Encoding of Spatial Location by Posterior Parietal Neurons

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Abstract. *The cortex of the inferior parietal lobule in primates is important for spatial perception and spatially oriented behavior. Recordings of single neurons in this area in behaving monkeys showed that the visual sensitivity of the retinotopic receptive fields changes systematically with the angle of gaze. The activity of many of the neurons can be largely described by the product of a gain factor that is a function of the eye position and the response profile of the visual receptive field. This operation produces an eye position–dependent tuning for locations in head-centered coordinate space.*

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The brain receives visual information in a coordinate frame derived from the focusing of images on the retinas. The retinal sensory sheets are mapped to several locations in the central nervous system through a series of parallel and approximately point-to-point projections. In fact, at least nine visual cortical fields have been identified in the monkey which contain orderly representations of the contralateral visual field, each formed by the systematic mapping of the corresponding nasal and temporal halves of the retinas (*1*). While such retinotopic representations of visual space are undoubtedly advantageous for many visual functions, they are not ideal for many aspects of visual-motor coordination. Since the positions of the eyes and head vary from moment to moment, the retinotopic locations of targets likewise change, although their spatial locations with respect to the head or body may not change. Since motor actions are directed to locations in space with great ease and accuracy, and since many of these actions are made too quickly for visual feedback, the brain must transform the retinal image into head- and body-centered coordinate frames that are accessible to the motor system (*2*). In addition to the motor system, the visual perceptual system probably also transforms the retinal representation into a spatial one. Such a transformation is thought to be required in order to perceive the world as stable, since we make, on the average, three eye movements a second with subsequently as many changes in the retinal image (*3*).

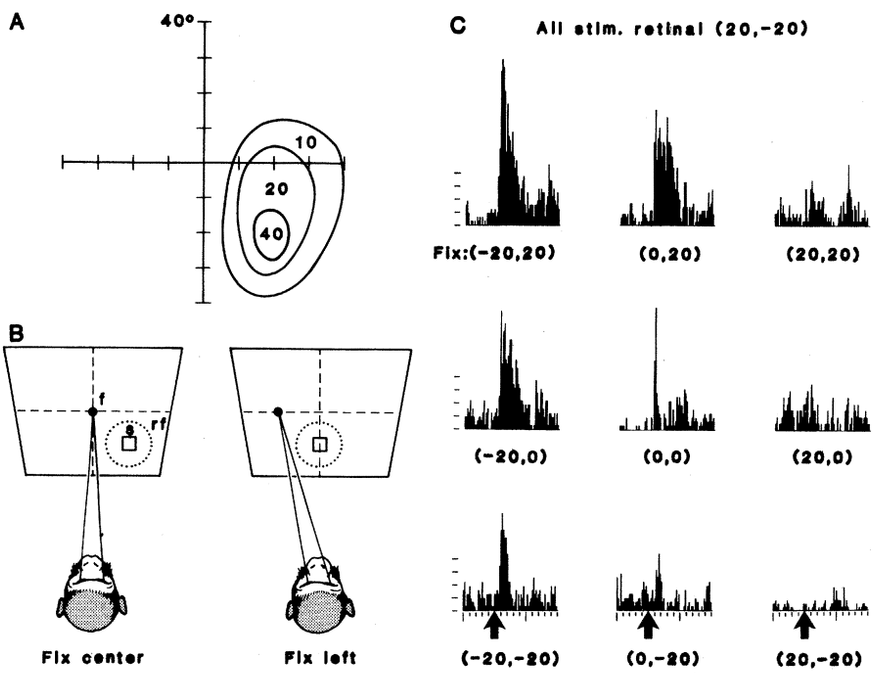

Fig. 1. (A) Receptive field of a neuron plotted in coordinates of visual angle determined with the animal always fixating straight ahead (screen coordinates 0,0). The contours represent the mean increased response rates in spikes per second. (B) Method of determining spatial gain fields of area 7a neurons. The animal fixates point f at different locations on the screen with his head fixed. The stimulus, s, is always presented in the center of the receptive field, rf. (C) Spatial gain field of the cell in (A). The poststimulus histograms are positioned to correspond to the locations of the fixations on the screen at which the responses were recorded for retinotopically identical stimuli presented in the center of the receptive field (histogram ordinate, 25 spikes per division, and abscissa, 100 msec per division; arrows indicate onset of stimulus flash).

The posterior parietal cortex is the most likely location of cells coding the location of visual stimuli in space, since clinical and experimental studies implicate it as essential for accurate spatial orientation and perception (*4*). In a previous study we reasoned that if this area represents space in at least head-centered coordinates, then the visual responsiveness of its neurons to retinotopically identical stimuli should change systematically as a function of the position of the eyes in the orbits; we found such an eye-position effect to exist (*5*). In the present study we determined that, for many of the neurons, this angle-of-gaze effect can be largely described by gain factors, which are a function of eye position, multiplied by the response profile of the retinal receptive fields. This operation results in the eye position–dependent tuning of these cells for locations in head-centered space.

Recordings from single neurons were made from awake rhesus monkeys performing visual fixation tasks (*5*). All aspects of the experiment were under computer control. The animal was required to fixate a small point of light back-projected at different locations on a large (100° by 125°) tangent screen. Since the animal's head was fixed to the primate chair by means of an acrylic skull cap, the eyes moved to different positions in the orbit with changes in the spatial location of the fixation target on the screen. The activity of single neurons was recorded with glass-coated platinum-iridium microelectrodes that were advanced through the dura into the cortex with a micropositioner. Eye position was monitored by the scleral search coil technique (*6*). The receptive fields of the neurons were mapped with a second visual stimulus that was flashed on the screen for 500 msec during the fixation trials. The intensity of the test stimulus was controlled by the computer such that the stimulus was of the same luminosity, measured at the animal's eyes, regardless of the position of the stimulus on the screen.

First we mapped the receptive fields of the light-sensitive neurons of area 7a with the animal looking straight ahead. A typical receptive field is shown in Fig. 1A. The receptive fields were generally large and homogeneously excitatory, with the maximum response zone situat-

ed in the center. These receptive fields were found in both the ipsilateral and contralateral visual fields. Next we had the animal look at nine different locations on the screen and always presented the stimulus at the same retinotopic location in the center of these receptive fields (Fig. 1B). A change in the angle of gaze of a few degrees almost always changed the magnitude of the response of the neuron (on average, 3 percent per degree normalized to the mean response). An example of the angle-of-gaze effect is shown in Fig. 1C; this neuron gave the best visual responses when the animal fixated up and to the left. We refer to such plots of the responses to retinotopically identical stimuli delivered at different eye positions as spatial gain fields.

For 87 of 102 neurons for which we determined spatial gain fields we further analyzed the mean evoked responses with a first-order linear model with horizontal and vertical eye positions as independent variables (*7*). Thirty-nine percent of the cells had planar gain fields (model $P < 0.05$; lack-of-fit $P > 0.05$). The spatial gain field in Fig. 1C is of the planar type with a plane tilted up and to the left giving the best fit to the data. For another 38 percent of the cells there was a statistically significant planar component and a significant lack of fit, usually indicating that there was a bump in the overall planar fit. For the remaining 23 percent of the gain fields, there was not a significant planar component ($P > 0.05$) and there was a significant lack of fit ($P < 0.05$); the profiles of these fields generally showed a peak at the center of the gain field.

After determining the spatial gain field of a cell, we remapped the entire receptive field for 34 neurons at the most and least preferred angles of gaze. In Fig. 2 the responses of four neurons are plotted with respect to retinotopic positions along a horizontal or vertical axis passing through the center of the receptive field. Generally the response magnitude at each point in the receptive field changed by a proportional amount with each gaze angle, indicating that the interaction of the visual and eye position signals is multiplicative.

To control for the possibility that the visual surround (which changes with each fixation location) could be affecting the response to the stimulus, we determined the spatial gain fields of 48 cells in both light and total darkness and found no difference between the two conditions. For an additional 22 neurons we deviated the eyes by having the animal look through prisms rather than by moving the fixation point. The advantage of this control is that it can be done in a lighted room, since the retinotopic position of the visual background does not change with changes in eye position. Deviating the eyes with prisms resulted in the same gain fields as were measured by moving the fixation target.

The existence of these gain fields indicates that the cells will respond best for visual stimuli located in restricted regions of head-centered coordinate space. A demonstration of this spatial tuning is

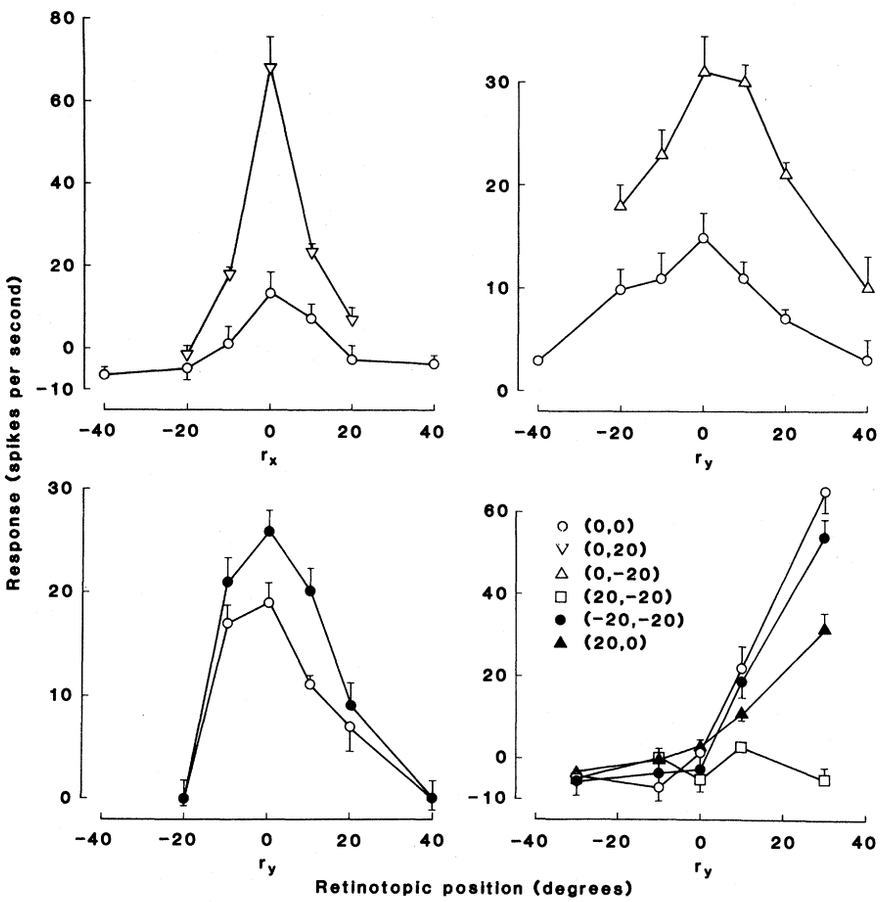

Fig. 2. Mean response rates for different eye positions plotted in retinal coordinates along horizontal (r_x) or vertical (r_y) axes passing through the centers of the receptive fields of four neurons; each graph shows data for one neuron. Each point represents the mean response (± standard error) to eight repetitions of the stimulus presented at the same retinal location. A randomized block design was used to present stimuli to different retinal locations in the receptive field of each cell. The reported response at each retinal location is equal to the activity during the presentation of the stimulus minus the background activity determined before the stimulus presentation.

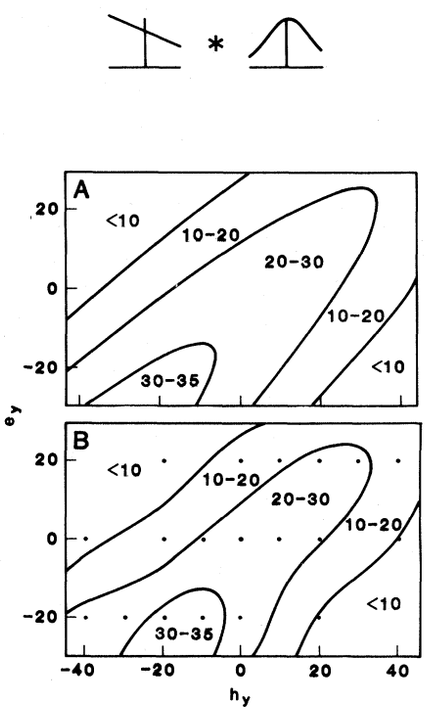

Fig. 3. (A) Computer simulation of the response (in spikes per second) of an area 7a neuron predicted by multiplying the vertical axis of a planar gain field by the vertical axis of a Gaussian receptive field. The results are represented on the contour plot with the stimulus head-centered coordinates (h_y) plotted along the abscissa and eye position (e_y) along the ordinate. (B) Contour plot of actual recording data for a cell with the same gain field and receptive field characteristics as the model neuron plotted in (A). Each data point represents the mean evoked response to eight repetitions of the stimulus; the average standard error for these data points was two spikes per second.

illustrated in Fig. 3. In Fig. 3A is a contour plot of the predicted response of an idealized neuron for visual stimuli occurring at different locations in head-centered coordinate space and viewed at different angles of gaze. For simplicity only one dimension, the vertical meridian, is considered. This contour plot was generated by multiplying a planar gain field, which is a sloping line in one dimension, by a receptive field approximated by a Gaussian and whose center lies on the vertical meridian. The resulting plot predicts that, within the range of eye positions considered, the cell will respond best when the stimulus occurs approximately 20° down in head-centered space when the animal is also looking 20° down. The response profile is also elongated along a diagonal that corresponds to the head-centered coordinate location of the center of the retinal receptive field at the different angles of gaze. A cell with no angle-of-gaze effect would have a gain of one at all eye positions; as a result it would have the same maximum response at every point along this diagonal and would not exhibit tuning for head-centered coordinate locations. Figure 3B shows a contour plot of neural data for an actual neuron with gain and receptive field properties similar to those of the cell modeled in Fig. 3A. The similarity of the two plots, both in this case and for seven other neurons for which there were sufficient data to enable this type of analysis, indicates that a simple multiplication of the retinal receptive field by the gain field is sufficient to approximate the spatial tuning behavior of these neurons.

These area 7a cells do not encode the spatial location of stimuli independent of eye position; however, computer simulations we have made show that such an eye position–independent response can be achieved by combining the activity of several neurons that have the same maximum head-centered location responses, but for different optimum angles of gaze. Such a convergence may take place in the projection of area 7a onto another brain structure. However, since this information already exists in the response of subpopulations of neurons in area 7a, it is likely that spatial locations are encoded in the activity of groups of these neurons and may not require an additional step of convergence.

Finally, there is the question of how space is represented topographically across area 7a. At present we do not have sufficient data to address this issue; one attractive possibility is that the space-tuned peaks of activity are ordered to form a systematic map of head-centered coordinate space across the tangential dimension of cortex.

References and Notes

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7. In collecting the data for each spatial gain field, eight replications of retinotopically identical stimuli were delivered at each of the nine eye positions by following a randomized block design. The visually elicited response rates were adjusted for background activity, which often varied with eye position. Conventional linear regression techniques were applied to partition the response variability into components dependent on x and y eye positions and residual "pure error" and "lack-of-fit" components for statistical testing [D. G. Kleinbaum and L. L. Kupper, *Applied Regression Analysis and Other Multivariable Methods* (Duxbury, North Scituate, Mass., 1978); J. Netter and W. Wasserman, *Applied Linear Regression Analysis* (Irwin, Homewood, Ill., 1983).

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Molecular Defects in a Human Immunoglobulin κ Chain Deficiency

Abstract. *The molecular basis of a human immunoglobulin deficiency characterized by the complete absence of κ chains has been investigated by nucleotide sequence analyses of a patient's κ constant region (C_κ) genes. Both of his C_κ genes had a single point mutation, resulting in the loss of the invariant tryptophan from one allele and of an invariant cysteine from the other allele. These results indicate that neither of the patient's C_κ alleles encoded a κ chain that could form a stable intradomain disulfide bond, although peculiarities in the expression of κ chains in the patient's family suggest that other factors may be involved.*

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One case of complete absence of immunoglobulin (Ig) κ chains (*1*) and a few cases of reduced expression of κ chains have been reported in humans (*2*). We have studied the molecular basis of the absence of κ chains in the completely κ-deficient individual. As B lymphocytes bearing κ chains could not be detected in this individual's blood or bone marrow, the defect did not seem to be due to an inability of κ-expressing lymphocytes to mature to a secreting stage (*1*). We hypothesized that the cause of the deficiency could be deletions of or mutations in genes coding for Ig κ chains; however, defects could also be in other genes that affect the expression of κ chains, since serum from the κ-deficient individual's sister contained a very low amount of κ chains, and sera from his parents and other sibling had approximately normal amounts of κ chains (*1*). We have investigated the cause of this case of κ chain deficiency by nucleotide sequence analysis of both of the κ constant region (C_κ) genes from the patient. Each C_κ gene had a different single point mutation, which resulted in the loss of the invariant tryptophan at amino acid position 148 from one allele and one of the two invariant cysteines (at position 194) from the other allele, so that neither of his C_κ domains should be able to form stable intradomain disulfide bonds.

To search for any gross rearrangements or large deletions in the patient's Ig κ genes, genomic DNA from blood leukocytes (more than 95 percent of which do not produce Ig) from the patient and his parents was analyzed by DNA blotting experiments (*3*). No differences were detected among restriction fragments containing Ig C_κ or J_κ (J, joining) genes in the patient, his parents, and in normal human placenta, with three different restriction enzymes (Bam HI, Eco RI, and Bgl II). To determine