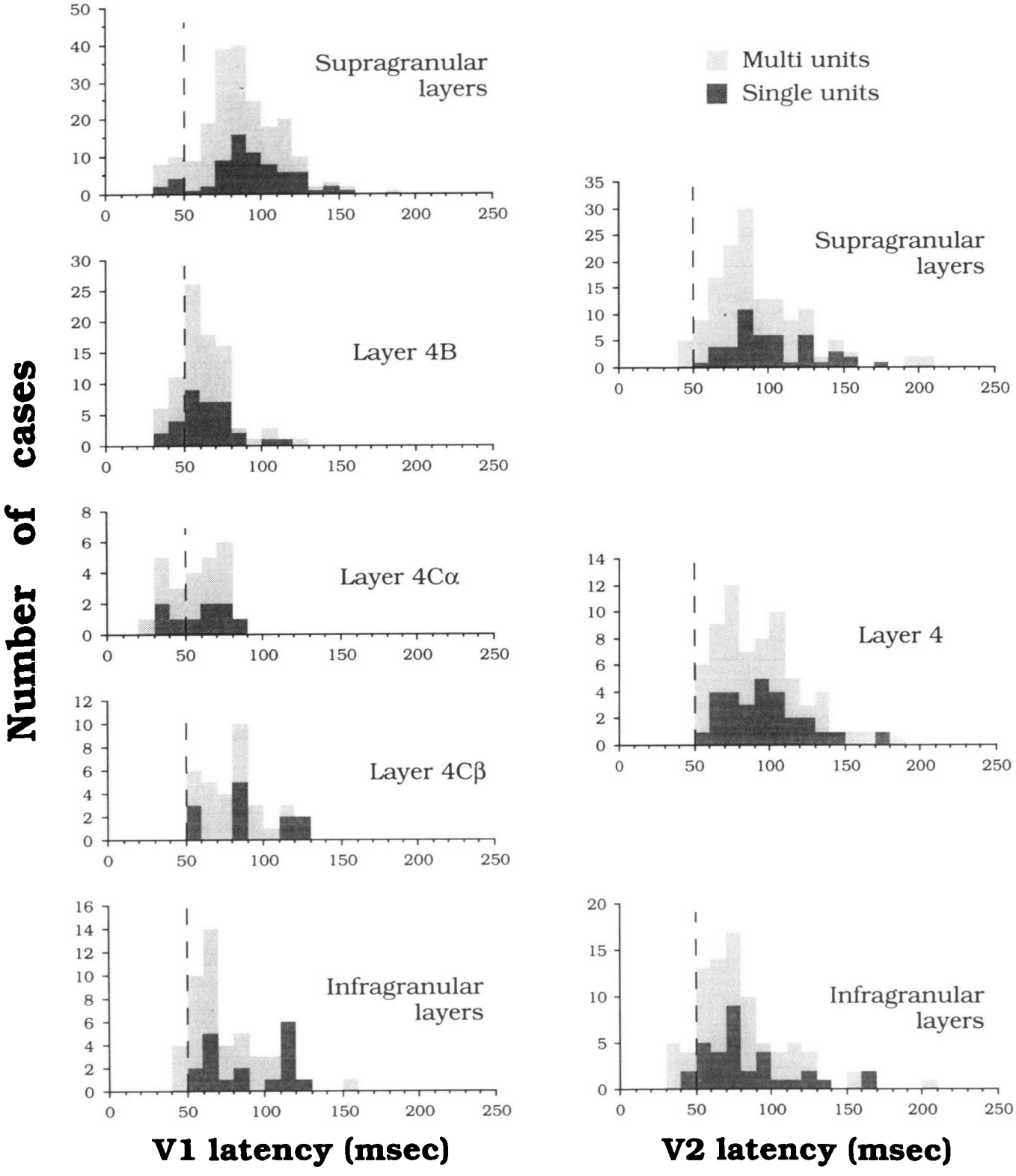

Fig. 5. Frequency distribution of latencies in different layers in areas V1 and V2. ON and OFF responses have been pooled.

and 4–6). However, the difference fails to reach the significance level between the supragranular layers of V1 and layer 4 of V2 (Tables 7–8). Latencies of supragranular layers neurons are not statistically different in the two areas (Tables 7–8). Similarly, neurons in the infragranular layers of V2 display latencies which are not significantly longer than those in the infragranular layers of V1 (Tables 7–8).

It is apparent in Fig. 6 that the major reason why the aver-

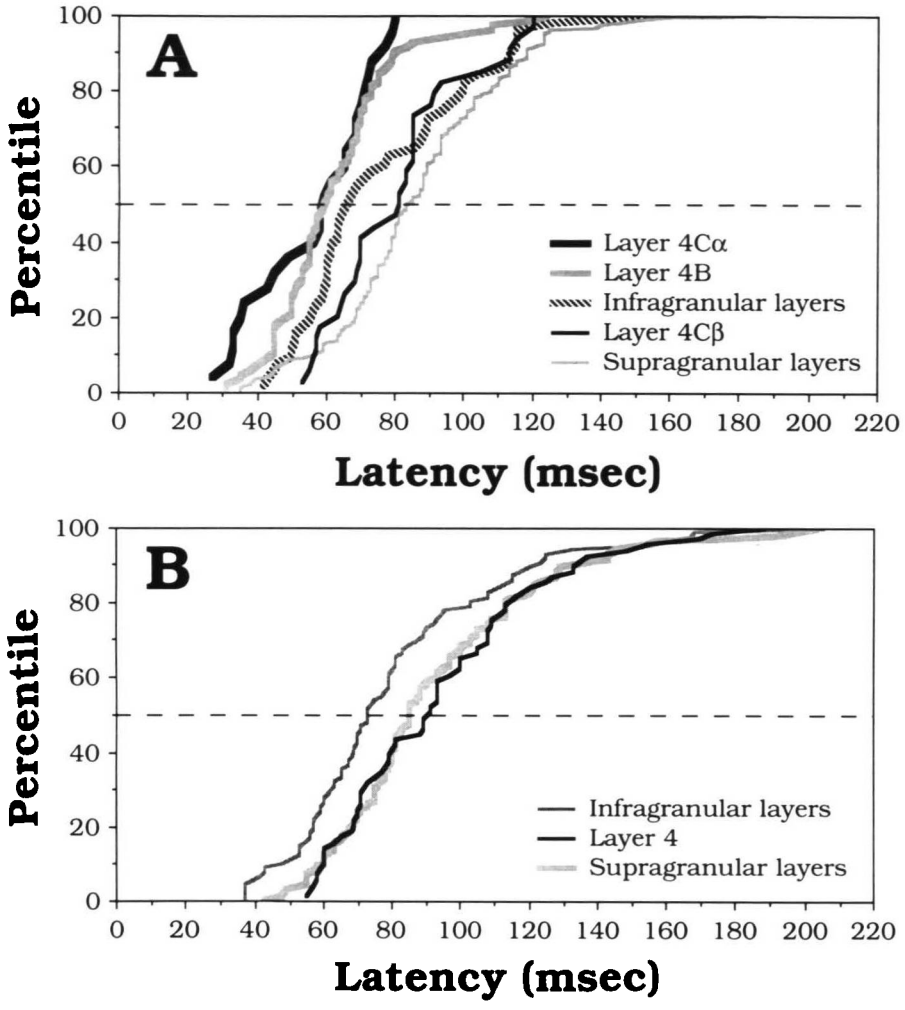

Fig. 6. Summary of latencies in different layers of areas V1 and V2. Cumulative distribution of latencies in different layers of V1 (A) and V2 (B). Most of the shortest latencies are found in layers 4Cα and 4B of V1. Latencies do not differ significantly in layer 4Cβ, infragranular and supragranular layers of V1. The shortest latencies in area V2 are observed in infragranular layers. Latencies in infragranular layers of V2 do not differ significantly from those found in infragranular layers in V1. Latencies in supragranular layers of V2 do not differ significantly from those in the supragranular layers of V1.

age latency of V1 neurons is shorter than that of V2 neurons is the presence of short latencies in layers 4Cα and 4B of V1. In fact, there is no significant difference between the latencies of V2 neurons and those of the V1 neurons located outside layers 4Cα and 4B ($P = 0.7$ in MWU test).

The relationship between response selectivity and latency was also investigated within areas V1 and V2. No significant differences were found in V1 between color- and noncolor-selective units, as shown earlier by Creutzfeldt et al. (1987), between direction-selective and nondirection-selective units and between orientation-selective and nonorientation-selective units. In contrast, we found significant differences for several of these properties in V2 (Fig. 7). Thus, color-selective units in V2 have longer latencies that noncolor-selective cells (Fig. 7B; mean values 93.8 and 81.6 ms, $P = 0.04$). The latency difference was significant only for ON latencies (means 93.4 and 79 ms, $P = 0.02$). We also observed a significant difference between oriented *vs.* nonoriented cells in V2 (Fig. 7A, mean values 79.1 and 92.4 ms, $P < 0.0001$). In this case also, the difference was significant only for ON latencies (mean values 76.7 and 92.8 ms, $P = 0.0001$; $P = 0.02$ for su, $P = 0.002$ for mu).

Discussion

We measured the latencies of neurons in areas V1 and V2 in response to stimulation by a small flashing stimulus and found similar latencies in the infragranular and supragranular layers of both areas. We also show that V1 neurons driven by magnocellular layers of the LGN have shorter latencies than those activated by the parvocellular layers.

Methodological issues

To measure the latency to visual stimulation, we used a statistical method based on the assumption of a Poisson distribution for the spontaneous activity of visual neurons. This appears to be a reasonable hypothesis (Smith & Smith, 1965; Légendy & Salcman, 1985). An alternative method would have been to use an algorithm based on signal detection theory that is independent of the statistical distribution of the arrival times of spikes. This would not change substantially our conclusions because of the large number of presentations and the good signal-to-noise ratio of most of our PSTHs (Fig. 1). As mentioned below, changing the bin width and the number of presentations hardly changed the values of the latencies and it can be expected that a similar result would be obtained with a method based on signal detection theory.

We used a large number of presentations, particularly in case of noisy records, and we optimized the bin width to obtain the best precision and signal-to-noise ratio in our latency measurements. We were concerned that this variability in our procedures might introduce some bias. We therefore recomputed the laten-

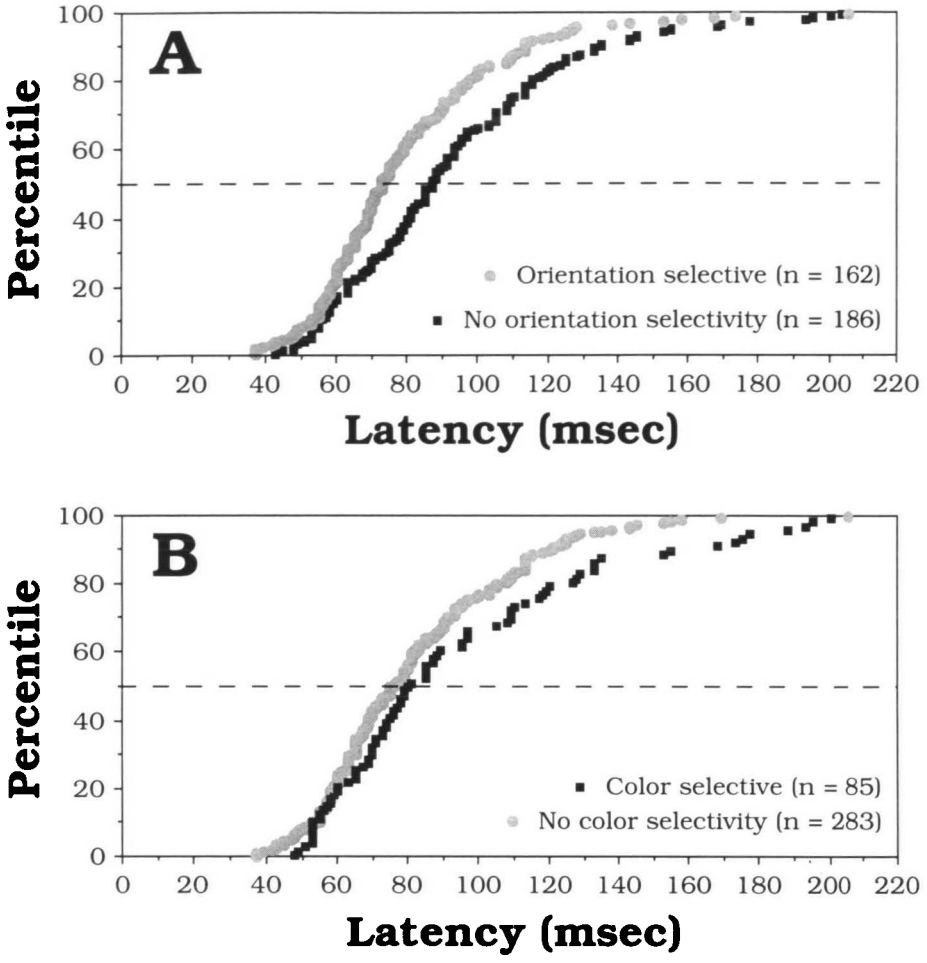

Fig. 7. Cumulative percentiles for latencies of neurons with different types of receptive-field properties in area V2. ON and OFF responses have been pooled.

cies with a fixed number of presentations (100) and a fixed bin width (5 ms) for the recordings with number of presentations larger than 100. Only a small number of latencies (15/740) could no longer be measured due to increased noisiness. As mentioned in the Methods section, the latencies calculated by the two methods were highly correlated ($r = 0.95$). In addition, the latencies calculated with fixed bin width and number of presentations were within a few milliseconds of the original values (between 3 and 0.5 ms, after splitting for single and multi-units, and for the different layers). Finally, the population differences that were significant with the former method remained significant with the latter. This is not unexpected since the latency differences between layers and between areas reported in this paper are of the order of tens of milliseconds. Therefore our conclusions are unlikely to depend on the methods of latency measurement.

To compare latencies across areas and layers, we had to use a common standard stimulus and not the optimal stimulus for each neuron. One may wonder whether using the optimal stimulus would have led to different conclusions. Although we did not systematically test the influence of all of the stimulus parameters on latency, our comparisons between the optimal color and a white stimulus for color-selective cells did not lead to a significant latency difference (Methods section). A lack of variation of the response latency with the color of isoluminant stimuli was also reported in an earlier study (Creutzfeldt et al., 1987). We also compared the latencies to small bars and small squares and again found no significant difference between the two populations.

It has been shown earlier that the latency varies with the orientation of the stimulus (Celebrini et al., 1993). This is not a problem in our study since we stimulated neurons at the optimal orientation. The use of short stimuli in our study made it possible to activate neurons with strong end-zone inhibition but may produce long latencies in neurons requiring elongated stimuli for optimal activation. This could have been a potential difficulty had we reported long latencies in layer 6 which contains such neurons (Gilbert, 1977; Grieve & Sillito, 1991). Since, instead, we found numerous layer 6 neurons with short latencies, it is unlikely that this procedure induces a systematic bias in our results.

Latencies in area V1

The latencies measured in V1 in the present study cover the same range as measured in the reports of Raiguel et al. (1989) using moving bars in anesthetized and paralyzed monkeys. Nevertheless, the mode of the distribution is between 70 and 80 ms in the present study instead of 100 ms in the report of Raiguel et al. This may result from the different methods of stimulation since we used flashes and Raiguel and collaborators used moving bars. The shortest V1 latencies are comparable in the

present report and in the studies in awake behaving monkeys (Celebrini et al., 1993; Maunsell & Gibson, 1992; Knierim & Van Essen, 1992; Vogels & Orban, 1990). On the other hand, a larger number of latencies beyond 100 ms was observed in the anesthetized preparation (Raiguel et al., 1989; present report) than in most reports on the awake animal (Celebrini et al., 1993; Maunsell & Gibson, 1992; Knierim & Van Essen, 1992; see however, Vogels & Orban, 1990 for latencies longer than 140 ms). This difference may result from different recording characteristics of microelectrodes used in awake and anesthetized animals or from the possible effects of anesthetic agents on response latencies. However, this does not invalidate our conclusions which are based on comparisons between latencies of neurons recorded, sometimes simultaneously, in the same preparation.

The laminar distribution of latencies in V1 shows that the shortest values are observed in layers 4Cα and 4B. It is known that these layers are activated by neurons of the magnocellular layers of the LGN (Hubel & Wiesel, 1972; Lund & Boothe, 1975; Bullier & Henry, 1980; Fitzpatrick et al., 1985). The average latencies measured in the parvo-recipient layer 4Cβ are 20 ms longer than those observed in layer 4Cα. That the shortest latencies in area V1 are found in the magno-recipient layers 4Cα and 4B is in keeping with the results of Marrocco (1976). This author found that, in the macaque LGN, neurons with transient responses and lack of color selectivity, which are likely to be located in the magnocellular layers (Dreher et al., 1976; Schiller & Malpeli, 1978), have visual latencies which are shorter on average by 20 ms than the latencies of color-opponent neurons which presumably belong to parvocellular layers (Wiesel & Hubel, 1966; Dreher et al., 1976; Schiller & Malpeli, 1978). This is also in keeping with the mean onset latencies reported by Irvin et al. (1986) in the galago, who found that neurons in the parvocellular layers of the LGN are responding 14 ms later than those of the magnocellular layers. Our results are also consistent with those of Maunsell and Gibson (1992) who showed that lesions in the magnocellular layers of the LGN lead to a disappearance of the shortest latencies in area V1. The results of Maunsell (1987) demonstrating shorter latencies for V1 neurons with transient responses to visual stimuli is also in agreement with our observation of shorter latencies in neurons activated by the magnocellular stream.

The latency difference observed between the parvocellular and magnocellular pathways in our results is observed in average and minimum latencies (Fig. 6) and does not change when latencies were recalculated with fixed bin width and number of presentations (see Methods section). It may be thought that a latency difference of 20 ms between neurons in layers 4Cα and 4Cβ is unexpected because differences of axonal conduction speed between magnocellular and parvocellular neurons of the LGN would predict a difference of only a few milliseconds (Schiller & Malpeli, 1978; Bullier & Henry, 1980). The latency difference observed in cortex is already present in the LGN, as mentioned above, and it must have its origin in the retinal processing time. Indeed, Bolz et al. (1982) reported a latency difference of 10–15 ms between X and Y ganglion cells in the cat retina.

Our finding of short latencies in 4B and long latencies in 4Cβ differs from the conclusions of Maunsell and Gibson (1992), who stated that the shortest latencies were found only in layer 4C. However, the precision of the laminar analysis of that study is limited by the fact that the authors, because they used a chronic preparation, relied mostly on the depth relative to the entrance of the microelectrode in layer 4C, as judged from the physiological responses (high spontaneous activity, lack of orientation selectivity). It is known that, in contrast to layer 4Cβ, layer 4Cα contains many orientation-selective neurons (Bullier & Henry, 1980; Livingstone & Hubel, 1984). It is therefore possible that what Maunsell and Gibson judged to be the upper limit of layer 4C was in fact that of layer 4Cβ. This would explain why the shortest latencies were mostly found above their estimate of the upper limit of layer 4C. These would correspond to our finding of short latencies in layers 4Cα and 4B.

According to Maunsell and Gibson, the shortest latencies are found in layer 4C and latencies of neurons in supragranular layers are longer by about 10 ms. They interpreted this finding as meaning that there is a delay of 10 ms to activate cells in upper layers. In contrast, our results suggest that part of the latency difference between the thalamo-recipient layers and supragranular layers can be accounted for by the presence of a slow parvocellular pathway and a faster magnocellular pathway. This does not, however, rule out a long delay for the transfer of the visual information between the different layers; indeed, there is a 6-ms latency difference on average between responses in layer 4B and its input layer 4Cα, which, although not significant, is likely to correspond to the transmission delay between them. Delays cannot be determined for the other layers since they are likely to receive mixed inputs of parvocellular and magnocellular origin.

The earliest responses in supragranular layers are displaced with respect to the earliest responses in layers 4Cα and 4B only by a few milliseconds (Figs. 5–6, Tables 3–4). These early latencies in the supragranular layers are certainly due to magnocellular layer signals relayed from layers 4Cα and 4B. This is supported by recent anatomical studies which have demonstrated the presence of projections from layer 4B to the supragranular layer blobs and a projection from the middle of layer 4C to the interblob region (Lachica et al, 1992; Yoshioka et al., 1994). Nevertheless, most latencies in supragranular layers are longer by a few milliseconds than those in layer 4Cβ. This is in keeping with the conclusion derived from two deoxy-glucose (DG) mapping that the supragranular layers of V1 are mainly under the influence of the parvocellular stream relayed in layer 4Cβ (Tootell et al., 1988; see, however, Lachica et al., 1992; Nealey & Maunsell, 1994). Long response latencies in the supragranular layers could also reflect drive of their neurons by thalamic afferents originating in the intercalated (or S or K) layers of the LGN (Fitzpatrick et al., 1983; Casagrande, 1994; Hendry & Yoshioka, 1994). In the LGN of the galago, neurons belonging to these layers have been shown to have latencies even longer (by more than 10 ms) than those of the parvocellular layers (Irvin et al., 1986).

Two groups of responses can be distinguished in the infragranular layers, one for which latencies are shorter than, and the other with latencies slightly longer than those of layer 4Cβ (Fig. 6). These two groups could possibly reflect different populations of neurons connected to parvocellular and magnocellular inputs (Blasdel & Lund, 1983; Hendrickson et al., 1978). These two groups could also have different outputs: layer 6 cells are (partially) segregated depending on whether they are projecting to the parvocellular or the magnocellular layers of the LGN (Fitzpatrick et al., 1994). Schiller et al. (1979) have shown that the layer 5 cells driving the superior colliculus neurons are themselves dominated by inputs of magnocellular origin.

The first response to flashes could also consist in an inhibition of the activity (Fig. 2). The detection of these responses required that the neurons have an important spontaneous activity. This was not always the case, and the correspondingly small number of observations precluded a detailed analysis. We have distinguished two groups in this type of response. The first one ("suppression") corresponds to the cessation of the tonic firing after stimulus onset or offset in cells having a sustained activity. The transient nature of the response and the fact that it was often followed by an excitatory response suggest that the other group ("inhibition") is more likely to reflect a real GABAergic inhibition. One could wonder whether the longest response latencies are always associated with the presence of inhibition. This is not the case. In layer 4Cβ, there are only three cases of inhibition followed by excitatory responses. In the supragranular layers of V1, where the largest number of inhibitory responses is observed, the distribution of latencies for excitatory responses preceded by inhibition overlaps that of excitatory responses without inhibition (not shown). In V2, the number of cases in which there is an inhibitory response preceding an excitatory one is very low: 17 cases, compared to 404 cases without preceding inhibition.

A transient inhibition as the first neuronal response to flash has been documented with intracellular recordings in cat visual cortex (Creutzfeldt & Ito, 1968; Creutzfeldt et al, 1969). In the somatosensory cortex of the mouse, a bimodal distribution of latencies to mechanical stimulation has been reported in layers 3 and 4 (Welker et al., 1993). It has been suggested that the two latency groups correspond to different functional cell types. The earliest latencies would correspond to activation of inhibitory interneurons, whereas longer latencies are recorded from excitatory neurons. In layer 4 of the primary visual cortex, thalamocortical axones synapse not only on excitatory, but also on inhibitory cells (Freund et al., 1985, 1989). The presence of early inhibitory responses observed in some neurons of V1 suggests that, in the visual cortex also, the earliest neurons to be activated may be the inhibitory cells.

Latencies in area V2 and interactions between V1 and V2

Excitatory latencies in area V2 are longer than in area V1, thus confirming the conclusions of Raiguel et al. (1989), which were based on a smaller sample. Although the mean latency is longer in layer 4 of V2 than in the supragranular layers of V1, the difference fails to reach significance level. This may reflect the possibility that the fastest responses observed in layer 4 of V2 are due to a drive from layer 4B of V1. This is in keeping with the known connectivity between these two areas (Livingstone & Hubel, 1987) and the latencies of V1 and V2 neurons following electrical stimulation of the optic radiations (Mitzdorf & Singer, 1979).

Within area V2, we found no significant latency difference between layer 4 and the supragranular layers. In area V2, the basal dendrites of layer 3 neurons freely cross the border between layer 3 and 4 (Lund et al., 1981). In addition, the terminals of afferent axons from area V1 are not confined to layer 4 but also enter the lower portion of the supragranular layers (Lund et al., 1981). Altogether, these anatomical findings are consistent with the observation that latencies in the supragranular layers of V2 can be as short as those in layer 4.

The latencies in infragranular layers of V2 are shorter than in layer 4. This is somewhat unexpected since anterograde tracing studies have shown that the major target of the area V1 projection is layer 4 of V2 (Rockland & Pandya, 1979; Lund et al., 1981; Weller & Kaas, 1983). However, using anterograde transport of tritiated amino acids, Lund and her collaborators (1981) found a small V1 input at the border of layers 5 and 6 in area V2 (their Figs. 13A and 13B). This was confirmed by studies using the anterograde transport of Phaseolus Vulgaris that showed that axons travelling from V1 to V2 sometimes send collaterals to the infragranular layers of V2 (Rockland & Virga, 1990). It may be that these inputs correspond to a specific input from layer 4B or from the fast supragranular layer neurons of V1 to the infragranular layers of V2. In addition, in contrast to what is observed in V1, the apical dendrites of infragranular layer pyramidal cells cross layer 4 without any loss of dendritic spines (Lund et al., 1981). It is therefore possible for these neurons to be targeted by fast afferents from V1 terminating in layer 4.

It is remarkable that neurons of the infragranular layers of areas V1 and V2, which project to some common targets like the superior colliculus or the pulvinar, also have similar latencies to visual stimulation. This is in keeping with the idea that synchronization of neural activity is important for the processing of converging information in target neurons. The similar latencies found in infragranular layers of V2 and in infragranular layers of V1 also suggest that processing in these layers is done simultaneously. Neurons in these layers of V1 are as likely to be driven by some neurons in infragranular layers of V2 as they are to drive neurons in V2. That the activity of some V1 neurons depends on visual activity in V2 was in fact demonstrated in a physiological study by Sandell and Schiller (1982) who noted that 65% of the infragranular layer neurons of V1 were affected (in general, made less responsive to visual stimulations) by the inactivation of area V2.

In conclusion, our study shows that short latencies in V1 are mostly due to those found in the magno-recipient layers 4Cα and 4B. Latencies are similar in the supragranular layers of V1 and V2 and in the infragranular layers of V1 and V2. Thus, much of the processing occurs simultaneously in the two areas, presumably involving interactions mediated by reciprocal connections between them and inputs from common sources of afferents (Kennedy & Bullier, 1985). Despite the anatomical and physiological demonstrations that the activity of area V2 requires that of area V1, and although latencies to visual stimulation are on the average longer in V2 than in V1, comparison of the latencies measured in different layers of these two areas do not support a strictly serial processing of information between areas V1 and V2.

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