Orientation discrimination in the cat: A distributed function

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ABSTRACT Cats were trained to make fine orientation discriminations with stimuli similar to those used in physiological experiments—narrow, light bars 12° long—before and after various combinations of lesions of areas 17 and 18. Discrimination thresholds were measured at different contrast levels and different bar widths, both pre- and postoperatively, for up to 1.5 years after the lesion. For high contrast stimuli, lesions restricted to area 17 or area 18 had little effect, but those lesions involving area 17 and a substantial part of area 18 raised thresholds. In the latter case there was a relationship between the amount of area 18 spared and the bar width at which discrimination was impaired. At low contrast deficits were seen only for narrow widths. These results lead to the following conclusions. (i) Orientation discrimination is a function distributed within and across areas 17 and 18. (ii) How this function is distributed in this cortex depends on stimulus width. (iii) The X system does not carry the signal necessary for orientation discrimination. (iv) Cells most narrowly tuned for orientation, which reside in the part of area 17 subserving central vision, do not determine the orientation discrimination threshold.

The visual system of primates and higher mammals, in general, is a system of considerable complexity. To understand how processing in this system gives rise to perception, it is necessary to use simplifying experimental strategies (1, 2). One such strategy is to measure thresholds for discriminating very simple stimuli differing only in a single dimension, the encoding of which is known at the single-cell level. Behavioral discrimination between simple stimuli differing in orientation has been shown in humans (3), monkeys (4), and cats (P.D.W., E.V., and G.A.D., unpublished work) to depend only on the stimulus orientation, which is encoded at the early cortical level (5). Threshold differences in orientation in the cat are larger than in the human by a factor of only three (3, 6, 7). Furthermore, orientation discrimination in the cat has many qualitative similarities to human discrimination, such as similar length and contrast dependence (ref. 3; P.D.W., E.V., and G.A.O., unpublished work). Anatomical and physiological information about the visual system of the cat is second to that of no other species (for review, see ref. 8), and the boundaries and connections of the visual areas of the cat are well established for useful lesion procedure. We restricted the study to the effects of area 17 and 18 ablations for two reasons. (i) In the cat, many cortical areas receive direct input from the lateral geniculate nucleus, but only areas 17 and 18 receive direct, parallel input from the paired A laminae of this nucleus (for review, see ref. 9) and are considered to be the major primary visual cortical areas. (ii) Lesions of areas 17, 18, and 19 have a devastating effect on visual behavior (10, 11), making testing at threshold level useless. We have therefore lesioned only areas 17 and 18; although area 19 cells are orientation selective, they are strongly endstopped (12–14) and are, therefore, unlikely to

contribute much to the representation of the long stimuli used in this study.

Cats were trained to discriminate between two light bars differing only in orientation in a discrimination apparatus (7, 15) designed after Berkley (16). In a single trial two light bars, one horizontal (the reference orientation) and the other differing in orientation by Δs degrees, were presented simultaneously, but the cat could see only one at a time because of the splitting screen parallel to the projection. The experimental design required the cat to wait for 350 msec, to enforce attention, and then to press the transparent nosekey corresponding to the side of the horizontal bar. Correct responses were rewarded by small quantities of pureed beef; wrong responses were followed by a longer intertrial interval than after correct responses. Each cat was tested daily, and a daily session consisted of 10 blocks of 30 trials. Only 5 of the 10 blocks used the horizontal orientation as reference; the other 5 blocks used an oblique orientation as reference. In the training sessions only one orientation difference (Δs) was presented per day. This difference was initially 30° and was decreased when the animal reached the average score of 80% correct during one session of 300 trials. Training was stopped when the animal reached the same asymptotic orientation difference three times. In the *testing* sessions that followed and in which the orientation discrimination thresholds were measured, five orientation differences straddling the presumed just noticeable difference [jnd] at 75% correct derived from the training] were presented in random order. The measurement of jnds for both reference orientations took 10 sessions, and therefore each point of the psychometric curve represents 300 trials. Testing was continued until discrimination performance and thresholds were stable. Orientation discrimination was assessed for different stimulus conditions, including two reference orientations and different bar widths and contrasts. Contrast is defined as $\log \Delta I/I$; I is the background illumination that was kept constant. Only changes in orientation discrimination at the horizontal reference orientation as a function of bar width and contrast will be considered here.

Once this assessment was completed, bilateral lesions of area 17 and/or 18 were made by subpial aspiration under sterile conditions and deep Nembutal anesthesia. The testing was resumed at such a time when neurological examination indicated the animal was fully recovered (usually 2 weeks). Those animals in which there was no retention were retrained according to the same schedule as naive cats. Postoperative testing lasted between 6 and 18 months depending on the severity of the deficit.

The extent of the lesions was assessed both histologically and physiologically. In a final experiment single cells, or in a few instances multiple units, were recorded in the remaining cortex adjacent to the lesions by using standard physiological techniques (17). The visual responsiveness of the units was assessed. The location of the receptive field (RF) of the visually driven units in the visual field was determined, and their velocity–response and contrast–response curves were

measured quantitatively whenever possible. Penetrations in which cells were recorded were marked by small electrolytic lesions for histological identification. The animals were sacrificed under Nembutal anesthesia. The borders of the cortical lesions were determined by using sections stained with the Mahon–Heidenhain fiber stain (every tenth section) and with cresyl violet cellular stain (adjacent sections) (18–21). The retinotopic limits of the degeneration in the laminae of lateral geniculate nucleus were determined (22). The extent of the lesion in terms of visual field derived from the histological reconstruction was then matched to the position of the RFs actually recorded in the remaining of area 18 or area 19. This allowed a more precise reconstruction because the maps of Tusa *et al.* (19–21) are standard maps, and certain variability among animals is present. The physiology assessed whether the cortical tissue that was anatomically spared was functional. In general, there was a fringe of tissue ≈ 1 –2 mm wide next to the lesion where the cells, although spiking, could not be visually driven. Hence the anatomical sparing indicated on the visual field maps (Fig. 1) overestimates the actual sparing and does so more in the peripheral part of the visual fields than in their central part because of the cortical magnification.

Nine animals were used in the present study. In six cats area 17 was removed bilaterally with various degrees of involvement of area 18. Histological and physiological controls of the lesions showed that these animals fell naturally into three groups, which we refer to as 17, 17 + $\frac{1}{2}$ 18, and 17 + 18 (Table 1). In two animals (cats 50 and 55) area 17 was removed almost completely with minor involvement of area 18 (up to 2–5° azimuth). In cats 21 and 22 areas 17 and 18 were removed almost completely (Fig. 1). Finally, in cats 27 and 28 the lesion was intermediate, including most of area 17 and a substantial fraction of area 18 (up to 10–15° azimuth). In three cats area 18 and part of area 19 were ablated bilaterally. However, the histological controls showed that in two animals the area 18 lesions were incomplete. Therefore, the

fourth group of animals, labeled 18, included only one animal (cat 30). It is worth pointing out that the physiological recordings showed that in cats with an area 17 lesion cells in the neighboring area 18 were visually responsive, as were those in area 19 after an area 17 + 18 lesion (Fig. 2).

The deficits in orientation discrimination in the animals with restricted lesions, groups 17 and 18, were very small (Table 1). There was no retention deficit in the 17 lesion group and jnds in orientation measured at both higher contrasts ($c = 1.5$ and $c = 2.5$) were normal (Fig. 3). Only when the contrast was reduced to a lower level ($c = 0.3$) did a deficit appear and only for narrow width (0.2°). It is worth mentioning that at a contrast of $c = 0.3$, both area 17 and 18 neurons still respond at 75–80% of their maximum response (B. Gulyas, G.A.O., H. Maes, and J. Duysens, unpublished work). Preoperatively, jnds in orientation did not change with alterations in contrast or width (Fig. 3). The deficits at lower contrast were documented in the two cats 3–6 months after lesioning. One animal (cat 55) was studied for another 6 months, and on retesting, 11 months after the lesion, much of the deficit had disappeared because the jnd at $c = 0.3$ and width = 0.2° had decreased from 10.5 to 6.6°, to an almost preoperative value. Similarly the area 18 lesion in cat 30 had little effect. Despite the fact that the preoperative jnd was very small (2.8°), no deficit was observed after the lesion at the highest contrast and narrow width. Because in all animals studied the discrimination at wider width was always equal to or better than that at narrow width, we can conclude that after type 18 lesions discrimination is normal at all widths at high contrast. Unfortunately we have no information about the jnds of this animal at lower contrast. Because there was no postoperative deficit in the stimulus conditions tested, postoperative testing was stopped after 6 months, without noticeable change in the animal's performance.

Contrary to the restricted lesions, the complete ablation of areas 17 and 18 had a profound effect on orientation discrimination. Postoperatively these animals had lost their discrim-

FIG. 1. Histological and physiological control of the lesions in cat 55, prototype of the area 17 lesion (A–C) and in cat 22, prototype of the area 17 + 18 lesion (D–F). (A and D) Surface view of the cortex indicating extent of the lesion. (B, C, E, and F) Visual fields represented contralaterally in area 17 (B and E) and in area 18 (C and F), indicating the part removed (stipple) and spared anatomically (no stipple). In B, C, E, and F the vertical (VM) and horizontal meridians (HM) are indicated; in these fields the iso-elevation and iso-azimuth lines are separated by 10°. The black dots in C indicate the locations of the RFs of visually driven cells recorded in area 18 near the lesion. All these cells had higher than normal contrast thresholds, except for the unit with the most eccentric RF in the left visual hemifield. Arrow in C indicates the RF of the cell illustrated in Fig. 2.

Table 1. Behavioral observations of the four lesions

Observation	Area 17 (cats 50 and 55)	Area 17 + ½ 18 (cats 27 and 28)	Area 17 + 18 (cats 22 and 21)	Area 18 (cat 30)
Amount of retraining necessary	None	Cat 27: 212 days (0.2°); cat 28: 42 days (1°)	Cat 21: 271 days (0.2°); cat 22: 146 days (1.2°)	None
Discrimination at the highest contrast $c = 2.5$	No deficit (jnd 5 and 5.5°)* at any width	Some deficit (jnd 10 and 16°) at large width, large deficit at narrow width (jnd 28°)	Large deficit (jnd 23.5 and 30°) at all widths	No deficit (jnd 2.8°) at any width
Discrimination at lower contrast $c = 0.3$	Deficit only at narrow width (jnd 16 and 10.5°)	Not tested	Only possible at 3° line width (jnd 27°)	Not tested
Time course of deficit	Improvement over 1 year	No improvement over 1.5 years	No improvement over 1.5 years	Tested for 6 months

*jnds are indicated for different animals in their order in the column heading; when only 1 jnd is given, it corresponds to the first animal in the heading.

inative ability, and considerable retraining was necessary. One animal (cat 21) was retrained with the narrow bar (0.2° width), which required 271 days. The other cat (cat 22) was retrained with a 1.2° wide bar, which took 146 daily sessions. This duration of retraining is of the same order as the training in naive animals, which on average equals 170 days (7). After retraining, jnds in orientation of cat 22 at higher contrasts ($c = 2.5$ and $c = 1.5$) exceeded 20° at all widths tested, 0.2, 1.2, and 3° (Fig. 3). These deficits and a similar one in cat 21 were seen up to 18 months after the lesion. Decreasing the contrast to $c = 0.3$ further increased the threshold of cat 22 but only for bar widths of 0.2 and 1.2° and not for a bar width of 3°.

Although we made no observations at lower contrast ($c = 0.3$) in the group 17 + ½ 18, it is clear that the effects of the intermediate lesion were in between those of the 17 and the

17 + 18 groups (Table 1). Indeed one animal (cat 27) had no retention and was retrained with a narrow bar (0.2° width); retraining took 212 days. The other animal (cat 28) was retrained with a 1° wide bar; retraining took only 42 days. This latter animal, contrary to all others, was only trained and tested on a single reference orientation. Jnds at the highest contrast ($c = 2.5$) were elevated to 10° at widths of 0.6 and 1.2° and to 28° at 0.2° width in cat 27. These results were obtained between 12 and 15 months after the lesion. The data of cat 28, although less complete, confirm that thresholds in this group at wide widths were lower than in the 17 + 18 group. Indeed, postoperatively the jnd for 1° width was 16°.

There is a striking difference between the effects of removal of areas 17 and 18 together and the removal of each of them separately. Indeed, almost complete removal severely

FIG. 2. (A and B) Velocity-response curves. (C and D) Peristimulus time histograms representing the average response of the same units (+, ●) to a bar moving at near optimal velocity, and contrast-response curve (E) of the same units described in A and C. The area 18 cell illustrated in A, C, and E had a RF near the vertical meridian (indicated in Fig. 1), and the stimulus used in A and C was a high-contrast ($c = 1.6$), 0.6° wide bar. In E this stimulus moved at 64°/sec. The responses illustrated in B and D are from a cluster of units recorded in area 19 of cat 22 ≈ 3 mm from the lesion border. The RF of this multiunit recording had an azimuth of 5.6° and an elevation of 0.5°. The stimulus was a medium-contrast ($c = -0.1$), dark square ($1 \times 1^\circ$). Arrows in A and B indicate the speeds used for the histograms in C and D. Horizontal dotted lines in A, B, and E indicate significance level. Horizontal bars below the histograms in C and D indicate motion duration; scales in C and D indicate 0.5 sec and 50 spikes per sec. Most area 17 and 18 cells have contrast thresholds between -1 and 0 and saturate at contrasts between 0.5 and 1.

FIG. 3. Pre- and postoperative performance of cats 55 (A–C) and 22 (D–F). (A and D) Psychometric curves obtained pre- (●) and postoperatively (○) with narrow (0.2°) light bar of the highest contrast ($c = 2.5$). (B and E) jnds in orientation obtained preoperatively, plotted as a function of bar width for three contrast levels: $c = 2.5$, triangles and dotted lines, $c = 1.5$, open squares and dashed lines, $c = 0.3$, solid squares and solid lines. (C and F) Jnds in orientation obtained postoperatively (6 months after the lesion in cat 55 and 13 months after the lesion in cat 22) plotted as a function of line width for different contrast levels indicated as in B and E. Horizontal lines in A and D indicate the 75% correct level used to define the jnd. Vertical lines indicate one SD when this was larger than symbol size. In F the jnds for 0.2 and 1.2° width and lower contrast were higher than we could measure, indicated by arrows.

impaired orientation discrimination at all widths and contrasts tested. Removal of area 17 or 18 produced no deficit at high contrast. The difference between the effect of the removal of the 17 + 18 complex and of its parts indicates that orientation discrimination is a function distributed within and across areas 17 and 18. This finding is probably correlated with the parallel innervation of areas 17 and 18 from the lateral geniculate nucleus. Although at high contrast, the complete removal of the 17 + 18 complex severely impaired discrimination at all widths, and the removal of area 17 with a little involvement of area 18 basically left the discrimination intact, the removal of area 17 plus a substantial part of 18 (up to 10 – 15° azimuth) severely impaired discrimination at narrow widths but less at larger widths. This dependence of the behavior on the amount of area 18 spared suggests that the behavior depended little on the sparing of small parts of area 17 representing peripheral vision because these parts were different in each animal (Fig. 1). The behavioral deficit seen at lower contrast and narrow width after the area 17 lesion might be related to the increase in contrast threshold seen physiologically in units recorded near the lesion in area 18 (Fig. 2). These physiological observations were made in cat 50 immediately after the behavioral observations reported here, but in cat 55 (Fig. 2) they were made more than 2 years after the lesion. As mentioned before, by that time the behavioral deficit had decreased but was still significant at $c = -0.25$. Part of the behavioral improvement for low contrast

stimuli could be due to a recovery of those cells, which had abnormally high contrast thresholds, due to the lesion.

It is well documented that the sensitivity of neurons in area 18 to high spatial frequencies decreases with increasing eccentricity, whereas band-width changes little (23). This fact implies that the high spatial frequency cut-off, which corresponds to the narrowest width to which cortical cells respond (24), also decreases with increasing eccentricity. This suggests that the population of area 18 cells responding as an ensemble to the stimulus orientation will change with bar width. Thus, the orientation of a narrow bar (0.2° width) will be represented by the activity of units with RFs relatively near the representation of central vision at the vertical meridian—e.g., up to 10° azimuth, whereas wider bars (0.6 and 1.2° wide) will be represented in a larger part of area 18—e.g., by units with azimuths up to 20 and 30° , respectively. Hence, removal of area 17 together with that part of area 18 representing up to 15° from the vertical meridian should affect discrimination at 0.2° width more than at 0.6° or 1.2° width. The fact that the increase in contrast threshold was only seen in units in a fringe near the lesion, combined with the effect of width on the representation of orientation, may explain why the effects of contrast decrease were observed behaviorally only at narrow width.

Finally, the complete removal of area 17, except for some parts representing peripheral vision, has little influence on orientation discrimination. Area 17 is the only cortical area

receiving X-type ganglion cell input. Hence, our results imply that X-type system signals are not required for fine orientation discrimination and that likely the Y-type ganglion cell system signals suffice. Others (25) have also stressed the role of the Y pathway in shape discrimination. Finally, qualitative (8) and quantitative (26) studies of orientation tuning show that area 17 cells and, in particular, the simple cells are more narrowly tuned than area 18 cells. Our results, therefore, also imply that the most narrowly tuned cells are not required for discrimination at threshold. This finding is in keeping with recent physiological studies showing that decisions based on single cell firing rates in area 17 are most accurate at the steepest point of the orientation tuning curve and are only loosely related to the tuning width (27, 28). These results show that controlled lesions together with pre- and postoperative physiological studies and psychophysical testing at threshold, dealing with the same stimulus dimensions, are a powerful combination that may lead to the understanding of how perception relates to brain activity and of which parts of the brain are involved.

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