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Research note

Influence of layer V of area 18 of the cat visual cortex on responses of cells in layer V of area 17 to stimuli of high velocity

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Abstract. Focal blockade of restricted regions in layer V of area 18 was used to assess the contribution of this region to the responses to high-velocity stimuli of cells in retinotopically matched, layer V in area 17. In 40% of cases, blockade within area 18 revealed responses of area 17 cells to high-velocity stimuli to which they previously showed only poor responses. Stimulus specificity of the cells in area 17 was otherwise unaltered. All effects were reversible and repeatable. We suggest that a component of the output of layer V from area 18 normally suppresses the responses of retinotopically matched cells within area 17 to stimuli of high velocity, thereby enhancing the specificity of those cells to stimuli of low velocity

Key words: Area 18 – Area 17 – Layer V – Stimulus velocity – Cat

Introduction

The selectivity of cells in visual cortical areas 17 and 18 for stimulus velocity is known to differ markedly (Duysens et al. 1982), with many cells in area 18 responding well to stimuli of high velocity to which the majority of cells in area 17 are unresponsive. Layer V of each of these areas contributes to the large, descending pathway to the superior colliculus (SC) (Gilbert and Kelly 1975; Harvey 1980; Palmer and Rosenquist 1974), at which level cortical input is known to affect velocity selectivity, since cooling of areas 17 and 18 markedly reduces the responses of SC cells to moving stimuli (Ogsawara et al. 1984). Previous evidence, utilising extensive ablation of cortical area 18 and extended recovery periods (Vandenbussche et al. 1991), failed to demonstrate changes in the range of velocity preference of cells in area 17, which nevertheless remained responsive, thereby suggesting that interaction between these two areas in the velocity selectivity domain is minimal. However, the role of layer V has not been specifically examined in this context. Here, we have addressed the possibility of an interaction between these two regions with stimuli of different velocity, attempting to define an influence of area 18 upon area 17 by utilizing focal, reversible blockade of small regions of area 18 while, at the same time, recording from a corresponding region of area 17.

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Materials and methods

Experiments were carried out on 20 adult cats. Animals were anaesthetised (5% down to 2% halothane in 70/30% N_2O/O_2 for induction and surgery, 1 mg/kg per hour pentobarbitone IV (for maintenance), and paralysed (gallamine triethiodide 10 mg/kg per hour). To ensure adequate anaesthesia, EEG, ECG and expired CO_2 were continuously monitored. Temperature was maintained at 37°C. Atropine sulphate and neostigmine were applied to the eyes to dilate the pupils and retract the nictitating membranes. The eyes were protected with plastic contact lenses and, with supplementary lenses, brought to focus on a tangent screen 1.14 m away, viewed through 3-mm artificial pupils.

In each experiment, a pair of individually-controlled recording electrodes were inserted into the visual cortex. A glass-coated platinum-iridium electrode was inserted into layer V of area 17 and a double-barrelled micropipette (containing 4 M NaCl for recording and 2% lignocaine HCl for local inactivation) into a retinotopically matched region of area 18. Electrode placement was determined by a combination of stereotaxic co-ordinates, electrode depth and cell's receptive field properties (Gilbert 1977). Lignocaine was applied by pressure ejection (volume < 5 nL) to create a region of inactivity of 200–300 μm in diameter. Inactivity in this zone lasted for an average of 20–30 min after each application (Malpeli and Schiller 1979). The extent of the region of blockade was determined by moving the pipette up and down after lignocaine was ejected until visual drive was re-encountered, within a radius of 100–150 μm. The radius of horizontal spread of inactivation was also measured at 150 μm. The duration of the effects observed closely paralleled the duration of the blockade in area 18. Cells were classified into simple (S), standard complex (St) and special complex (Sp) classes, according to the criteria of Gilbert (1977).

For each cell pair, optimal orientation and velocity (peak values) were quantitatively determined using computer controlled, randomly interleaved light bar stimuli swept back and forward across the receptive field. Optimal stimuli were used throughout subse-

quent tests; usually a bar of 0.5–1° width and 15° in length. The responses of each single unit in area 18 were taken to represent the responses of the population of cells within that region of blockade, since visual properties and cell types are known to be locally clustered within visual cortex (Hubel and Wiesel 1962; Schwarz and Bolz 1991). During focal blockade of area 18, the responses of the cell isolated in area 17 were re-examined, both at its previously determined optimal velocity and at the optimal velocity for its area 18 counterpart. After each period of blockade, the cells were re-tested to the point of full return to pre-blockade response levels. Recording sites were marked (5 μA, 5–3 s with the metal electrode; 20 μA, 20 s or a 5 min ejection of thionine with the pipette), and after Nissl staining the laminar position and cortical area of each recorded cell was fully reconstructed histologically at the termination of the experiment. The area 17/18 border was identified using the criteria of Garey (1971) and Harvey (1980). All cells had receptive fields within 10° of the area centralis.

Results

Ten cell pairs (20 cells) were selected for this study of velocity selectivity from a total population of 70 cell pairs recorded in a broader study of orientation selectivity. These cell pairs were chosen on the basis of the area 17 cell having a velocity preference at least 22°/s lower than its area 18 partner, thereby marking potential effects for the velocity parameter more clearly visible. When tested with stimuli of the velocity appropriate for area 18, each of the cells in area 17 showed a marked decrease in responsiveness in comparison to stimuli of optimal velocity, in many cases being essentially unresponsive. Of area 17 cells, 40% ($n=10$) showed significant reversible and repeatable alterations in their visual responses during blockade of layer V in area 18. Figure 1 illustrates the effect of focal blockade for one cell pair. The relative positions of the two receptive fields (bottom of Fig. 1) show that the area 18 field completely overlapped the receptive field of the cell in area 17. Control responses were obtained from each cell for stimuli of 'low' velocity (2.2°/s, optimum for the cell in area 17) and 'high' velocity (80°/s, the optimum for the cell in area 18), and are illustrated in Fig. 1A (area 18) and Fig. 1B (area 17). During lignocaine-induced blockade of a region of layer V in area 18, the cell in area 17 (a standard complex cell) was retested (Fig. 1C). It is striking that there was a selective enhancement of the responses of the high-velocity stimulus: responses to the low-velocity stimulus were unaffected. The response returned to control levels (Fig. 1D) after complete recovery of area 18 from the blockade, as indicated by the return of normal visual responses of the cell some 20 min later.

A second example of this effect is shown in Fig. 2. Again, the receptive fields of the two cells overlapped (see bottom of Fig. 2). Once more, when tested with a high-velocity stimulus (in this case 46°/s, the optimum for the cell in area 18, Fig. 2A), the cell in area 17 was almost unresponsive, having a preferred velocity of 5.5°/s (Fig. 2B). During focal blockade of layer V of area 18, the cell in area 17 again showed substantially increased responses to the high-velocity stimulus (Fig. 2C), without a significant change in the responses to the low-velocity stimulus. In this case, however, the velocity-selective increased re-

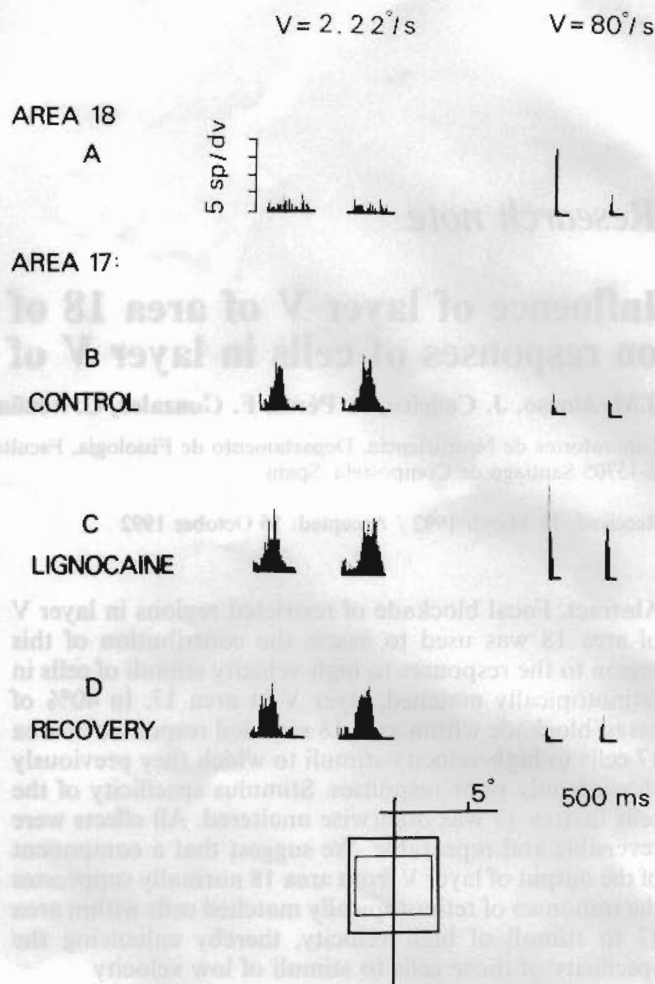

Fig. 1A–D. Visual responses from a pair of simultaneously recorded cortical cells, a standard complex (St) cell in layer V of area 18 (**A**) and a St in layer V of area 17 (**B–D**). All responses are peristimulus time histograms (PSTHs) to stimuli swept forward and backward over the receptive field at the optimum orientation. The *left* PSTHs show the responses to slow velocity (2.2°/s) and the *right* to high velocity stimuli (80°/s). All responses are averaged over 20 sweeps. Control responses of the cells in area 18 (**A**) and 17 (**B**) before lignocaine-induced blockade in area 18, centred on the cell shown in **A**. **C** The responses of the cell in area 17 during blockade. **D** Responses of the cell in area 17 when the area 18 response was fully recovered, approximately 20 min later. Bin width 80 ms. The positions of the two receptive fields are shown beneath the PSHTs (outer frame, area 18; inner frame, area 17). PSTHs record the total spikes

sponsiveness was accompanied by a non-specific increase in background firing (compare Fig. 2B and Fig. 2C). When the blockade of area 18 wore off, some 25 min later, both the visual responses and spontaneous activity of the area 17 cell returned to control levels (Fig. 2D). Two of the four cells whose responses to high-velocity stimuli were enhanced during blockade of area 18 showed significant increased spontaneous activity. The spontaneous activity of all other cells was unaffected.

Table 1 summarizes the data obtained from the population of 10 cell pairs. The right-hand column shows the percentage change in the response to a high-velocity

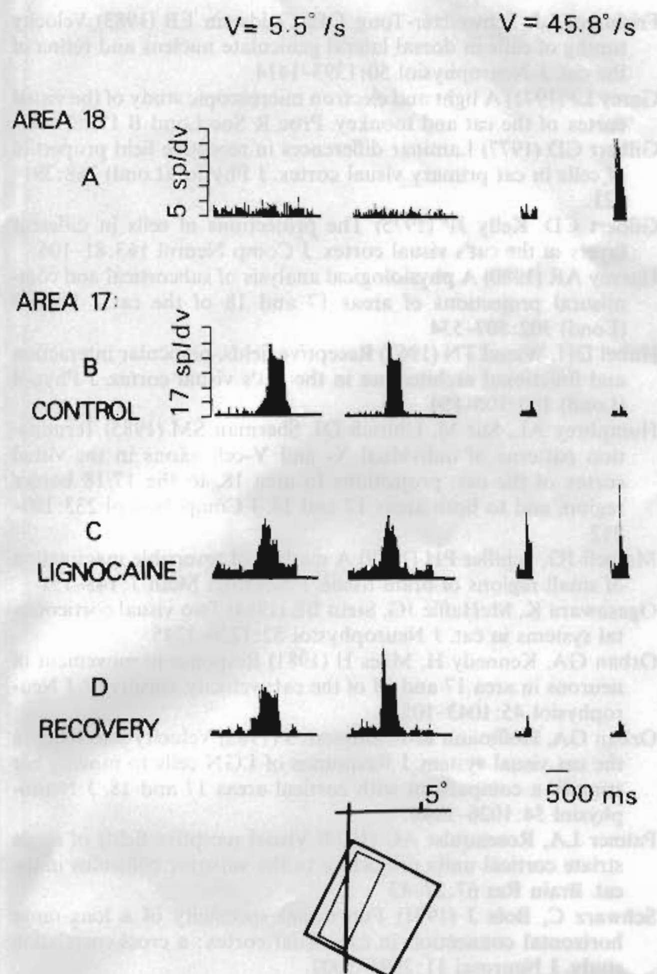

Fig. 2A–D. Visual responses of a second pair of cortical cells, a special complex (*Sp*) cell in area 18 and a standard complex (*St*) cell in area 17. **A**, **B** Control responses of the area 18 and 17 cells, respectively, to stimuli of low (*left*) and high (*right*) velocity. **C** Responses of the area 17 cell during blockade of area 18 (note the increase in background firing). **D** Responses of the cell in area 17 after recovery from the blockade some 25 min later. Receptive fields are shown below the PSTHs. Twenty sweeps, bin width 50 ms. PSTHs record the total spikes

Table 1. Data obtained from 10 pairs of cells in areas 17 and 18

Cell Class area 17/ area18	Optimum velocities area 17/ area 18 (°/s)[a]	Change of area 17 control response vs response during blockade of area 18 (low velocity)	Change of area 17 control response vs response during blockade of area 18 (high velocity)
St/St	12.6/35.3	2%	2%
St/St	4.7/28.3	8%	3%
S/St	2.5/24.6	8%	2%
St/S	23.33/60	3%	7%
St/St	3.5/123.3	16%	14%
S/St	4.1/26.2	2%	3%
St/Sp	5.5/45.8	1%	166%*
S/St	3.3/25.8	2%	150%*
St/St	2.2/80	11%	225%*
Sp/St	18.3/50	9%	241%*

S, simple; Sp, special complex; St, standard complex (Gilbert 1977)
* ($P < 0.0001$, Wilcoxon test (in each case the result of increased responses to high-velocity stimuli)
[a] Values obtained from velocity-tuning curves prior to blockade of are 18

stimulus during blockade, compared to pre-blockade levels. It is clear that the data fall into two distinct groups: the first six cells show little or no change during the blockade but the remaining four cells show significantly increased responses ($P < 0.0001$, Wilcoxon test). The responses to the low-velocity stimulus (optimum, second column from the right) were unchanged in all 10 cases. The two columns on the left indicate the cell types and preferred velocities of each of the pairs of cells. There was no obvious correlation between cell type or preferred velocity between the two groups.

Discussion

The data demonstrate an influence of layer V in area 18 on the velocity tuning of layer V cells in area 17, enhancing selectivity within area 17 by providing the mediating input causing a suppression of response to high-velocity stimuli. This influence, although seen in only 40% of our sample, was surprisingly specific and potent given the relatively large receptive field size (Harvey 1980) and less ordered retinotopic organization of area 18 (Tusa et al. 1979). There are many possible routes via which the effect could be mediated, since, for example, layer V cells are known (at least in striate cortex) to contribute heavily to the receptive fields of the underlying layer VI cells, the source of the cortico-geniculate and cortico-claustral loops (Bolz and Gilbert 1989). Layer V is also known to project heavily to the superficial layers of the cortex, which are also the source of a projection between areas 18 and 17. Furthermore, blockade using the local anaesthetic lignocaine not only suppresses the activity of neurons, but also the activity of fibres of passage, thereby influencing the transfer of information to and from some cells remote from the blocked region. However, the simplest hypothesis to account for such an effect is to suggest that an output from the blocked region of layer V in area 18 directly impinges upon a set of inhibitory interneurons within area 17 which, in turn, provide the high-velocity 'cut-off' inhibition to neighbouring cells within area 17. Such a hypothesis is in keeping with the view that long, cortico-cortical connections are themselves excitatory (Gilbert and Kelly 1975), but suggests that, within area 17, at least a subpopulation of cells capable of responding to stimuli of such high velocity should exist. This view is supported by the work of Orban and co-workers who demonstrated that area 17 contains some cells with broad-band or high-pass, velocity-tuning properties (Orban et al. 1981). Furthermore, Duysens et al. (1985) have suggested that cells responding only to high velocities (VHP) inhibit cells responding only to low velocities

(VLP). Fourty percent of our sample of area 17 cells showed an influence of area 18 of this type. This proportion, however, may not be an accurate reflection of the true weight of the area 18 input, since we deliberately selected those cell pairs showing the greatest difference in preferred velocity. Thus, more subtle shifts in velocity-tuning curves might be revealed in a more detailed study. Nevertheless, a substantial proportion of cells, of all types, were unaffected by the blockade paradigm, suggesting that this mechanism is restricted to some subset of layer V cells not restricted by cell class. The proposed type of inter-areal influence is also supported by anatomical and physiological observations demonstrating both X and Y cell geniculate input to area 17, and exclusively Y cell input to area 18 (Humphrey et al. 1985; Stone et al. 1979). Y cell afferents are known to respond well to stimuli of high velocity and X cells preferentially to lower velocities, when cells of similar retinal eccentricities are compared (Frishman et al. 1983; Orban et al. 1985; Stone et al. 1979). Interestingly, there is now evidence to suggest that corticotectal cells in areas 17 and 18 may themselves receive controlling influences from different populations of geniculate cells having radically different conduction velocities (Weyand et al. 1991). Thus, a 'fast' Y cell influence from layer V of area 18 may serve to suppress inappropriate or mistimed Y cell input to the colliculus from area 17, switching the bias of the corticofugal output according to stimulus velocity.

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