Neural Basis of Orientation Perception in Primate Vision

Abstract. *Orientational differences in human visual acuity can be related parametrically to the distribution of optimal orientations for the receptive fields of neurons in the striate cortex of the rhesus monkey. Both behavioral measures of acuity and the distribution of receptive fields exhibit maximums for stimuli horizontal or vertical relative to the retina; the effect diminishes with distance from the fovea. The anisotropy in the neuronal population and in visual acuity appear to be determined by postnatal visual experience.*

Extensive neurophysiological observations have established that differential responses to oriented stimuli are a distinguishing feature of most receptive fields for neurons in the visual cortex of the cat and monkey (*1–4*). Anatomical homologies in the visual system of man and other primates (*5*) as well as the limited electrophysiological recordings from human visual neurons (*6*) suggest similarities of receptive field organization. The present study indicates how orientation information is extracted and organized at the neuronal level in terms of a horizontal and vertical coordinate system.

In the normal human visual system, horizontal and vertical lines or objects can be detected or discriminated more easily and identified more rapidly and accurately than can ones that are oriented obliquely relative to the retina (*7*). The orientation effect is not due to the optics of the eye, since interference fringes produced on the retina by laser techniques that bypass the eye's dioptrics result in a similar anisotropic discrimination function (*8*). The locus of the effect is more central than the retina, for Maffei and Campbell (*9*) found that the amplitude of the potential evoked by a moving grating, recorded from the occipital scalp, is reduced when the grating is oblique compared with a vertical or a horizontal orientation; the inequality was not found by recording the electroretinogram. The visual evoked potential recorded from the occipital scalp arises from both cortical and subcortical structures, but since the evoked potential is sensitive to the orientation of an adapting grating (*9*) at least part must arise from neurons that are highly sensitive to orientation. Since the primary visual cortex is known to contain neurons sensitive to orientation, it seems likely that neurons in the striate cortex are involved. In the occipital cortex of man, where the region subserving foveal vision is relatively large and close to the surface, the variation with grating orientation of evoked potential amplitude may be produced predominantly by the activation of foveal cortical neurons.

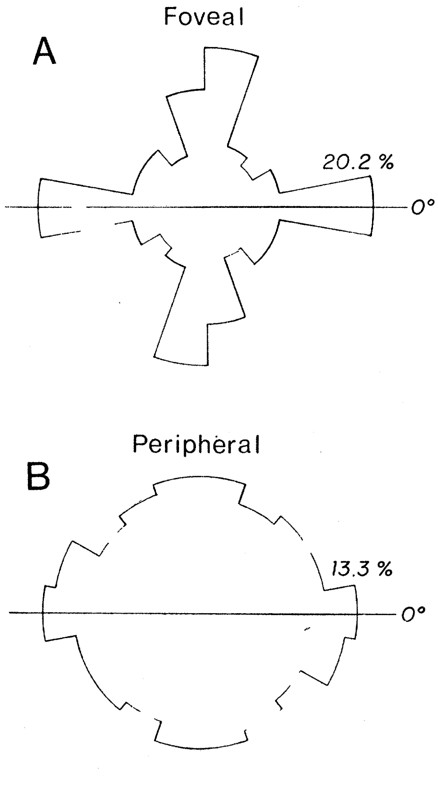

Fig. 1. Circular histograms of optimal orientations for striate cortical neurons. The histograms sum for each 20° interval the number of neurons that discharged maximally for an oriented bar or edge target moved across the unit's receptive field. The horizontal direction relative to the retina is indicated by 0°. (A) Optimal orientations for 79 neurons from the region of striate cortex corresponding to the central 2° in the temporal hemiretina. The two modes are approximately orthogonal, and each accounts for about 20 percent of the entire distribution. The distribution of orientations differs from one in which every orientation is equally represented in a statistically significant manner ($\chi^2 = 16.1$, d.f. = 8, $P < .05$). (B) Optimal orientations for 113 neurons from the region of striate cortex corresponding to an area 6° to 8° from the fovea. The two modes are orthogonal about the horizontal and vertical axes, but each accounts for about 13 percent of the entire distribution. A larger sample size, on the order of 1500 neurons, would be required to demonstrate the statistical validity of the trend. For the present distribution the chi-square statistic is not significant ($\chi^2 = 1.11$, d.f. = 8, $P > .05$).

I have sought to establish the neuronal basis for the orientation effect and hence an early stage of orientation processing in the visual system of primates. The method involved an examination of the receptive field properties of neurons in the striate cortex of the rhesus monkey. Experiments were carried out in five normally reared rhesus monkeys (*Macaca mulatta*), weighing 3 to 5 kg (*10*). Animals were prepared under sodium pentobarbital and later were studied in the awake, unanesthetized state (paralyzed with gallamine triethiodide and artificially respired) in a pain-free condition (*11*). Adequate fluid and electrolyte intake were given intravenously, and temperature and end-tidal carbon dioxide were monitored. Each animal was refracted by retinoscopy, and a tangent stimulus screen at 4 m distance was brought into focus on the animal's retina by appropriate contact lenses. No correction for astigmatism was found to be necessary. Single unit extracellular recording was undertaken with platinum-iridium microelectrodes, glass-insulated except for a 15-μm tip exposure. Unit activity was led through a field effect transistor source follower to a band-pass amplifier and recorded on magnetic tape with a concurrent voice and stimulus channel. Single cortical units were electrically isolated with metal microelectrodes, and the location, extent, and optimal orientation of the unit's minimal discharge field were determined with projected or hand-held stimuli and mapped on the tangent screen. By these procedures, the optimal orientation could be determined to an estimated accuracy of ±7.5° (*12*).

In order to determine the population profile of a receptive field characteristic, such as the axis of optimal orientation sensitivity, it is essential to obtain statistically independent samples and to evaluate the samples by an objective criterion. The correlation of unitary properties by vertical or horizontal proximity that is evident in the columnar or slab organization of striate cortex for orientation (*1, 2*) could introduce substantial bias. To achieve independent sampling, multiple microelectrode penetrations were separated by at least 500 μm by using the radial grid of a Gerbrands micropositioner, and for the purposes of analysis only the first quantitatively studied unit from each penetration was considered. The tabulated orientations displayed in polar form in Fig. 1 are the result of 192 microelectrode penetrations. As an additional control for refractive and constant errors and to determine variations with retinal eccentricity, penetrations were made both in the foveal projection region (central 2°) and in a region subserving an eccentric portion of the retina (6° to 8° from the fovea) (*13*).

The variations in acuity and in evoked potential amplitude with stimulus orientation could result from changes in the response specificity for neurons with receptive fields of different orientation preferences, or, on the other hand, the effects could be produced by a relative predominance of receptive fields with particular optimal orientations. The orientation tunings of receptive fields with preferred orientations of 0° and 90° were not observed to be noticeably sharper than those with oblique orientation preferences. However, for the neurons sampled from the foveal projection region of striate cortex (Fig. 1A), an anisotropy is evident in the population profile of orientation sensitivity. Many units responded maximally to stimuli oriented vertically or horizontally but relatively few to stimuli at oblique orientations. The departure from a distribution in which every orientation is equally represented was evaluated by the chi-square test and is statistically significant at the 5 percent level ($\chi^2 = 16.1$, d.f. $= 8$, $P < .05$). The correspondence, then, between the population profile of orientation sensitivity for foveal cortical neurons in the rhesus monkey and the acuity performance function for foveal stimulation in human observers suggests the hypothesis that the orientation effect in man is the result of a predominance of cortical neurons with receptive fields optimally sensitive to horizontal and vertical stimuli.

A question of considerable interest is whether the anisotropic distribution of optimal orientations in cortical neurons is genetically programmed or results from visual experience. Several studies have demonstrated that early postnatal exposure of kittens to vertical or horizontal striped patterns produces a relative increase in the number of visual cortical neurons with receptive fields having maximal sensitivity at the conditioning orientation (*14*). Even less severe visual deprivation such as experimentally induced astigmatism in kittens still produces a similarly skewed distribution of cortical receptive fields (*15*). Such kittens as adults exhibit reduced acuity for stimuli oriented orthogonally to that present in their early visual environment (*16*). Early astigmatism in man produces a comparable acuity deficit that is not subject to correction by refraction (*17*) and can be demonstrated in cortical evoked potentials (*18*). A neural modification analogous to that found in the cortices of visually deprived kittens may occur in the visual system of primates. The early and continuing predominance of vertical and horizontal contours in the normal visual environment may be sufficient to produce the observed neural anisotropy of orientation detectors.

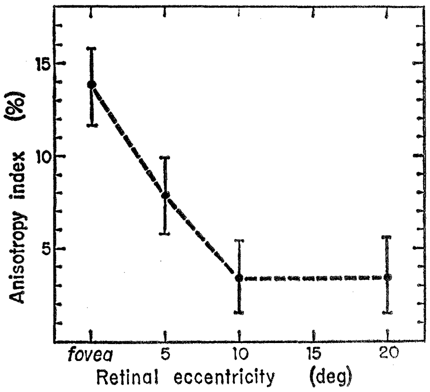

Fig. 2. Retinal gradient of orientational differences in visual acuity. An index of acuity anisotropy (equal to one minus the ratio of the acuity for horizontal and vertical gratings divided by that for the 45° and 135° obliques) is plotted against the location of the test target relative to the fovea. The measure of acuity was the spatial cut-off frequency for a square-wave grating. Each data point is the mean value of the index for three emmetropic observers; the bars indicate the standard error of the mean.

A comparison of the two distributions of receptive field orientations shown in Fig. 1 with those found in other experiments suggests the existence of a gradient of orientation anisotropy from central to peripheral vision. During development the central projection of the visual field may be more susceptible to environmental influences. Typically, distributions of cortical neurons composed in the main of units with receptive fields in the noncentral visual field exhibit little or no orientation anisotropy (*2, 4*). For both cat and monkey data Hubel and Wiesel (*2*) concluded that no marked orientational differences were present in their samples, which were mainly from noncentral projection regions. For simple cells in the cat, some evidence suggests a greater prevalence of units maximally sensitive to vertical and horizontal contours in the center of the visual field (*3*). In congruence with these neurophysiological observations, a gradient of orientation anisotropy can be demonstrated psychophysically in human observers, as shown in Fig. 2.

Three emmetropic observers viewed monocularly square-wave grating targets briefly exposed for periods of 0.5 second duration at various retinal eccentricities relative to a steady foveal fixation point at the same optical distance. Visibility thresholds were determined by varying the viewing distance of the target in a threshold tracking procedure (*17*). In addition to the decrease of visual acuity with degree of retinal eccentricity, the acuities for vertical and horizontal stimuli relative to those for oblique stimuli were found to decrease. The parametric correlation between human visual acuity for oriented gratings and the distributions of optimal orientations for monkey cortical neurons strongly suggests that the performance profile of striate neuron reflects the processing of behaviorally relevant orientation information in primates.

The global mechanisms of visual form perception are at present a matter for conjecture. One attractive hypothesis is that the visual system employs a process of Fourier-like analysis and synthesis. Several lines of evidence support the view that the visual system analyzes spatial patterns in terms of their horizontal and vertical Fourier components of spatial frequency (*19*). Neurophysiological studies of unit responses in the striate cortex of the cat indicate that spatial frequency is encoded at the neuronal level (*20*). Ginsburg (*21*) has pointed out that the orientation anisotropy evident in visual performance could be viewed as a two-dimensional filter in the Fourier transform space of the visual patterns. The distribution of receptive field orientation sensitivity found here for striate neurons may well serve as the mechanism of that filter.

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References and Notes

1. D. H. Hubel and T. N. Wiesel, *J. Physiol. (Lond.)* **160**, 106 (1962); *J. Neurophysiol.* **28**, 229 (1965).
2. ———, *J. Physiol. (Lond.)* **195**, 215 (1968).
3. J. D. Pettigrew, T. Nikara, P. O. Bishop, *Exp. Brain Res.* **6**, 373 (1968).
4. F. W. Campbell, B. G. Cleland, G. F. Cooper, C. Enroth-Cugell, *J. Physiol. (Lond.)* **198**, 237 (1968).
5. For review of evidence, see G. F. Poggio, in *Handbook of Medical Physiology*, V. B. Mountcastle, Ed. (Mosby, St. Louis, ed. 12, 1968), vol. 2, p. 1592.
6. E. Marg, J. E. Adams, B. Rutkin, *Experientia* **24**, 348 (1968).
7. M. M. Taylor, *J. Opt. Soc. Am.* **53**, 763 (1963); S. Appelle, *Psychol. Bull.* **78**, 266 (1972); F. Attneave and R. K. Olson, *J. Exp. Psychol.* **74**, 149 (1967).
8. F. W. Campbell, J. J. Kulikowski, J. Levinson, *J. Physiol. (Lond.)* **187**, 427 (1966); D. E. Mitchell, R. D. Freeman, G. Westheimer, *J. Opt. Soc. Am.* **57**, 246 (1967).
9. L. Maffei and F. W. Campbell, *Science* **167**, 386 (1970); F. W. Campbell and L. Maffei, *J. Physiol. (Lond.)* **207**, 635 (1970).
10. The monkeys were feral, mature, female animals imported directly from India.
11. V. B. Mountcastle, W. H. Talbot, H. Sakata, J. Hyvärinen, *J. Neurophysiol.* **32**, 452 (1969); G. F. Poggio, F. H. Baker, Y. Lamarre, E. Riva Sanseverino, *ibid.*, p. 892.
12. The optimal orientation was determined by auditory monitoring of the unit's discharge and varying the orientation of the target stimulus, typically a black or white bar of high contrast relative to the uniformly illuminated tangent screen, until the maximum discharge was produced. Independent repeated determinations were used to assess the accuracy of the procedure
13. The locations were estimated by comparing the penetration sites with the known retinotopic projection onto the rhesus striate cortex [G. F. Poggio, *Invest. Ophthalmol.* **11**, 368 (1972); B. M. Dow and P. Gouras, *J. Neurophysiol.* **36**, 79 (1973); G. F. Poggio, F. H. Baker, P. Grigg, R. J. W. Mansfield, A. Sillito, in preparation].
14. C. Blakemore and G. F. Cooper, *Nature (Lond.)* **228**, 447 (1970); H. V. B. Hirsch and D. N. Spinelli, *Science* **168**, 869 (1970).
15. R. D. Freeman and J. D. Pettigrew, *Nature (Lond.)* **246**, 359 (1973).
16. D. W. Muir and D. E. Mitchell, *Science* **180**, 420 (1973).
17. D. E. Mitchell, R. D. Freeman, M. Millodot, G. Haegerstrom, *Vision Res.* **13**, 535 (1973).
18. R. D. Freeman and L. N. Thibos, *Science* **180**, 876 (1973).
19. F. W. Campbell and J. G. Robson, *J. Physiol. (Lond.)* **197**, 551 (1968); C. Blakemore and F. W. Campbell, *ibid.* **203**, 237 (1969); C. Blakemore, J. Nachmias, P. Sutton, *ibid.* **210**, 727 (1970); N. Graham and J. Nachmias, *Vision Res.* **11**, 251 (1971); M. B. Sachs, J. Nachmias, J. G. Robson, *J. Opt. Soc. Am.* **61**, 1176 (1971); L. Maffei and A. Fiorentini *Nature (Lond.)* **240**, 479 (1972).

→ L. Maffei and A. Fiorentini, *Vision Res.* **13**, 1255 (1973); D. A. Pollen, J. R. Lee, J. H. Taylor, *Science* **173**, 74 (1971).

21. A. P. Ginsburg, *Proc. IEEE 1973 NAECON*, Dayton, Ohio (1973), p. 309.
22. I gratefully acknowledge many helpful discussions with G. F. Poggio and V. B. Mountcastle, and thank D. B. Yntema, D. G. Green, and M. Alpern for valuable suggestions and comments. This research was supported by the William F. Milton Fund (Harvard University) and PHS grant NS-02974.

29 July 1974; revised 9 September 1974 ■

Retrograde Amnesia and the "Reminder Effect"

Retrograde amnesia produced by such treatments as electroconvulsive shock is often temporary. Retention of an avoidance response, for example, can be restored by subjecting amnesic animals to a noncontingent footshock (NCFS) (*1*). These data have led some (*2*) to theorize that the amnesic treatments interfere with retrieval of stored information and that NCFS reactivates the retrieval process. Recently, Gold *et al.* (*3*) advanced an alternative explanation. They argued that NCFS provides a learning experience and predicted that for the learning experience to restore retention it must add to weak retention. Consistent with this prediction they reported that NCFS restores retention in animals that are either partially amnesic or weakly trained (animals showing weak retention), but that NCFS does not restore retention in animals that are either completely amnesic or untrained. While the results of this study are clear, the interpretation is not, and the logic connecting the effects of NCFS on partially amnesic and weakly trained animals is tenuous.

In the first phase of their experiment, Gold *et al.* determined the extent to which NCFS restores retention in partially and completely amnesic animals. Animals were trained, subjected to an amnesic agent, and then tested. During the test, the behavior was variable, some animals showing partial and others complete amnesia. After the test, the animals were given NCFS, and the next day they were given a second retention test. Behavior on the second test also varied; the NCFS restored retention in those animals that had shown partial amnesia on the first test, but did not restore retention in those animals that had shown complete amnesia. On this basis, Gold *et al.* concluded that NCFS restores retention because it summates with weak retention shown by the partially amnesic animals. This may be true, but, in my opinion, indeterminable from their experiment.

Gold *et al.* did not include a basic control group. It is clear that partially amnesic animals show recovery of retention after NCFS and that completely amnesic animals do not. It is not clear, however, that the NCFS is necessary to initiate recovery. It is possible that partially amnesic animals would recover retention even if they were not given NCFS. What is needed is a control group that receives no NCFS: the control animals in this case must of course be partially amnesic since completely amnesic animals show no recovery even with NCFS. Gold *et al.* presented data for a control group that received no NCFS and, indeed, showed no signs of recovery. The problem is that the animals in this control group, judging from the median and interquartile range of their behavior during the first test, appear completely amnesic.

Even if the experiment had contained the appropriate controls, the data would still be difficult to interpret because of the ex post facto experimental design. It seems clear from their report (*3*) that it was not the experimenter who, by manipulating a variable, determined which animals would be in the partially and which in the completely amnesic groups. Rather, the experimenter gave the animals in each group the same treatment and then constructed the two groups on the basis of individual differences in reaction to the amnesic agent. However, individual differences in this case can be due to any number of variables, including strength of initial learning, susceptibility to the disruptive effects of the amnesic agent, or motivation to drink. Gold *et al.* failed to establish whether these uncontrolled variables, rather than strength of retention, were instrumental in determining the ultimate reaction to NCFS.

In a second phase of the experiment, to cross-validate the relation between strength of retention and effectiveness of NCFS, Gold *et al.* included two additional groups distinguished by different training conditions rather than by individual differences in reaction to the amnesic agent. One group received weak training, a second group received no training, and neither group received an amnesic agent. The next day the groups behaved as expected: the weakly trained group showed weak retention, the other group of course showed no retention. After the test, both groups were given NCFS and the next day were given a second test; the NCFS improved retention in the weakly trained but not in the untrained group. In contrast to the amnesic data, the training data are interpretable since the groups, including appropriate con-