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The Effects of Parvocellular Lateral Geniculate Lesions on the Acuity and Contrast Sensitivity of Macaque Monkeys

William H. Merigan,[1,3] Laurence M. Katz,[4] and John H. R. Maunsell[2,3]

Departments of [1]Ophthalmology and [2]Physiology and [3]Center for Visual Science, University of Rochester Medical Center, Rochester, New York 14642 and [4]Department of Ophthalmology, Henry Ford Hospital, Detroit, Michigan 48202

The effects of ablating the visual pathway that passes through the parvocellular (dorsal) LGN were tested in 2 macaque monkeys by measuring acuity and both luminance and chromatic contrast sensitivity. Thresholds were tested monocularly before and after ibotenic acid was used to lesion parvocellular layers 4 and 6 of the contralateral geniculate. The injections were centered at the representation of 6° in the temporal field on the horizontal meridian, and vision was tested with localized stimuli at this location. In addition, in one of the monkeys, a lesion was made in magnocellular layer 1 of the opposite geniculate, and the same thresholds were tested. Physiological and anatomical reconstructions demonstrated complete destruction of the target layers in 1 parvocellular lesion and in the magnocellular lesion, and sparing of the nontarget layers in the tested region.

Parvocellular lesions caused a 3–4-fold reduction in visual acuity within the affected part of the visual field, while the magnocellular lesion did not affect acuity. Both luminance and chromatic contrast sensitivity, tested with stationary gratings of 2 c/degree, were severely reduced by parvocellular lesions, but not affected by the magnocellular lesion. However, when luminance contrast sensitivity was tested with 1 c/degree gratings, counterphase modulated at 10 Hz, it was reduced by both parvocellular and magnocellular lesions. This study demonstrates that the parvocellular pathway dominates chromatic vision, acuity, and contrast detection at low temporal and high spatial frequencies, while the magnocellular pathway may mediate contrast detection at higher temporal and lower spatial frequencies.

Parallel retinocortical pathways in the primate share many properties but show some striking differences, and it is of interest to determine which of their characteristics are most closely related to their contribution to vision. For example, most retinal ganglion cells contribute to the pathway through the parvocellular LGN (P pathway); about 80% according to Perry et al., 1984, and therefore this pathway might be expected to contribute importantly to spatial resolution (Merigan and Katz, 1990). On the other hand, the spatial resolution of individual cells in this pathway and those in the pathway through magnocellular geniculate (M pathway) are virtually identical, suggesting a more balanced role of the 2 pathways in acuity (Lee et al., 1987). Neurons in the parvocellular cell pathway show physiological color opponency (Kaplan and Shapley, 1982; Derrington et al., 1984), suggesting a dominant role in color vision. However, numerous reports have described a nonsigned second harmonic response of neurons in the M pathway to chromatic stimuli (Lee et al., 1989), suggesting a possible role in color vision. Finally, it has been suggested that, because neurons in the P pathway carry both luminance and chromatic information, this pathway should contribute as much to luminance contrast sensitivity as it does to chromatic contrast sensitivity (Derrington and Lennie, 1984). On the other hand, most investigators have found that the contrast sensitivity of individual neurons of the M pathway is higher by about a factor of 8 than that of neurons of the P pathway (at some spatiotemporal frequencies; Kaplan and Shapley, 1982), suggesting a greater role for the M pathway in luminance contrast sensitivity.

The present study is part of an effort (Merigan and Maunsell, 1990) to use psychophysical measures after lesions of these pathways to determine their roles in visual perception. This approach will help clarify which aspects of the anatomy and physiology of visual pathways provide the best insights into their functional contribution. In this study, we used ibotenic acid lesions of the P and M pathways in the LGN to examine the role of these pathways in visual acuity and contrast sensitivity.

Materials and Methods

Subjects

The subjects were 2 adult, female monkeys (*Macaca nemestrina*) of approximately 5 kg body weight. They had free access to monkey chow, supplemented regularly with fresh fruit. Their water was withheld for approximately 20 hr before threshold testing 5 d each week. All thresholds were measured monocularly during controlled fixation, and neither monkey had more than 0.5D of refractive error in either eye.

Implantation of scleral search coil and headmount

After initial training on the psychophysical tasks (see below), a scleral search coil was implanted in the right eye of each monkey, under isoflurane anesthesia, so that eye position could be monitored, and a stainless-steel sleeve was attached to the skull so that the monkey's head could be immobilized (Judge et al., 1980). The monkeys' thresholds were then measured on all of the tasks described below, after which lesions were placed in the lateral geniculate nucleus.

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Correspondence should be addressed to William H. Merigan, Box 314, University of Rochester Medical Center, Rochester, NY 14642.

Apparatus and procedure

Testing procedures. All thresholds were tested monocularly using a horizontal–vertical grating orientation discrimination. While the monkey fixated within ±0.3° of a red fixation spot, the test stimulus was presented to the right or left of fixation (i.e., along the horizontal meridian of the retina), at a single location in each daily session. Stimulus contrast onset followed a raised cosine of 0.5 Hz, and the stimulus remained on until either a choice response had been made or the monkey's fixation moved outside of the window. The interval between trials was 4 sec, correct choices (see below) were rewarded with fruit juice, incorrect choices were followed by a 6-sec beeping tone, and fixation breaks or premature responses were followed by a 3-sec beeping tone. The spatial frequency (during acuity testing) or contrast (contrast sensitivity testing) of the stimulus was varied according to a staircase, becoming easier by 1 step (0.33 octave spatial frequency or 2-dB contrast) after each error and harder with probability 0.33 after each correct choice. Daily sessions consisted of 200 trials, and thresholds were taken at 75% correct, responding either by linear interpolation or by probit fits to the daily psychometric functions (Finney, 1971).

Acuity. The monkey faced a high-resolution oscilloscope display (Tektronix 606 with P-31 phosphor), on which were presented vertical or horizontal sinusoidal gratings of variable spatial frequency and of 0.55 Michelson contrast $[(L_{max} - L_{min})/(L_{max} + L_{min})$, where L_{max} is the highest and L_{min} the lowest luminance in the grating]. (This display was used to test acuity because the color monitor described below could not be moved a sufficient distance to present high-spatial-frequency stimuli.) The mean luminance of the display was maintained at 16 cd/m^2, and it was viewed through a circular aperture (dimensions below) in an unilluminated surround. Each trial began with presentation of the dim red fixation spot. When the monkey fixated, a 4500-Hz tone was presented, and stimulus onset was initiated. The tone remained on for 1.8 sec, and then went off, signaling the opportunity to respond. Responses to the left button on the response panel were correct when the stimulus was a vertical grating, and those to the right button were correct when a horizontal grating was present.

Acuity was tested monocularly along the horizontal meridian of the right eye in both monkeys. Measurements were made at eccentricities of 0°, 3°, 6°, 12°, 20°, and in some cases 25° or 30° (visual subtense of the fixation spot from the nearest edge of the stimulus). In addition, a single location was tested at 6° eccentricity in the temporal field of the left eye of monkey 675 to examine the effect of a magnocellular lesion. The test target was 185 cm from the observer for eccentricities of 0°, 3°, and 6° and 93 cm for eccentricities beyond 6°. The diameter of the stimulus aperture was 0.5° for 0° eccentricity, 1° for 3° and 6° eccentricity, 1.5° for 12° eccentricity, and 3° for 20°, 25°, and 30° eccentricities. Thus, the stimulus was always approximately 20 cycles of a just-resolvable grating.

Contrast sensitivity. The seated monkey faced a 19″ color monitor (Conrac 7211) at a distance of 211 cm. The fixation spot was projected onto the face of the monitor, and contrast thresholds were measured as above by having the monkey report the orientation of the stimuli, which were small patches of vertical or horizontal grating displayed on the monitor. The grating targets were Gabor functions (cosinusoidal gratings multiplied by horizontal and vertical gaussian weighting functions) generated on an Adage 3006 raster display unit and presented at a frame rate of 60 Hz (noninterlaced). The horizontal and vertical gaussian weighting functions had equal space constants (s = standard deviation of gaussian) but these were larger for monkey 676 (s = 2.04°) than for monkey 675 (s = 1.35°). Target size was reduced for monkey 675 to reduce the possibility that targets would extend beyond the lesion borders. Thus, the grating was above 37% of peak contrast (full width at ±s) over a central region of 4.08° and 2.7°, respectively, for the 2 monkeys.

The monitor displayed an equal-energy white background of 63 cd/m^2, and Gabor functions were superimposed on this background for 3 measures of contrast sensitivity: (1) Chromatic contrast sensitivity was tested with patches of isoluminant gratings of 2 c/degree spatial frequency and no temporal modulation. These stimuli were modulated in a constant-blue direction in color space (approximately red–green) around the white point of the monitor background, and cone contrast sensitivity was calculated from the sum of the contrast delivered to middle- and long-wavelength cones (Merigan, 1989). (2) Static luminance contrast sensitivity was measured with patches of luminance grating of 2 c/degree spatial frequency and no temporal modulation. Contrast sensitivity for this and the following condition was calculated from the Michelson contrast of the stimuli. (3) Temporally modulated luminance contrast sensitivity was measured with patches of grating of 1 c/degree modulated in counterphase at 10 Hz. We wanted to use a slightly lower spatial frequency than 2 c/degree for this rapidly modulated stimulus, but were unable to go to very low spatial frequencies because we were limited by the size of the Gabor patch.

Placement of lesions. After stability was achieved on all psychophysical measurements, small ibotenic acid lesions were placed in parvocellular layers 6 and 4 of the left lateral geniculate nucleus of both monkeys and magnocellular layer 1 of the right geniculate of monkey 676. For each lesion, the monkey was anesthetized with Sufenta (sufentanil citrate, 2 μg/kg/hr) and paralyzed with Norcuron (vecuronium bromide, 100 μg/kg/hr). Recordings with platinum/iridium microelectrodes were used to locate the target visual field representation in the relevant layers. The electrode was then withdrawn and replaced by a glass probe that could be used for both recording and injection. Physiological recording was again used to place the tip of the pipette first in layer 6 and then in layer 4 (contralaterally driven parvocellular layers) of the left geniculate of each monkey, or layer 1 (contralaterally driven magnocellular layer) of the right geniculate of monkey 676, approximately 6° temporal of the fovea along the horizontal meridian. Two microliters of ibotenic acid (5 μg/μl) were then pressure injected at each location.

Reconstruction of lesions. At the conclusion of behavioral testing, the lesions placed in monkey 675 were reconstructed with anatomical and physiological techniques. Immediately prior to death, a physiological map was made of layer 1, the magnocellular layer, and layers 4 and 6, the parvocellular layers, in both lateral geniculate nuclei. Electrolytic lesions (10 μA × 10 sec) were made to facilitate reconstructing the penetrations. After physiological recording, the monkey was killed and perfused with a saline rinse followed by 4% paraformaldehyde in phosphate-buffered saline. The brain was removed and blocked, and 40-μm sections were cut on a freezing microtome. One section in 4 was reacted for cytochrome oxidase activity (Wong-Riley, 1984), and another was stained with cresyl violet. The lesions were then reconstructed from these anatomical sections using the location of electrode penetrations to map them precisely onto the visual field. Monkey 676 died unexpectedly after the completion of behavioral testing, so no physiological mapping was possible in this animal. Its lesion was reconstructed anatomically as described above.

Results

Figure 1 illustrates the appearance of the parvocellular lesion from monkey 675 in frontal sections through the lateral geniculate. Cytochrome oxidase staining clearly revealed the boundaries of neuronal loss, and such a section is shown in Figure 1*A*. This section represents the location where the extent of the lesion was greatest, involving all 6 of the geniculate layers. However, the damage to magnocellular layer 1, shown in this section, corresponded to a visual field locus well outside of the region tested psychophysically (Fig. 2), and layer 1 was intact in the region tested. Figure 1*B* shows, in a Nissl-stained section, that the lesion boundaries were very sharply demarcated, and that there was complete loss of neurons within the lesion and no apparent neuronal loss beyond the lesion. Figure 1*C* is a reconstruction of the lesion on drawings of frontal sections through the geniculate, progressing from posterior (left) to anterior (right). The anatomical and physiological reconstruction was used to create visual field maps of the lesion for each layer. Figure 2 shows visual field representations ablated by the parvocellular geniculate lesions in both monkeys and the magnocellular lesion in monkey 675. Each set of axes in each panel shows the central portion of the temporal field representation in a particular geniculate layer. Small squares represent some of the receptive field centers that were plotted during the final mapping session. The borders of the lesion were located in a series of histological sections and, in monkey 675, related to visual field locus by interpolating between nearby recording sites. Hatched regions

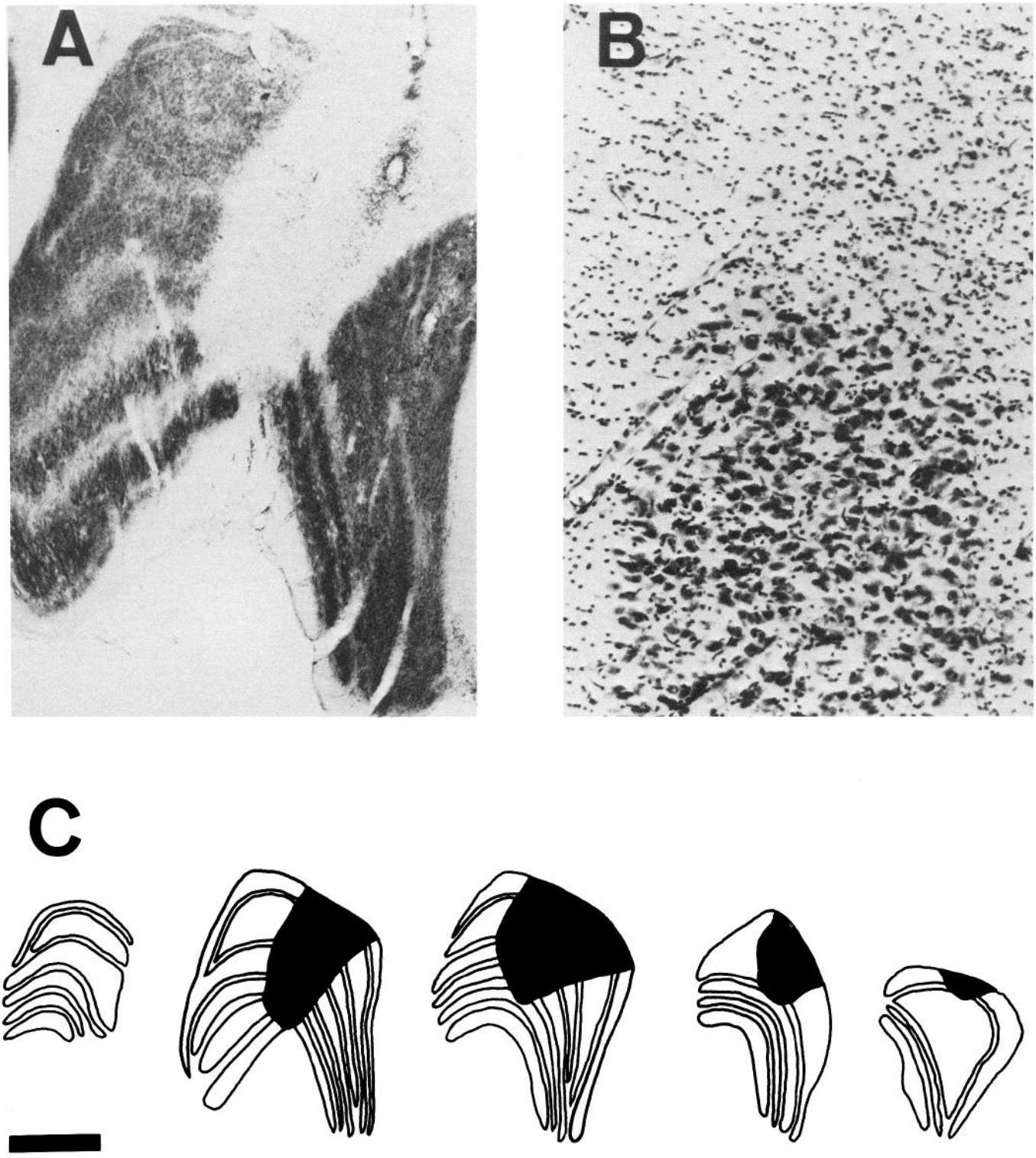

Figure 1. Histological reconstruction of the left LGNP lesion from case 675. The photomicrograph in *A* is of a cytochrome oxidase-stained section. The lesion is visible as a lightly stained wedge. This section was taken from the region where the lesion was most extensive, involving all 6 layers. Although a small portion of the magnocellular layers was ablated in this region, the magnocellular representation of the tested part of the visual field was intact (see Results and Fig. 2). *B* is a higher-magnification photomicrograph of an adjacent section that was stained with cresyl violet. This section shows the remarkably sharp border of the lesion, which was a consistent feature of ibotenic acid lesions. *C* is a series of tracings from sections through the entire LGN, showing the full extent of the lesion. Scale bar, 2 mm (allowing for 15% shrinkage during histological processing).

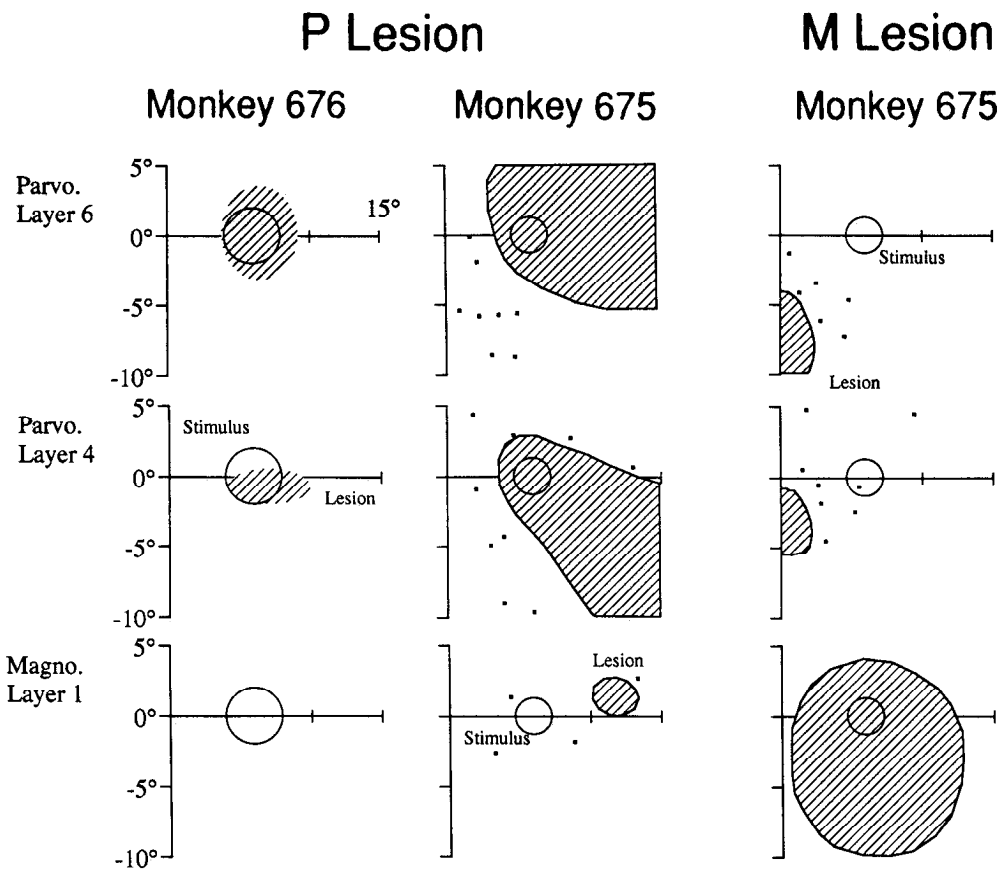

Figure 2. Visual field maps showing the portion of the visual field representation lesioned in different geniculate layers in the 2 monkeys. The *horizontal* and *vertical lines* represent the horizontal and vertical meridia of the visual field, and the fixation point is at their intersection. Axes for parvocellular lesions represent the temporal field of the right eye (left LGN). Those for the magnocellular lesion represent the temporal field of the left eye (right LGN) and have been mirror reversed to facilitate comparison. A *circle* on each plot shows the $\pm s$ for the Gabor test patch used to measure contrast sensitivity. *Small squares* on the plots for monkey 675 show the receptive fields for some of the physiological recording sites where normal activity was found after the conclusion of behavioral testing, and the *hatched areas with borders* show a reconstruction of the lesion from anatomical sections and physiological mapping. Physiological mapping was not done in monkey 676, and thus the lesion reconstructions (*hatching with no border*) are estimates made from anatomical sections and receptive fields recorded at the time the lesion was made.

with a border indicate representations in monkey 675 completely destroyed by the lesions, and the unbordered hatched regions for monkey 676 show the estimated location on the lesion. The affected representations for animal 676 are based on the size of the lesion and approximate magnification factors calculated from receptive fields from 6 electrode penetrations made in the vicinity of the lesion at the time the ibotenic acid was injected. The circle on each horizontal meridian shows the location and size of the test stimulus for contrast sensitivity. The ibotenic acid invariably spread into noninjected layers and was particularly prone to spread up the electrode track. However, unintended damage to other layers was restricted to untested parts of the visual field. Both parvocellular and magnocellular lesions in monkey 675 completely destroyed the stimulus representation in target layers while completely sparing the nontarget layer(s). On the other hand, the parvocellular layer lesion in monkey 676 destroyed the target region in layer 6 and spared layer 1 completely, but we estimate that only a portion of the target region in layer 4 was destroyed.

The effect of parvocellular and magnocellular lesions on visual acuity are shown in Figure 3. The parvocellular lesion in monkey 676 caused a decrease in visual acuity that was maximally about 3-fold and extended only over a very narrow part of the visual field (over 3°). The larger parvocellular lesion in monkey 675 produced a greater acuity drop (about 4.5-fold) and extended over more of the visual field (over 7°). Acuity thresholds away from the lesions were not decreased in the postlesion testing. The magnocellular lesion in monkey 675 had no effect on visual acuity, which was measured only in the center of the ablated representation. These measures constitute the only threshold mapping of the extent of the parvocellular lesions in this study, and they appear to correlate rather well with the extent of lesion in the less damaged parvocellular layer, layer 4. In both monkeys, the drop in acuity, like the lesion itself, began beyond 3° eccentricity. In monkey 676, both extended to less than 11° eccentricity, whereas in monkey 675 both extended beyond 12° eccentricity.

Chromatic contrast sensitivity before and after geniculate lesions is shown in Figure 4. The parvocellular lesion in monkey 676 caused about a 4-fold loss of chromatic sensitivity, whereas that in monkey 675 made it impossible to measure any threshold. Because the highest chromatic contrast we could present was about 0.26, we can conclude that residual contrast sensitivity (reciprocal of contrast threshold) could not have been higher than about 4. No effect was seen of the magnocellular lesion in monkey 675 on chromatic sensitivity.

Effects on luminance contrast sensitivity measured with a 2-c/degree unmodulated stimulus are shown in Figure 5. The results were virtually identical to those obtained with the chromatic stimulus that are displayed in Figure 4. A 4-fold loss of sensitivity was found in monkey 676 after the parvocellular lesion, and no threshold could be measured in monkey 675. Because stimulus contrast extended to 0.8, sensitivity must be lower than about 1.25. On the other hand, there was no effect of the magnocellular lesion in monkey 675.

Finally, results obtained with a 1-c/degree 10-Hz counterphase-modulated stimulus are shown in Figure 6. Sensitivity for this stimulus was decreased about 2–3-fold by parvocellular lesions in both monkeys and was also reduced almost the same amount by the magnocellular lesion in monkey 675.

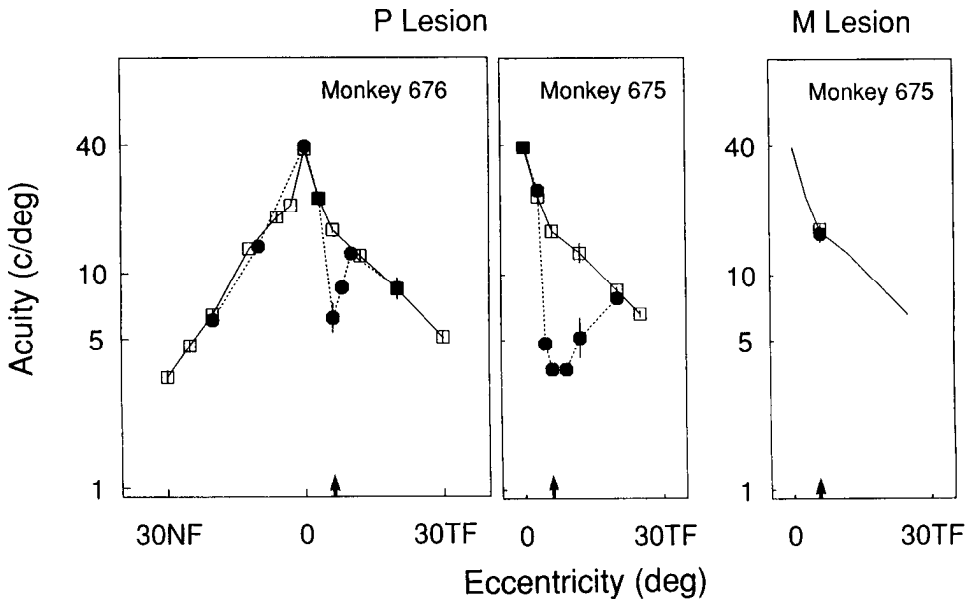

Figure 3. Monocular visual acuity of the 2 monkeys as a function of eccentricity along the horizontal meridian. *Open squares* and *solid lines* show acuity before lesions were made, and *solid circles* and *broken lines,* acuity after the placement of lesions. The site of the ibotenic acid injection along the horizontal meridian is indicated by *arrows.* Parvocellular lesions were made in the left geniculate, and the data are from the contralateral (right) eye; the magnocellular lesion was made in the right geniculate, and the data are for the eye contralateral to the lesion (left). As in Figure 2, the plot for the magnocellular lesion has been mirror reversed to facilitate comparisons. The *solid line* in the magnocellular lesion plot shows the acuity function for the temporal field of the opposite eye; only the single point shown was measured in this eye. *NF,* nasal field; *TF,* temporal field. Error bars = ±SEM.

Discussion

This study demonstrates clearly that the parvocellular retinogeniculate pathway (P pathway) mediates the perception of high spatial frequencies, chromatic stimuli, and luminance-defined contours modulated at low temporal frequencies. The detection of each of these classes of stimuli was devastated by lesions of the pathway through parvocellular geniculate, but not affected by the lesion of the pathway through magnocellular geniculate. A role for the M pathway was shown for the detection of stimuli of slightly lower spatial but substantially higher temporal frequency. This outcome is consistent with previous work (Merigan and Eskin, 1986; Merigan and Maunsell, 1990) that suggests that the pathway through parvocellular geniculate dominates most of visual detection, but that the M pathway may be important for detection at lower spatial and higher temporal frequencies.

Spatial resolution

The magnocellular lesion in monkey 675 produced no drop in visual acuity, indicating that visual acuity depends only on the P pathway and not on an interaction of the P and M pathways. This is not a surprising conclusion, given that the falloff in visual acuity with distance from the fovea can be accounted for rather precisely simply by the average Nyquist frequency derived from the spatial sampling density of "on" or "off" parvocellular ganglion cells assuming triangular packing (Merigan and Katz, 1990). This finding confirms the point, by now well established (Thibos et al., 1987; Merigan and Katz, 1990), that eccentric spatial resolution in the primate is limited by parvocellular ganglion cell sampling density and not by cone density. Magnocellular ganglion cells are less numerous than parvocellular cells by about a factor of 8 at all eccentricities (Perry et al., 1984), suggesting that resolution by the M pathway should be lower by approx-

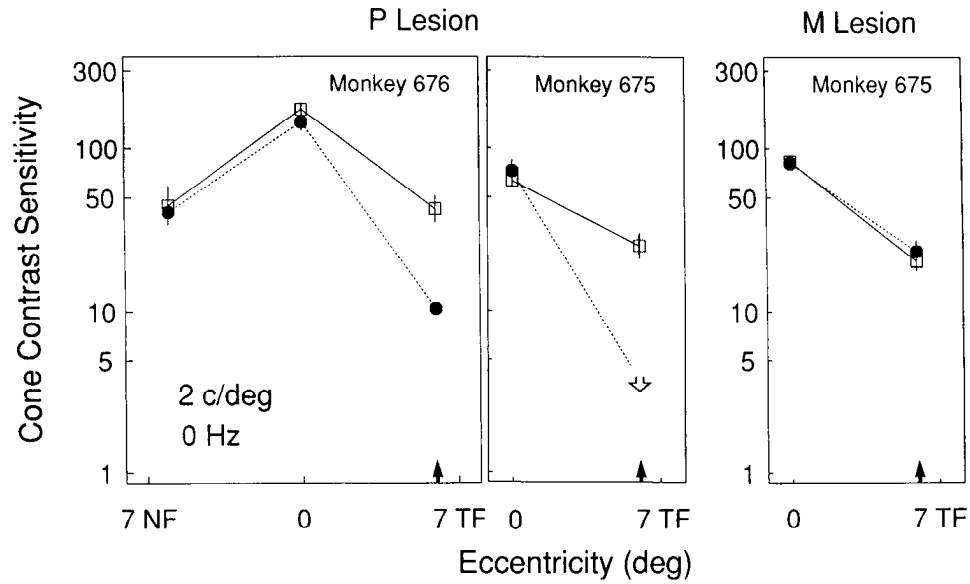

Figure 4. Chromatic contrast sensitivity before and after geniculate lesions. Thresholds were measured with patches of isoluminant gratings of 2 c/degree spatial frequency modulated in color space along a constant-blue (approximately red–green) axis. The axis provides no stimulation to short-wavelength-sensitive cones and thus selectively stimulates the "red–green" color-opponent mechanism. Cone contrast sensitivity is calculated from the sum of the contrast delivered to middle- and long-wavelength-sensitive cones, using the Smith and Pokorny cone fundamentals (Smith and Pokorny, 1975). Results before (*open squares*) and after (*solid circles*) lesion placement are shown. Abbreviations are the same as in Figure 3.

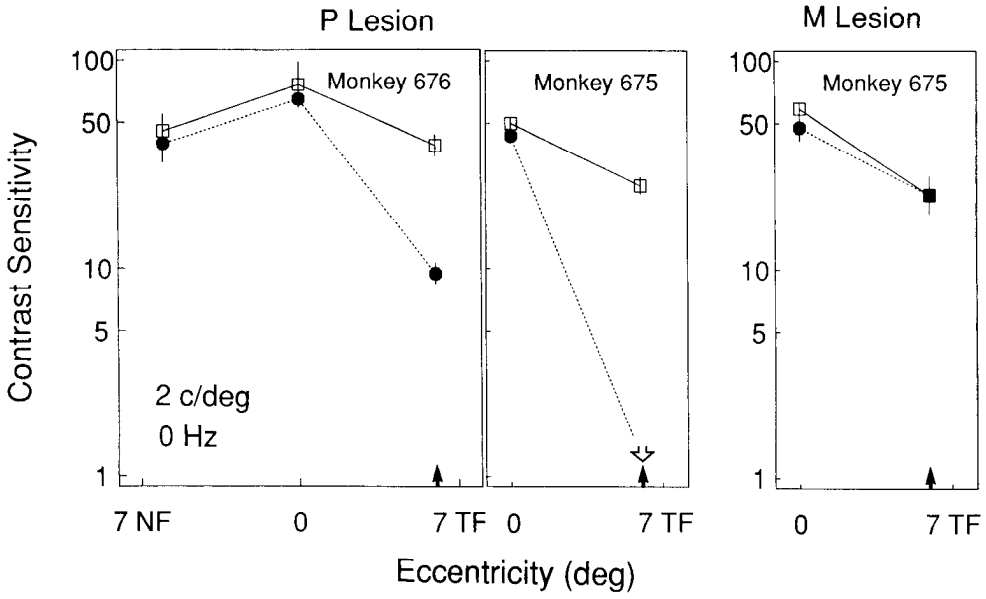

Figure 5. Luminance contrast sensitivity for a patch of 2-c/degree stationary grating before and after geniculate lesions. Data before (*open squares*) and after (*solid circles*) lesion placement are shown.

imately 2.8-fold. The maximal resolution of the M pathway could be estimated from the residual resolution after a P pathway lesion. This approach does not seem appropriate for monkey 676, because the layer 4 lesion was quite narrow, and there is little suggestion in Figure 2 of a flat minimum of acuity in the parvocellular lesion. However, the data for monkey 675 show a broader minimum within the lesion, indicating about a 4-fold loss from damage to the P pathway.

An important implication of this result, which is consistent with previous behavioral studies (Merigan and Eskin, 1986; Schiller et al., 1990), is that visual perception in the entire high-spatial-frequency portion of the spatiotemporal stimulus space (at least down to a spatial frequency 4-fold lower than acuity) is mediated by the P pathway. One curious aspect of this result is that, while the P pathway is usually thought of as the color-opponent pathway (see below), the poor spatial resolution of chromatic mechanisms (Ingling and Martinez-Uriegas, 1983; Mullen, 1985) dictates that only luminance variation can be seen over most of the high-spatial-frequency region dominated by the parvocellular system. Selective stimulation of the P pathway can thus be achieved by luminance stimuli that contain only high spatial frequencies.

We have ascribed the major difference of P and M pathways in spatial resolution entirely to anatomical differences (spatial sampling density) in the retina. Curiously, physiological studies in the retina and geniculate do not indicate that receptive field differences are an important contributor to this difference. Crook et al. (1988) measured spatial resolution and receptive field center size of parvocellular and magnocellular retinal ganglion cells, while Derrington and Lennie (1984) calculated receptive field center size for geniculate neurons in the macaque. Both studies found a very small difference in center size or resolution

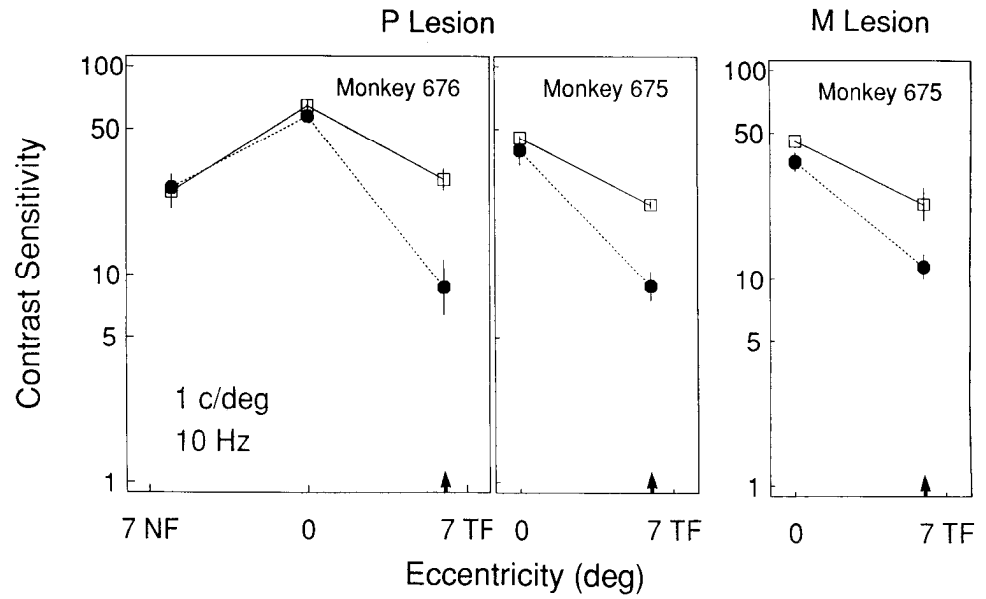

Figure 6. Luminance contrast sensitivity for patches of 1-c/degree, 10-Hz counterphase-modulated gratings before and after lesions of the geniculate. Data before (*open squares*) and after (*solid circles*) lesion placement are shown. Abbreviations are the same as in Figure 3.

of individual parvocellular and magnocellular geniculate cells, indicating that the poor resolution of the magnocellular is attributable only to low sampling density, and not to averaging across the receptive field center. One suggestion of this analysis is that undersampling by the M pathway may have produced aliased fringes (Williams, 1985) when the monkeys viewed gratings in the representation of a parvocellular lesion.

Chromatic contrast sensitivity

The results shown in Figure 4 indicate that chromatic contrast sensitivity is too low to measure after damage to the P pathway (monkey 675), but that damage to the M pathway causes no change in chromatic sensitivity. The residual chromatic sensitivity of monkey 676 is likely due to the small size of the lesion in layer 4 and the resulting extension of the test stimulus into nonlesioned portions of the P pathway. The conclusion that color vision depends only on the P pathway has also emerged from a previous study of the chromatic contrast sensitivity of macaques with severe loss of retinal parvocellular ganglion cells (Merigan, 1989). That study, like the present one, found severe loss of sensitivity for red–green (or more precisely, constant-blue) color modulation due to damage to the P pathway. The earlier study also examined sensitivity along 3 other directions in color space and found equally profound losses. Together, these results, and the recent report by Schiller et al. (1990), suggest that damage to the P pathway produces a profound reduction of all aspects of color sensitivity.

These results are entirely consistent with physiological studies of the P and M visual pathways. Overt color opponency is found only in the response of neurons of the P pathway (DeMonasterio and Gouras, 1975; Derrington et al., 1984; Lee et al., 1987), which provides the major input to the cortical "form and color" pathway (Maunsell and Newsome, 1987). Neurons in the parvocellular layers of the geniculate respond optimally to 1 of the 2 "cardinal" directions of color space (Derrington et al., 1984) that have been identified in psychophysical studies in human subjects (Krauskopf et al., 1982). However, there has been intense interest in recent years in the possibility that the M pathway could play an important role in color perception. This has been driven in part by the observation (Schiller and Colby, 1983; Lee et al., 1989) that M pathway neurons respond with a frequency-doubled response to the alternation of isoluminant chromatic stimuli. As has been pointed out recently (Merigan, 1989), such a physiological response represents a loss of phase, and therefore color identity information, and could not underlie chromatic vision. However, the frequency-doubled response does preserve temporal and border information and could be used to derive temporal frequency or velocity from moving isoluminant stimuli, or to segment regions of stimuli on the basis of their color.

The present results and those of Merigan (1989) also suggest a tight correspondence between the luminance and chromatic sensitivity mediated by the P pathway (see below). It is especially noteworthy that chromatic sensitivity is best at low temporal frequencies (Horst and Bouman, 1969), which precisely mirrors the low temporal frequency contribution of the P pathway to luminance sensitivity, but contrasts sharply with the poor sensitivity of the M pathway to low temporal frequencies (Merigan and Eskin, 1986; present study). When damage to the P pathway is incomplete (monkey 676 in the present study; monkeys 117 and 561 in Merigan, 1989), decreases in luminance and chromatic contrast sensitivity were virtually identical, suggesting a common neuronal substrate.

Luminance contrast sensitivity

The measures of luminance contrast sensitivity illustrated in Figure 5 were obtained with a stimulus of moderate spatial frequency not much different from that used to collect the data shown in Figure 6, but of a very low temporal frequency. The low temporal frequency content was achieved by presenting a stationary grating with a very slow onset and reducing temporal variation due to eye movements by requiring controlled fixation. Fixation was routinely near the center of the $\pm 0.3°$ fixation window, with the result that little temporal modulation was introduced by eye movements. The results (for monkey 675) indicate that the visibility of such relatively stationary stimuli is determined solely by the P pathway. As was pointed out above, the residual sensitivity of monkey 676 is likely due to overlap of the test stimuli onto nonlesioned areas of the P pathway. This finding suggests that the P pathway dominates vision not only at high spatial frequencies (see above), but also at low temporal frequencies. A similar conclusion emerged from a previous study of macaques with damage to the P pathway (Merigan and Eskin, 1986), though this previous study differed in showing a less pronounced loss of sensitivity at low temporal frequencies. It is likely that the greater residual sensitivity in the previous study was due to the absence of controlled fixation testing, which may have resulted in greater temporal modulation of the stationary stimuli by eye movements.

When the test grating was modulated at 10 Hz (Fig. 6), the results of geniculate lesions were very different. Lesions of the P pathway produced a moderate (2–3-fold) loss in sensitivity in both monkeys, but a similar effect was produced by an M pathway lesion in monkey 675. This latter effect was the only decrement in visual performance seen after an M pathway lesion in this study, and it suggests some role of the M pathway in the detection of higher temporal frequencies. This is a less dramatic finding than that in the earlier study of Merigan and Eskin (1986), which indicated that contrast detection at 10 Hz was determined largely by the M pathway for stimuli of lower spatial frequency. The less pronounced contrast sensitivity loss in the present study may be due to the use of fixation control and the testing of sensitivity at an eccentricity of 6°. At this eccentricity (under the stimulus conditions of this experiment), visual acuity is slightly over 2-fold lower than at the fovea, suggesting that the 1-c/degree test stimulus used in here would be comparable to a stimulus of over 2 c/degree in the earlier study. Indeed, the interpolated data of Merigan and Eskin (1986) show that at this higher spatial frequency contrast sensitivity would be decreased only slightly by damage to the P pathway.

More recent studies in our laboratory on the effects of M pathway lesions tested at 6° eccentricity (W. H. Merigan and J. H. R. Maunsell, unpublished observations) indicate that the effects of M pathway lesions depend critically on the temporal frequency of the test stimulus. Such lesions do not reduce contrast sensitivity for 1-Hz modulated stimuli, but they clearly reduce it for 20-Hz modulated stimuli. These preliminary results support the findings in this study in showing that the P pathway mediates detection at low temporal and high spatial frequencies, but that the M pathway plays an increasingly important role in detection as temporal frequency is raised and spatial frequency lowered.

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