

THIS study shows the existence in humans of independent neural processing streams in early visual cortex, which had previously been demonstrated in macaque monkeys. This evidence was obtained by controlled fixation testing of a subject who had suffered a small stroke in the right fusiform gyrus. The patient showed a severe disruption of color perception, shape discrimination and contrast sensitivity for stationary gratings in the upper left quadrant of his visual field. However, motion perception and contrast sensitivity for drifting gratings were relatively preserved. These results support the view that there are independent visual processing streams early in human visual cortex, and that these streams may subserve such functions as motion and color/form perception.

**Key words:** Color; Contrast sensitivity; Motion perception; Shape discrimination

## Parallel processing streams in human visual cortex

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### Introduction

Modularity (independent processing of different visual attributes by different cortical streams) is evident at the highest levels of human visual cortex from the results of cortical lesions.<sup>1</sup> An important early observation was that temporal cortex lesions caused difficulties in identifying or remembering objects, while parietal lesions produced visual neglect and disordered representations of space.<sup>1</sup> Brain imaging methods, such as positron emission tomography (PET)<sup>2,3</sup> and functional magnetic resonance imaging (fMRI),<sup>4</sup> also support regional specialization in human visual cortex, and lesion studies indicate that selective losses can include color perception,<sup>5,6</sup> motion perception,<sup>7,8</sup> and face recognition.<sup>9</sup> While this evidence makes it clear that parallel processing is present in human visual cortex, it has not been able to clarify where independent processing is first seen and how it varies in different parts of visual cortex.

In contrast, our knowledge of parallel visual processing in macaque monkeys is much more detailed because of the range of experimental methods used to examine their visual cortex.<sup>10</sup> Macaques have parallel processing streams throughout their visual cortex,<sup>11</sup> and there are important differences between parallel streams at low and high levels of visual cortex, differences that are difficult to study in humans for the following reasons.

Only the highest levels of primate visual cortex, temporal and parietal areas, are usually tractable to

lesion studies. Parallel cortical streams at this level are large and widely separated in the brain, and lesions can selectively disrupt one stream, with sparing of the other. In addition, the resulting visual loss is not retinotopic, but is found throughout the visual field, and controlled fixation testing is not required to detect perceptual defects. Most functionally selective lesions that have been reported in humans have probably occurred at this level.<sup>8,12</sup>

It is more difficult to detect lesions of intermediate levels in primate visual cortex (areas MT, V4 and V2) because parallel streams at this level are more compact, making it more likely that a lesion will affect multiple streams. Also, cortical areas at this level are retinotopic and retinotopic visual deficits<sup>13,14</sup> will be missed if testing is done without controlled fixation. Curiously, patients with retinotopic defects that do not entirely eliminate the foveal representation seem to be completely unaware that they have a visual problem, and for that reason they do not come easily to the attention of neurologists. The patient reported in this study showed a precisely retinotopic defect, suggesting an effect at an intermediate level of cortex, and he was unaware before the start of formal testing that he had any visual loss.

Lesions are currently of little value in studying the distinctive processing streams at the lowest level in primate visual cortex because their close proximity results in damage to multiple streams. As a result, lesions of primary visual cortex in macaques or humans result in virtually complete blindness in a region of the visual field.<sup>14,15</sup>

The subject of the study reported here did not show the complete visual loss that is characteristic of V1 lesions, but his loss was precisely retinotopic, suggesting a lesion at an intermediate level in visual cortex. We measured a variety of visual capabilities along an arc of visual field locations  $20^\circ$  from the fovea that extended counterclockwise from upper right to lower left. At all locations in the upper left quadrant of his visual field this subject showed severe loss for all discriminations involving color, form or stationary luminance variations, but sparing for those involving motion.

## Materials and Methods

**Subject:** The subject (RP) was a 63 year-old-man who had suffered an infarct in his right extrastriate visual cortex about two years earlier. At the beginning of testing he was unaware that he had any visual loss, although, as a machinist, he used his vision extensively.

The location of the lesion in the patient's brain is indicated in Fig. 1 with T1 weighted magnetic resonance (MR) images obtained after the intravenous administration of a contrast agent. It was confirmed, with concurrent T2 weighted MR images and clinical followup, that the lesion was a subacute enhancing infarct. The region most severely damaged, shown in the image as white, is located on the fusiform gyrus at a level just posterior to the pons and mesencephalon.

**Visual testing:** General: visual performance was tested with the patient's head supported in a chin and forehead rest at 36 cm from a computer controlled display. Test stimuli were presented on the center of the display, and visual field location was varied by having the subject maintain fixation on a small dark spot on the testing display or on an annular surround. Fixation was monitored by the experimenter on a CCTV monitor that displayed a high gain image of the patient's eye. Before each test run, the patient

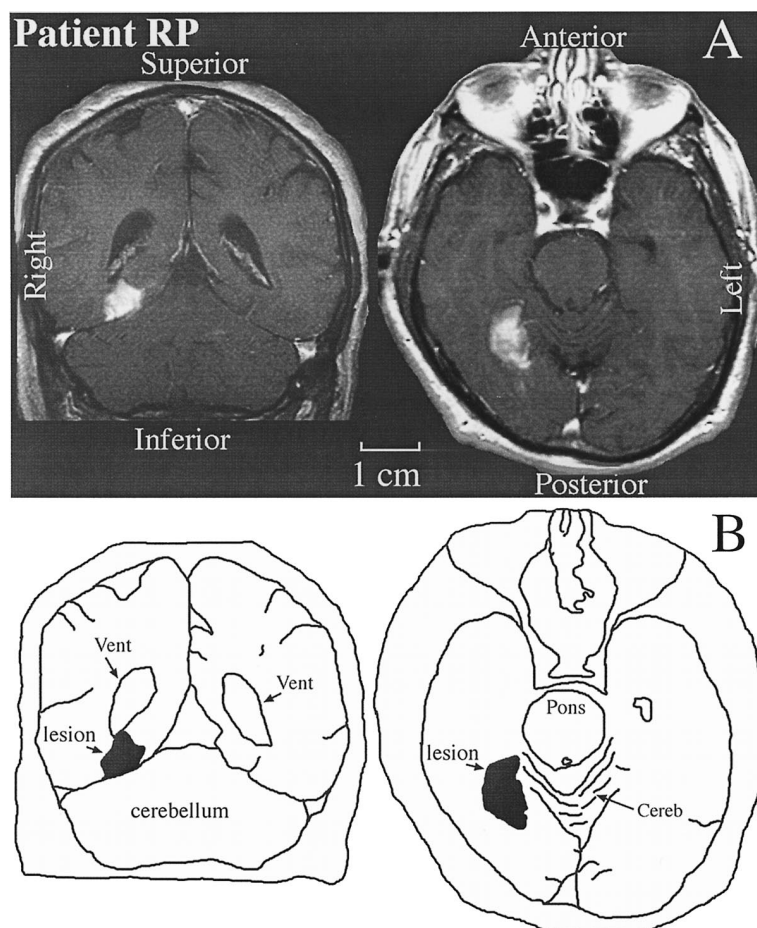


FIG. 1. **A.** Magnetic resonance images, in the coronal (left) and axial (right) planes, of the brain of subject RP, showing the location of the lesion as leakage of the contrast agent gadopentetate dimeglumine (white region). TR = 600, TE = 34, FOV = 20 cm,  $256 \times 192$  pixel image. **B.** Line drawings of the images in A showing the location of some neural landmarks and of the lesion.

was asked to foveate the fixation spot and then the test stimulus, and the experimenter marked the position of the eye for each. The subject was able to fixate very steadily, although if fixation breaks occurred, data from that trial were discarded. Thresholds were measured using either a staircase or a QUEST procedure.<sup>16</sup>

#### *Mapping of the visual field:*

All testing in this portion of the study was done along an arc at 20° eccentricity, extending counterclockwise from upper right (45°) to just below the left horizontal meridian (195°). This arc was chosen because it avoids the blind spot (temporal horizontal meridian at about 12–17°), and is far enough from the fovea so small variations in fixation are not critical, but is close enough so that form and color vision are reasonably good.

*Object naming:* the subject named common objects depicted by line drawings. At each of the seven locations tested, 10° high line drawings of common objects were presented. The drawings were chosen so that each group of ten included a full range of difficulties, from very easily recognized objects (e.g. hammer) to more complicated shapes (e.g. flamingo). To the right of Fig. 2A is an illustration of one of the tested objects, a flamingo.

*Chromatic increment sensitivity:* was tested with a two alternative temporal forced-choice procedure in which a 6° circular patch of color was presented in either the first or second test interval, which were marked by tones. Isoluminant red stimuli varied from the background white toward red along the constant blue axis in the DKL color space.<sup>17</sup> Isoluminant yellow stimuli varied from white toward yellow along the constant red–green axis of the DKL color space.<sup>17</sup> Stimuli were presented with a gaussian temporal waveform with the standard deviation of the gaussian (sigma) equal to 0.3 sec.

*Luminance contrast sensitivity (stationary):* was tested with a vertical–horizontal orientation discrimination using circular, 3° diameter, patches of stationary sinusoidal gratings of 1 or 2 c/°.

*Dot motion coherence thresholds:* was measured with a right–left direction discrimination using a circular 6° target that contained 200 dots. Thresholds were measured for the fraction of dots moving coherently (the remainder moved in random directions).

*Luminance contrast sensitivity (10 Hz drift):* was tested with a direction discrimination using 3° diameter patches of grating of low spatial frequency

(0.5 c/°), drifting at 10 Hz. Gratings moved either to the right or left, while the circular aperture remained stationary.

#### *Comparisons of affected versus intact visual field locations:*

These studies compared the patient's visual performance in the center of the affected location (upper left visual field at 20° eccentricity) with that in a control location in the middle of the upper right visual field.

*Luminance contrast sensitivity, comparison of drifting vs counterphase stimuli and detection vs discrimination thresholds:* thresholds were measured for detection and orientation discrimination using counterphase modulated gratings, and for detection and direction discrimination using drifting gratings. All thresholds were measured under two conditions of spatio-temporal frequency; slow modulation (1 c/°–1 Hz) and fast modulation (0.25 c/°–10 Hz). Stimuli were circular patches of grating of 4° diameter.

*Luminance contrast sensitivity: temporal frequency of counterphase modulation:* This study examined in more detail the counterphase orientation discrimination of the previous study over a wider range of temporal frequencies and for a single spatial frequency. Sensitivity was measured for 3° circular patches of 1 c/° gratings with temporal frequencies of counterphase modulation that ranged from 0–20 Hz.

*Chromatic contrast sensitivity: temporal frequency of drift modulation:* 10° circular patches of vertical, isoluminant grating of 0.15 c/° were modulated along the constant blue (i.e. red–green) direction in DKL color space. Stimuli drifted to the right at temporal frequencies of 0–7.5 Hz. Thus, at the 7.5 Hz rate of temporal modulation, the stimulus moved at 50°/sec. The subject performed a temporal forced-choice, responding to whether the stimulus appeared in the first or second interval. The time course of the stimuli was the same as for the color increments described above. After each response, the subject was asked whether he could see the color and direction of the stimulus.

## Results

Figure 2A illustrates shape naming along an arc of locations at 20° eccentricity. Filled triangles show performance of the patient and open circles that of a normal control. Patient RP identified all objects at the two locations that fell outside of the upper left visual field quadrant, 195°, (slightly below left

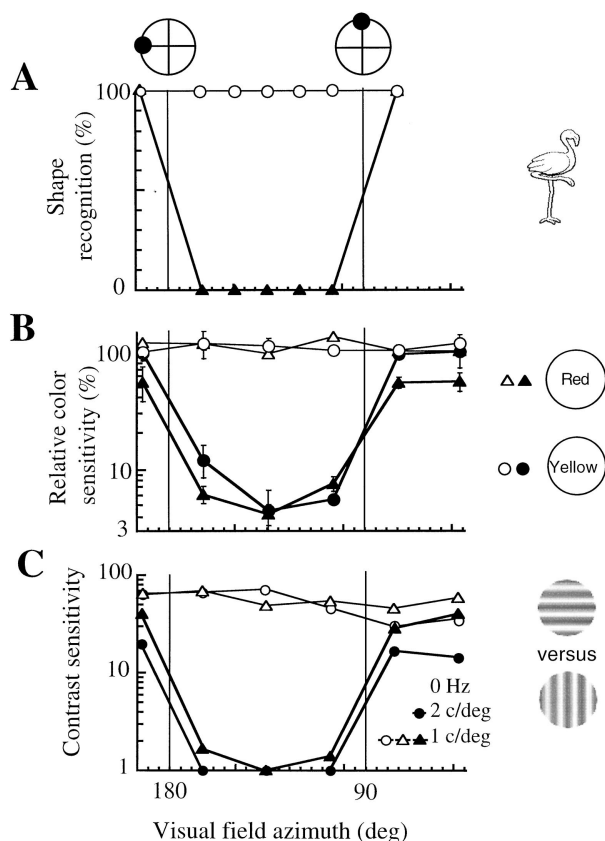


FIG. 2. Three measures of visual sensitivity that showed severe loss along an arc at 20° eccentricity that extended counterclockwise from 45° (upper right) to 195° (left and slightly down). **A.** Percent correct naming of ten objects presented at each of seven locations in and near the upper left visual field for subject RP (filled symbols) and a control observer (open symbols). At each tested location 10° high line drawings of common objects were presented. The drawings were chosen so that each group of ten included a full range of difficulties, from very easily recognized objects (e.g. hammer) to more complicated shapes (e.g. flamingo). To the right of the graph is an illustration of one of the tested objects, a flamingo. **B.** Relative sensitivity to red (triangles) and yellow (circles) color contrast for subject RP (filled symbols) and a control observer (open symbols). A sensitivity of 100 indicates the same sensitivity as the mean across all tested locations of the control observer's sensitivity. Error bars show  $\pm$  SD. **C.** Luminance contrast sensitivity for orientation discrimination measured with circular, 3° diameter, patches of static sinusoidal grating of 2 c/° (filled circles) or 1 c/° (all other symbols) spatial frequency, presented with onset and offset that followed a raised cosine of 2 sec period. Grating patches were either vertical or horizontal in orientation. Filled symbols show measures for subject RP, and open symbols measures for two control observers, who were tested at 1 c/°.

horizontal) and 75° (slightly to the right of the vertical meridian). However, at all five locations tested in the upper left quadrant he was unable to name any of the objects, although there was a very wide range of object difficulties at each location.

It can be seen that the same distribution of loss across the visual field was found for color detection (Fig. 2B). Detection of isoluminant red (triangles) and yellow (circles) targets was severely degraded for the patient (filled symbols) relative to a control observer (open symbols), at the same visual field locations

where shape recognition was impaired, but not at locations across the vertical or horizontal meridia of the visual field. The maximum extent of loss was approximately a factor of 20 for both red and yellow stimuli. At all locations RP could detect the presentation of the color stimulus at high color contrasts, but he was unable to discriminate its color under any conditions within the affected visual field. By contrast, at the three tested locations outside of the affected quadrant, he was able to describe the color of the test stimulus even at close to threshold contrasts.

Figure 2C shows severe loss over the same portion of the visual field for luminance contrast sensitivity measured with an orientation discrimination using stationary, slow onset stimuli. Again the patient data are shown as filled symbols for two different spatial frequencies, and the control data as open symbols. Although the patient had somewhat lower sensitivity than the control outside the upper left quadrant, his sensitivity within that quadrant was reduced at least 20-fold compared to his thresholds in the upper right and lower left field.

Figure 3 illustrates two visual thresholds which showed little or no impairment in the upper left visual field quadrant. Motion coherence thresholds, (Fig. 3A), were not decreased in the region where Fig. 2 showed devastated visual sensitivity for color, shape and stationary luminance stimuli. Fig. 3B, like Fig. 2C, shows luminance contrast sensitivity, but in this case tested with direction discrimination for fast moving, drifting stimuli. Sensitivity loss for the patient (filled triangles) was approximately two-fold, compared to the more than 20-fold illustrated in Fig. 2C.

Figure 4 shows loss of luminance contrast sensitivity for subject RP in the affected upper left visual field relative to the unaffected upper right visual field. Sensitivity in the affected field was reduced only slightly for measures of detection (triangles) or direction discrimination (diamonds) measured with drifting gratings. Loss for both of these measures was approximately two-fold at low spatio-temporal frequency and even less at higher spatio-temporal frequency. On the other hand, sensitivity loss for detection (circles) and orientation discrimination (squares), measured with counterphase modulated gratings, was more substantial. Loss was approximately ten-fold for both of these measures at the low spatio-temporal frequency, but less than three-fold at higher spatio-temporal frequencies.

Figure 5 compares luminance contrast sensitivity for the control and affected field of the subject over a wide range of temporal frequencies of counterphase (flicker) modulation. Contrast sensitivity in both fields shows the typical peak at mid frequencies with

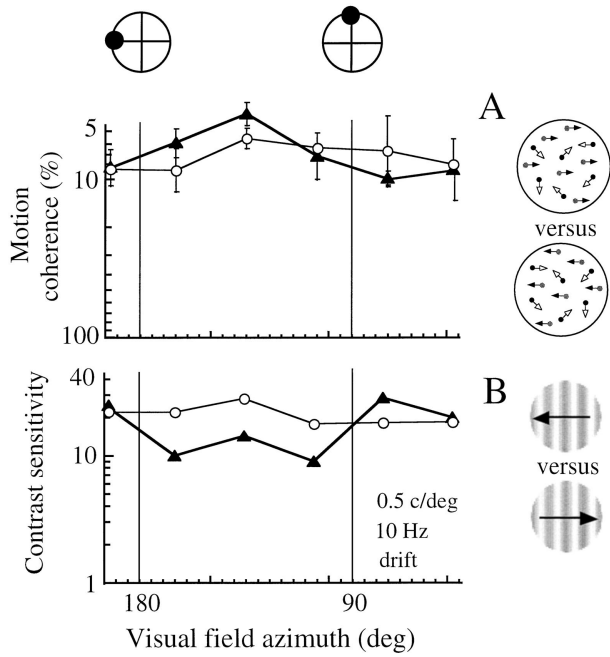


FIG. 3. Two measures of visual sensitivity that showed little loss along the same arc of the visual field as in Fig. 2 **A**. Motion coherence thresholds measured with random dot stimuli for subject RP (triangles) and a control subject (circles). The ordinate shows the proportion of dots that moved in the direction to be discriminated (the others moved in random directions). RPs sensitivity was best, as well as better than that of the control subject, at some locations within the affected quadrant of the visual field. Error bars show  $\pm$  SD. **B**. Luminance contrast sensitivity for direction discrimination measured with circular  $3^\circ$  diameter patches of grating of  $0.5\text{ c/deg}$  spatial frequency, drifting at 10 Hz. Gratings moved either to the right or left, while the circular aperture remained stationary. Filled symbols show data for subject RP, and open symbols for a control observer.

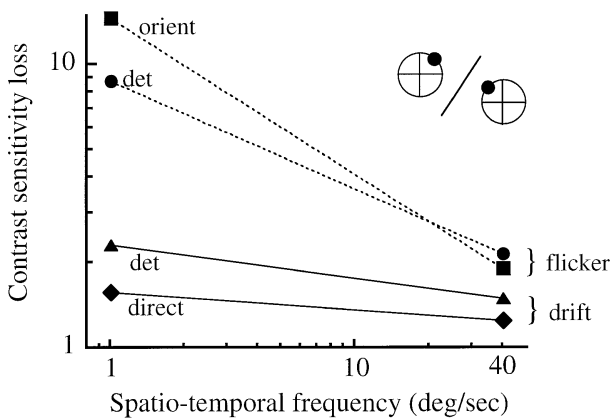


FIG. 4. Luminance contrast sensitivity loss for subject RP in the affected quadrant of his visual field (azimuth  $135^\circ$  (upper left) compared to the control location (upper right)). Eight thresholds were measured at each of these locations for combinations of the following conditions: grating spatio-temporal frequency was slow ( $1\text{ c/deg}$  vs fast ( $0.25\text{ c/deg}$  10 Hz); grating modulation was counterphase vs drifting; and the psychophysical task was detection (counterphase or drifting) vs discrimination (orientation or direction). Diamonds show direction discrimination and triangles simple detection, both measured with drifting stimuli. Squares show orientation discrimination and circles simple detection, both measured with counterphase modulated (i.e. flickering) gratings. For all of the above measures, stimuli were circular patches of  $4^\circ$  diameter presented at  $20^\circ$  eccentricity.

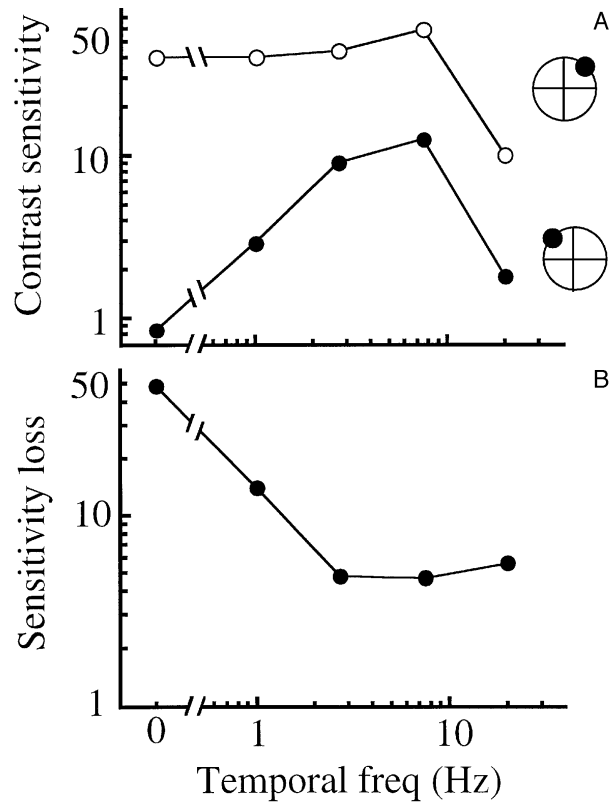


FIG. 5. **A**. Luminance contrast sensitivity for subject RP in the affected upper left (filled symbols) and unaffected upper right (open symbols) quadrants of his visual field. Thresholds were measured with patches of grating of  $1\text{ c/deg}$  as a function of the temporal frequency of counterphase modulation. Grating patches were circular with a diameter of  $3^\circ$  and were presented at  $20^\circ$  eccentricity. **B**. Sensitivity loss in the upper left, compared to the upper right, visual field.

a falloff for temporal frequencies above 10 Hz. The loss of contrast sensitivity in the affected, compared to the unaffected, field was maximal at 0 Hz and then decreased by almost a factor of ten before reaching an asymptote about 3 Hz.

The effect of drift rate on chromatic contrast sensitivity is shown in Fig. 6 for control and affected fields. The major result seen here is that the magnitude of sensitivity loss (lower figure) did not decline at higher temporal frequencies (speed of drift). Questioning the subject during this experiment revealed that he could see the direction of motion in his affected field at detection threshold, but could not discern the color of the stimuli, even for the highest chromatic contrasts that could be presented, which were well above his detection threshold.

All of the testing described above was done approximately two years after the occurrence of the stroke. Monitoring of sensitivity throughout the period of testing was accomplished by repeatedly measuring contrast sensitivity in the affected quadrant, and thresholds remained constant over the testing period.

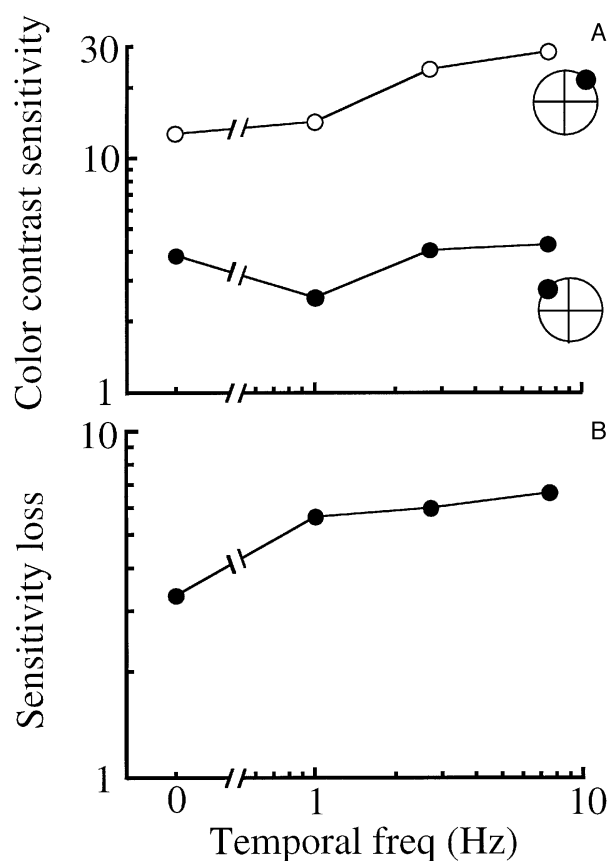


FIG. 6. **A.** Chromatic contrast sensitivity for subject RP in the affected upper left (filled symbols) and unaffected upper right (open symbols) quadrants of his visual field. Thresholds were measured with patches of grating of  $0.15\text{ c}/^\circ$  as a function of the temporal frequency of drift modulation. Gratings were isoluminant red-green (constant blue in the DKL color space),<sup>17</sup> and test patches were circular with a diameter of  $10^\circ$  and were centered at  $20^\circ$  eccentricity. A sensitivity of 1 represents the maximal r/g contrast that could be produced on the video display. **B.** Sensitivity loss in the upper left, compared to the upper right, visual field.

## Discussion

This study demonstrates that an apparently focal cortical lesion caused a loss of form vision, color perception, and sensitivity to those luminance stimuli that were stationary or slowly flickered, but little or no sensitivity loss for moving gratings or dots. This selective loss suggests, as is true in macaques,<sup>11,18</sup> that in humans there are separate cortical processing streams with different responses to basic stimuli, that underlie the perception of motion versus the perception of color and form. The deficit was confined to one quadrant of the visual field, showing both that the loss was not due to a global perceptual disorder, and that a retinotopic part of the visual pathways was involved, perhaps a portion of near-extrastriate cortex.<sup>19</sup> Furthermore, the deficit was present several years after the occurrence of the cortical lesion that gave rise to it, suggesting that there was little or no amelioration of the deficit by cortical plasticity.

**Does the loss involve more than simply reduced sensitivity to slowly changing stimuli?** In Fig. 4 the greatest loss of sensitivity was for stimuli that underwent slow counterphase modulation. Similar loss was seen for stimuli that were stationary with slow onset (Fig. 2C). Comparison of these results makes it clear that the loss was not due to the orientation discrimination, since contrast loss was essentially identical whether contrast thresholds were measured using detection or orientation discrimination. Indeed, when we tested contrast sensitivity for orientation discrimination with 1 Hz drifting stimuli, (not shown), sensitivity loss was no greater than was found for detection of the 1 Hz drifting stimulus in Fig. 4. Sensitivity for luminance stimuli was optimal if they drifted rapidly, although slow drift or rapid flicker also resulted in substantial sparing (Figs. 4, 5). These findings raised the possibility that the severe losses illustrated in Fig. 2 might be due only to the minimal speed or temporal variation of the stimuli. However, the results shown in Fig. 6 make clear that even very high velocities (up to  $50^\circ/\text{sec}$ ) did not ameliorate the loss of color vision. Likewise, the deficit in shape recognition did not appear to be secondary to the severe loss of sensitivity for slowly changing stimuli. The line drawings represented in Fig. 2A were presented with abrupt onset, a type of presentation that in other studies (not described here) greatly improved RPs sensitivity to high spatial frequency gratings. In informal tests, we also drifted the shape stimuli during the observation period, but this change greatly disrupted naming in the unaffected field and did not help in the affected field. Therefore, this study demonstrated a correlated loss of sensitivity to shape, color and slowly changing luminance stimuli, with relative preservation of sensitivity to more rapidly changing stimuli.

**Are the spared functions mediated by non-cortical pathways?** Our interpretation of these results as selective damage to a particular cortical processing stream, with preservation of another, is viable only if the preserved motion sensitivity is mediated cortically. We can completely rule out a lesion of the optic radiations, since this patient does not show the profound visual field loss that accompanies such lesions. Preservation of a few visual capabilities (localization of bright stimuli, detection of object motion, etc.) after lesions of cortical area V1 has been observed by several investigators and termed 'blindsight'.<sup>20</sup> The present results would be most uninteresting if it were simply the case that the disrupted shape and color perception depended on cortical processing, but that the spared motion perception was mediated by non-cortical mechanisms. There are several points that argue against this possibility. First, the dot stimuli consisted of either

200 dots in a 6° diameter stimulus or 100 dots in a 3° diameter stimulus (data not shown). In both cases the dot density is rather high (7 or 14 dots/°<sup>2</sup>) for resolution by non-cortical mechanisms. Furthermore, all motion was within a stationary aperture, i.e. the dot stimulus did not translate across the visual field, giving little opportunity for the observer to infer direction by making a successive location discrimination.<sup>21</sup> Under these conditions it is very likely that direction discrimination is cortically mediated. Finally, it has previously been shown in macaques<sup>22</sup> that a lesion of cortical areas MT/MST severely disturbs motion coherence sensitivity. Thus the preserved motion perception seems to depend on cortical mechanisms.

**Which cortical pathways were affected, and which spared?** The lesion was located in the region most commonly associated with achromatopsia, as well as the loss of object recognition,<sup>5,9,23</sup> both of which this patient shows. Conversely, area V1, which is located in the calcarine cortex, posterior and superior to the location of the lesion, showed no evidence of damage. The fact that the border between impaired and spared portions of the visual field lies along the horizontal meridian suggests that the locus of damage is likely to be in cortical areas that represent quadrants of the visual field,<sup>19</sup> as extrastriate visual areas V2 and V4 do in the macaque. This suggestion is supported by the finding that lesions of these cortical areas in macaques disrupt shape perception.<sup>13,14,24</sup> While color vision loss has not been found after lesions of cortical areas V2 or V4 in the macaque,<sup>14,25</sup> the more severe effects found here in a human subject may be due to damage to fiber pathways between cortical areas, which was not present in the macaque studies. Finally, the sparing of motion coherence sensitivity, found in this study, suggests that cortical areas analogous to MT and MST in the macaque were likely spared, since lesions of these areas markedly disrupt motion coherence thresholds.<sup>22</sup>

## Conclusions

We have found visual loss confined to a quadrant of the visual field in a subject who had suffered a

localized cortical lesion. The visual defect was severe for color and shape perception as well as for contrast sensitivity tested with stationary or slowly changing stimuli. However, sensitivity for visual motion was preserved. These results appear to reflect parallel processing (independence of color and shape perception from motion perception) in a region of near extrastriate human visual cortex (which is marked by precise localization to one portion of the visual field).

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