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Both Striate Cortex and Superior Colliculus Contribute to Visual Properties of Neurons in Superior Temporal Polysensory Area of Macaque Monkey

CHARLES J. BRUCE, ROBERT DESIMONE, AND CHARLES G. GROSS

SUMMARY AND CONCLUSIONS

1. Although the tectofugal system projects to the primate cerebral cortex by way of the pulvinar, previous studies have failed to find any physiological evidence that the superior colliculus influences visual activity in the cortex. We studied the relative contributions of the tectofugal and geniculostriate systems to the visual properties of neurons in the superior temporal polysensory area (STP) by comparing the effects of unilateral removal of striate cortex, the superior colliculus, or of both structures.

2. In the intact monkey, STP neurons have large, bilateral receptive fields. Complete unilateral removal of striate cortex did not eliminate visual responses of STP neurons in the contralateral visual hemifield; rather, nearly half the cells still responded to visual stimuli in the hemifield contralateral to the lesion. Thus the visual properties of STP neurons are not completely dependent on the geniculostriate system.

3. Unilateral striate lesions did affect the response properties of STP neurons in three ways. 1) Whereas most STP neurons in the intact monkey respond similarly to stimuli in the two visual hemifields, responses to stimuli in the hemifield contralateral to the striate lesion were usually weaker than responses in the ipsilateral hemifield. 2) Whereas the responses of many STP neurons in the intact monkey were selective for the direction of stimulus motion or for stimulus form, responses in the hemifield contralateral to the striate lesion were not selective for either motion or form. 3) Whereas the median receptive field in the intact monkey extended 80° into the contralateral visual field, the receptive fields of cells with responses in the contralateral field that survived the striate lesions had a median border that extended only 50° into the contralateral visual field.

4. Removal of both striate cortex and the superior colliculus in the same hemisphere abolished the responses of STP neurons to visual stimuli in the hemifield contralateral to the combined lesion. Nearly 80% of the cells still responded to visual stimuli in the hemifield ipsilateral to the lesion.

5. Unilateral removal of the superior colliculus alone had only small effects on visual responses in STP. Receptive-field size and visual response strength were slightly reduced in the hemifield contralateral to the collicular lesion. As in the intact monkey, selectivity for stimulus motion or form were similar in the two visual hemifields.

6. We conclude that both striate cortex and the superior colliculus contribute to the visual responses of STP neurons. Striate cortex is crucial for the movement and stimulus specificity of neurons in STP. The superior colliculus is crucial for the visual responses that survive striate removal. We suggest that STP may contribute to the visually guided behavior that survives total removal of striate cortex.
INTRODUCTION

Many regions of the primate cerebral cortex have neurons that respond to visual stimuli, and all of these areas appear to receive either direct or indirect inputs from striate cortex. Most of them, however, could also receive visual information from the superior colliculus by way of the pulvinar (17, 18). Furthermore, considerable visually guided behavior survives removal of striate cortex in primates (1, 25, 28, 36, 38, 39, 59), and tectofugal pathways to cortex might contribute to this behavior.

In previous studies it was found that visual responses of neurons in V2 and inferior temporal cortex are abolished by cooling or removal of striate cortex (46, 49), indicating that the visual functions of these two extrastriate areas depend primarily or entirely on the geniculostriate system. Beyond this, little is known about the relative importance of striate cortex and the superior colliculus for the visual properties of cells in different cortical areas.

In the present study we investigated the contributions of the geniculostriate and tectofugal systems to visual responses in the superior temporal polysensory area (STP). STP lies in the upper bank and fundus of the anterior portion of the superior temporal sulcus of the macaque monkey (15). Its thalamic efferents are from the medial pulvinar (9) which, in turn, receives projections from the deep laminae of the superior colliculus (4, 5, 24). STP also receives projections from several cortical areas containing visually responsive neurons, including inferior temporal cortex and posterior parietal cortex (27). In a previous study (6) we found that the activity of most cells in STP suggests a role in spatial vision, which classically has been associated with the tectofugal system (51, 55). Almost all STP neurons are visually responsive, and many also respond to auditory and somesthetic stimuli. Visual receptive fields of STP neurons are very large and often encompass most of the monkey’s visual field. Many STP cells are directionally selective, including cells sensitive to motion toward or away from the fovea and cells sensitive to motion in depth. Most STP cells respond to stimuli of nearly any size, contrast, or shape, although a minority are selective for particular complex forms such as faces (6, 40).

We compared the effects of unilateral lesions of striate cortex, of the superior colliculus, and of both structures on visual responses of STP neurons. Because nearly all STP visual receptive fields in the intact monkey are bilateral, unilateral lesions allowed us to also compare the responses of each neuron to stimuli in the intact ipsilateral visual hemifield with its responses to stimuli in the hemifield contralateral to the lesions. We found that nearly half the neurons in STP still respond to visual stimuli in the hemifield contralateral to the striate cortex lesions and that this surviving activity is critically dependent on the superior colliculus. These findings demonstrate that STP receives visual inputs from both the geniculo and tectofugal systems, and indicate that STP could contribute to visually guided behaviors in the absence of striate cortex.

METHODS

Subjects

Eight male monkeys (Macaca fascicularis) weighing between 4 and 5 kg were used. Table 1 summarizes, for each monkey, the type of lesions and the number of cells recorded following each lesion. Five monkeys received an ablation of the left striate cortex and were allowed 1-6 mo recovery. They were then recorded from four to nine times over a period of 1-2 mo. The left superior colliculus in three of these monkeys was subsequently ablated and, after 4-6 days recovery, the monkeys were studied in 2-4 more recording sessions. Three additional monkeys were recorded from three to four times each after lesions of the left superior colliculus, with 3-5 days recovery before the initial recording session. For all monkeys given a collicular lesion, the final recording session occurred at least 2 wk after the lesion was made.

After the last recording session all monkeys were given an overdose of pentobarbital sodium and perfused with saline followed by buffered formalin. Frozen sections were cut in the coronal plane, stained with cresyl violet, and examined to determine the recording sites in STP and the extent of the cortical and collicular lesions.

Lesions of striate cortex and superior colliculus

Lesions of the left striate cortex were made using the method described by Mishkin (35). First, a partial occipital lobectomy was performed, using a coronal cut 8 mm posterior to the lunate sulcus. The remaining striate cortex on the lateral and medial surface was then aspirated under direct vision, and striate cortex in the depths of the calcarine fissure was aspirated with the aid of an operating microscope. The dural flap was sutured, and the soft tissue was closed in anatomical layers.
TABLE 1. Summary of lesions and STP cells studied

<table>
<thead>
<tr>
<th>Monkey</th>
<th>Striate Cortex Lesion</th>
<th>No. STP Cells</th>
<th>Colliculus Lesion</th>
<th>No. STP Cells</th>
</tr>
</thead>
<tbody>
<tr>
<td>S7</td>
<td>Fovea sparing</td>
<td>112</td>
<td>Complete</td>
<td>79</td>
</tr>
<tr>
<td>S8</td>
<td>Complete</td>
<td>66</td>
<td>Complete</td>
<td>82</td>
</tr>
<tr>
<td>S9</td>
<td>Peripheral sparing</td>
<td>99</td>
<td>Complete</td>
<td>83</td>
</tr>
<tr>
<td>S10</td>
<td>Complete</td>
<td></td>
<td>Complete</td>
<td>38</td>
</tr>
<tr>
<td>S11</td>
<td>Complete</td>
<td>108</td>
<td>Complete</td>
<td>33</td>
</tr>
<tr>
<td>S12</td>
<td>Complete</td>
<td>106</td>
<td>Complete</td>
<td>97</td>
</tr>
</tbody>
</table>

Total units: after striate cortex lesion alone, 491; after superior colliculus lesion alone, 154; after both lesions combined, 258.

In each monkey, five radiofrequency lesions were made which collectively destroyed all of the left superior colliculus. The placement of the five lesions was guided by the location of visual receptive fields recorded on several microelectrode penetrations into the superior colliculus (10).

Recording procedure

Our recording procedure has been fully described elsewhere (6, 15). One week prior to recording, a stainless steel chamber (3-cm diam) and a headbolt were fixed to the skull. The headbolt held the monkey in a stereotaxic instrument during recording. The recording chamber was located on the dorsal surface of the skull, with its center at stereotaxic coordinates 15-mm frontal and 1-mm lateral.

For each recording session the monkey was first anesthetized with halothane and nitrous oxide and intubated for subsequent respiration. The monkey was then paralyzed with pancuronium bromide (Pavulon) and anesthetized for the remainder of the recording session with a mixture of nitrous oxide and oxygen (70:30). Both eyes were treated with cyclopentolate and were focused at 57 cm with contact lenses. EKG and expired carbon dioxide were monitored throughout the recording sessions, which lasted 10–15 h. At the end of each session the Pavulon infusion was stopped, and when the monkey was breathing normally it was returned to its cage. At least 2 days intervened between successive recording sessions.

Single cells were isolated with varnish-coated tungsten microelectrodes oriented in the coronal plane, but angled 20–30° toward the midline (see Fig. 1). To protect the electrodes, they were retracted inside a guide tube that was lowered through the dura before the electrode was advanced. The identification of penetrations and location of recording sites was aided by the pattern of small lesions (4 μA, 20 s) made during the recordings and by studying cells in cortical areas above and below STP.

Visual stimuli

For each neuron isolated, visual responses in both the left and right hemifield were studied independently. In particular, each hemifield was tested for 1) visually elicited responses, 2) receptive-field borders, 3) relative response strength in the two hemifields, and 4) direction of movement and stimulus preferences.

Both projected and front-illuminated stimuli were used. Projected stimuli included light slits and spots, dark bars and edges, and a variety of complex patterns on 35-mm slides. Stimuli were rear-projected onto a 70- × 70-cm Polacoat tangent screen 57 cm from the monkey. The background screen illumination was 1.3 FT-L, and light and dark stimuli were roughly 1.5 log units above or below this level. Front-illuminated stimuli included white and patterned cardboard squares (ranging from 0.5 to 100 cm in extent) and various objects and pictures. They were illuminated by a 150 W reflector-flood lamp located above and behind the monkey’s head and were shown against uniform backgrounds, usually a black cloth. Background luminance was typically 0.2 FT-L, and most stimuli were ~1 log unit brighter.

Stimuli were presented either manually or automatically. Projected stimuli could be presented by an optical bench under computer control. Three motorized devices allowed computer-controlled presentation of front-illuminated objects. One device was an arm that moved stimuli in an arc (radius 51 cm) about the monkey’s head, usually along either the horizontal or vertical meridian. Another device moved stimuli along linear paths and was used primarily to move stimuli in depth, that is, directly toward or away from the monkey’s eye. A third device rotated objects about their centers, that is, created a spinning motion. Potentiometers mounted on these devices monitored stimulus location, thus allowing the compilation of unit activity as a function of stimulus position or motion.

Visual receptive fields

Receptive fields were plotted on either the tangent screen described above, on a translucent hemisphere (radius 45 cm, centered on the contralateral eye), or on both. The optic disk and fovea of each eye
FIG. 1. Recording sites in superior temporal polysensory area (STP) and lesion reconstructions in one monkey (S8) with complete unilateral lesions of striate cortex and the superior colliculus. Coronal section E shows 3 representative electrode penetrations with terminal marking lesions in STP. Arrows on lateral view of left hemisphere (top middle) indicate total extent of STP recording sites across all monkeys. Striate cortex lesion is black (top middle), with the intact right hemisphere (top right) shown for comparison. Sections 1–3 show the striate cortex lesion; striate cortex in the intact right hemisphere is indicated by dashed lines. Sections 4–6 show superior colliculus lesion. Sulci abbreviations: ca, calcarine; ce, central; ip, intraparietal; l, lunate; la, lateral; oi, inferior occipital; ot, occipito-temporal; po, parieto-occipital; tma, anterior middle temporal; ts, superior temporal. Structure abbreviations: Cd, caudate; Gld, dorsal lateral geniculate; Gr, central grey; H, habenula; Hi, hippocampus; IC, inferior colliculus; Md, medial-dorsal nucleus; P, inferior pulvinar; Pl, lateral pulvinar; Pm, medial pulvinar; Pr, pretectum; Put, putamen; SC, superior colliculus; Thal, thalamus.
were projected, using an ophthalmoscope and a
corner-cube prism, and marked on both the hemi-
sphere and the tangent screen. The horizontal me-
idian was taken to be the line passing through the
fovea and the center of the optic disk, and the ver-
tical meridian to be the line perpendicular to the
horizontal meridian and intersecting the fovea.
Receptive fields were first mapped using the eye con-
tralateral to the recording site, and then using the
ipsilateral eye to determine the receptive field's ex-
tension into the ipsilateral monocular crescent. At
the end of each recording session receptive-field
plots on the hemisphere were photographed.

Nonvisual stimulation
Auditory stimuli included clicks, tones, jangling
keys, and vocalizations. Somesthetic stimuli in-
cluded light taps, stroking the skin, manipulation
of the limbs, and deep pressure.

RESULTS

We studied 903 neurons in STP in the eight
monkeys with lesions. As shown in Fig. 1, STP
recording sites extended over the anterior two-
thirds of the superior temporal sulcus. Addi-
tional cells from cortical areas adjacent to STP
were also studied.

Table 1 summarizes the STP cells sampled
in each lesion group. The effects of the lesions
on visual responses in STP were assessed in
three ways. First, we compared responses in
these monkeys to the responses of a sample of
452 STP cells from six normal monkeys that
we previously studied using essentially iden-
tical procedures (6). Second, we compared the
visual responses across the different lesion
groups; in particular, responses after striate le-
sions were compared with responses from the
same monkeys after the superior colliculus in
the same hemisphere was ablated. Finally, we
compared visual responses in the visual fields
ipsilateral and contralateral to the lesions. This
last comparison most directly assesses the ef-
facts of the unilateral lesions of striate cortex
and the superior colliculus as these structures
represent only the contralateral visual field.

Effects of striate cortex lesions

HISTOLOGICAL VERIFICATION. Examination
of stained sections through visual cortex and
the dorsal lateral geniculate nucleus and pul-
vinar indicated that for three monkeys (S8,
S12, and S14) the striate lesions were com-
plete, and extrastriate damage was minimal
(Fig. 1). For one monkey (S7) a small strip of
striate cortex on the lateral surface along the
border with V2 was spared, and the topo-
graphically corresponding region of the lateral
geniculate nucleus was normal in appearance.
For another monkey (S9) a portion of striate
cortex buried in the anterior end of the cal-
carne sulcus was spared, and the correspond-
ing part of the lateral geniculate was normal.

The intact striate cortex in the depths of the
calcarine fissure in monkey S9 undoubtedly
contributed to visual responses in the contra-
lateral hemifield and, thus, affected measure-
ments of STP receptive fields. Consequently,
we excluded cells recorded from this monkey
from statistics, tables, and figures that sum-
marize the effects of striate lesions on visual
receptive fields. Based on the visual topogra-
phy of striate cortex (13) and the lateral ge-
niculate nucleus (32) the spared tissue repre-
sented a large expanse of the peripheral con-
tralateral visual field. In fact, 83% of the
visually responsive STP cells from this monkey
responded to stimuli in the contralateral
hemifield, versus 47% in the monkeys with
complete striate lesions. Furthermore, only in
this monkey did STP cells continue to respond
to stimuli in the contralateral hemifield after
a superior colliculus lesion was added ipsilat-
eral to the striate lesion.

Conversely, the spared striate cortex at the
V2 border on the lateral surface in monkey S7
could have only minimally affected receptive-
field measurements. We estimate that the
spared tissue represented a small strip of visual
field along the vertical meridian in the upper
visual field, 1–2° wide and 3–4° tall. We did
not classify receptive fields that crossed <3°
into the contralateral field as bilateral because
such small overlaps could reflect scatter in the
crossing at the optic chiasm, errors in plotting
the retinal landmarks, or small eye movements
after the retinal landmarks were plotted. In
fact, visual responses in the contralateral
hemifield of this monkey usually could be
elicited 40–60° from the vertical meridian, and
the overall results from this monkey were very
similar to those from the three monkeys with
complete striate lesions. Therefore, we in-
cluded cells from this monkey in all summa-
ries of the effects of striate lesions.

INCIDENCE OF VISUAL RESPONSES. We stud-
ied 491 cells in five monkeys with unilateral
striate cortex lesions (Table 1). Table 2 sum-
TABLE 2. Modality of STP responses, percent

<table>
<thead>
<tr>
<th>Lesion</th>
<th>None*</th>
<th>Striate</th>
<th>Colliculus</th>
<th>Both</th>
</tr>
</thead>
<tbody>
<tr>
<td>Vis</td>
<td>41</td>
<td>39</td>
<td>47</td>
<td>37</td>
</tr>
<tr>
<td>Vis + Aud</td>
<td>21</td>
<td>11</td>
<td>16</td>
<td>10</td>
</tr>
<tr>
<td>Vis + Som</td>
<td>17</td>
<td>11</td>
<td>11</td>
<td>12</td>
</tr>
<tr>
<td>Vis + Aud + Som</td>
<td>17</td>
<td>18</td>
<td>7</td>
<td>20</td>
</tr>
<tr>
<td>Vis, total</td>
<td>96</td>
<td>79</td>
<td>81</td>
<td>80</td>
</tr>
<tr>
<td>Not Vis</td>
<td>1</td>
<td>7</td>
<td>3</td>
<td>7</td>
</tr>
<tr>
<td>Not responsive</td>
<td>2</td>
<td>16</td>
<td>17</td>
<td>14</td>
</tr>
<tr>
<td>Total no. cells</td>
<td>383</td>
<td>440</td>
<td>148</td>
<td>226</td>
</tr>
</tbody>
</table>

* From Bruce, Desimone, and Gross, 1982 (6). Vis, visual; Aud, auditory; Som, somesthetic.

TABLE 3. Receptive-field laterality of STP neurons, percent

<table>
<thead>
<tr>
<th>Lesion</th>
<th>Bilat</th>
<th>Contra</th>
<th>Ipsi</th>
<th>No. Cells</th>
</tr>
</thead>
<tbody>
<tr>
<td>None*</td>
<td>92</td>
<td>7</td>
<td>1</td>
<td>256</td>
</tr>
<tr>
<td>Striate cortex</td>
<td>47</td>
<td>1</td>
<td>52</td>
<td>216</td>
</tr>
<tr>
<td>Superior colliculus</td>
<td>97</td>
<td>3</td>
<td>0</td>
<td>99</td>
</tr>
<tr>
<td>Both</td>
<td>1</td>
<td>0</td>
<td>99</td>
<td>129</td>
</tr>
</tbody>
</table>

* From Bruce, Desimone, and Gross, 1982 (6).

marizes the percentages of visual, nonvisual, and nonresponsive cells. The proportion of visually responsive cells (79%) after unilateral lesions of striate cortex was less than the proportion in intact monkeys (96%), and there was a corresponding increase in the proportion of nonresponsive cells and of cells responsive to only auditory or somesthetic stimuli.

VISUAL RECEPTIVE FIELDS. As expected from our previous results in normal monkeys, most cells responded to visual stimuli in the intact visual hemifield ipsilateral to the striate lesions. Surprisingly, 47% of the visually responsive cells also responded to stimuli in the hemifield contralateral to the striate lesion. Fifty-two percent responded only in the field ipsilateral to the lesion, and only 1% responded exclusively in the field contralateral to the lesion. Thus, as summarized in Table 3 and Fig. 2,

![Diagram](http://jn.physiology.org/)

**FIG. 2.** Superior temporal polysensory area (STP) receptive fields in the different lesion groups. Histograms show percentage of bilateral (B), contralateral (C), and ipsilateral (I) receptive fields for each condition. Diagrams below indicate the median STP receptive field for each lesion condition; for the striate cortex lesion both bilateral and ipsilateral fields are depicted. Fields for normal monkeys are from Ref 6.
FIG. 3. Responses of superior temporal polysensory area unit to flashed stationary stimuli in a monkey with a unilateral lesion of striate cortex. The stimulus was a 5° square light presented in 6 locations, 3 in each hemifield. The unit responded at all locations, including loci in the hemifield contralateral to the lesion. Bars under histograms indicate stimulus presentation. Each histogram is based on 10 trials.

Almost half the visually responsive STP cells retained their bilateral receptive fields following unilateral lesions of striate cortex.

Visual receptive fields of cells with bilateral responses were large and uniform. Figure 3 shows responses of a cell to a stationary stimulus presented at three locations within each hemifield: this cell responded to stimuli anywhere on the 64° square tangent screen and was not selective for stimulus form or motion. Figure 4 shows responses of another cell to moving stimuli in the hemifield contralateral.
to the striate lesion. This cell responded to motion commencing anywhere within its large receptive field.

For most cells the visual receptive fields extended beyond the tangent screen in one or more directions, and hence were plotted on the translucent hemisphere. The population of receptive fields after striate lesions was bimodal in that almost all receptive fields were either bilateral or ipsilateral, and within each class receptive-field size and shape were fairly homogeneous. Bilateral and ipsilateral receptive fields obtained from one electrode penetration in a monkey with a striate cortex lesion are shown at the top of Fig. 5. As illustrated in this figure, cells with bilateral fields usually responded to stimuli over 30° eccentric from the fovea in any direction; however, these bilateral receptive fields were not as large as fields in the intact monkey (6, 23). Considering only cells that responded to stimuli in the visual hemifield contralateral to the striate lesion, the median contralateral receptive field extended 50° (n = 64) into the contralateral hemifield. The median contralateral border in the intact monkey was 80°, a difference of 30°. In contrast, the other principal receptive-field borders were all within 10° of what we previously found in the intact monkey. The median ipsilateral border was 60° (n = 66); in the intact monkey it was 70°. The median upper and lower receptive field borders were +45° and −45°, respectively (n = 43 for both); in the intact monkey these median borders were +50° and −55°.

Figure 5 also shows ipsilateral receptive fields found on the same electrode penetration that yielded the bilateral fields shown. Although receptive fields confined to the ipsilateral hemifield are rare in the intact monkey.

FIG. 5. Typical receptive fields of superior temporal polysensory area units in monkeys with different experimental lesions. Both bilateral and unilateral fields were found after striate removal alone, in fact all the fields shown were recorded on the same electrode penetration. Mostly bilateral fields were found after the superior colliculus lesions, and only ipsilateral fields were found after the combined lesions of striate cortex and superior colliculus. Fields were plotted on a translucent hemisphere, and the spacing of the isoeccentricity lines reflects the photographic procedure used to record the plots.
(≈1%, see Ref. 6), 52% of the visually responsive cells from monkeys with striate cortex lesions had receptive fields confined to the ipsilateral hemifield (Table 3). The contralateral border of these receptive fields almost always coincided with the vertical meridian, and the other receptive-field boundaries usually extended into the far periphery; hence these ipsilateral receptive fields typically encompassed much of the monkey's ipsilateral visual hemifield.

As described in the METHODS, we measured the full extent of receptive fields by switching the eye occluder so that each monocular crescent could be tested. As in the intact monkey (6), nearly all STP cells responded comparably through either eye within the binocular portions of their receptive fields.

VISUAL RESPONSE STRENGTH. Striate lesions diminished the strength of visual responses in the visual field contralateral to the lesion, even for cells with bilateral receptive fields. This difference in response strength was especially evident when stimuli were moved across the midline: most cells increased their rate of discharge as the stimulus crossed the vertical meridian into the intact ipsilateral field. For 70 cells responsive in both visual hemifields, we compared responses in each hemifield by alternately testing each side with identical visual stimuli. As summarized in Table 4, for most (77%) cells responses elicited from the part of the receptive field contralateral to the striate cortex lesion were weaker than responses elicited from the ipsilateral part of the receptive field. Some (17%) cells were equally responsive across their bilateral receptive fields, and only a few (6%) were more responsive in the contralateral part of their receptive field. These proportions following striate cortex lesions differ significantly from those found in the intact monkey (chi-square = 161.2, df = 2, \( P < 0.001 \)). Most (63%) STP cells in the intact monkey were equally responsive across their bilateral receptive fields, some (34%) were more responsive in the contralateral part of their receptive field, and very few (4%) were more responsive in the ipsilateral part.

### Table 4. Most responsive zone of bilateral receptive fields, percent

<table>
<thead>
<tr>
<th>Lesion</th>
<th>Equal</th>
<th>Contra</th>
<th>Ipsi</th>
<th>No. Cells</th>
</tr>
</thead>
<tbody>
<tr>
<td>None*</td>
<td>63</td>
<td>34</td>
<td>4</td>
<td>205</td>
</tr>
<tr>
<td>Striate cortex</td>
<td>17</td>
<td>6</td>
<td>77</td>
<td>70</td>
</tr>
<tr>
<td>Superior colliculus</td>
<td>56</td>
<td>33</td>
<td>11</td>
<td>71</td>
</tr>
<tr>
<td>Both</td>
<td>0</td>
<td>0</td>
<td>100</td>
<td>1</td>
</tr>
</tbody>
</table>

* From Bruce, Desimone, and Gross, 1982(6).

VISUAL RESPONSE SELECTIVITY. Striate lesions had a dramatic effect on the selectivity of visual responses of STP neurons. In the intact monkey about half of the cells in STP were nonselective, but the other half had preferences either for direction of type of stimulus movement or, more rarely, for stimulus form (6). Such selectivity was almost completely absent in the hemifield contralateral to the striate cortex lesions. Most telling were cells that exhibited either motion or stimulus selectivity in the intact ipsilateral visual field. Such cells either did not respond to visual stimuli in the contralateral field, or responded in a nonselective and weak manner. Of 41 cells selective for direction of stimulus movement in the ipsilateral hemifield, only two were also selective in the contralateral hemifield. Figure 6 shows responses of a cell selective for rotational motion in the ipsilateral hemifield; the cell had sustained excitation for one direction of rotation and was strongly suppressed by the opposite direction. In the intact monkey such selectivity, if present, was always found throughout the visual field. In contrast, there was no response to any direction of rotation in the hemifield contralateral to the striate lesion. The only visually elicited activity in this hemifield was a weak, transient response to the initial appearance of a visual stimulus.

Specificity for stimulus form was also strongly affected by striate lesions. No cell was selective for stimulus form in the visual hemifield contralateral to the striate lesion, including 55 cells that responded best to particular stimulus forms in the ipsilateral visual field. As in previous studies, the majority of these preferences were for complex forms such as faces rather than simple stimulus properties like edge orientation. Unlike in other studies (e.g., 6, 14, 40), few of these selective neurons were analyzed to determine which features of the preferred stimuli were critical for the responses.

Not only were no cells selective for stimulus form in the contralateral visual field, but 87% of the 52 cells with a form preference in the
ipsilateral visual field had no response at all to visual stimulation in the contralateral hemifield, leaving only 13% with bilateral receptive fields. These proportions differ significantly from that of cells lacking form selectivity (57% bilateral, 41% ipsilateral, 2% contralateral; chi-square = 33.6, df = 2, \( P < 0.001 \)). In other words, cells with form selectivity were much less likely to be responsive in the visual field contralateral to the striate lesion, in comparison with cells lacking form specificity.
Most STP cells in the intact monkey responded comparably to visual stimuli over a wide range of sizes, even to tiny (<1°) objects in the far periphery. In contrast, after striate cortex lesions most STP neurons responded either best or exclusively to large stimuli (e.g., >20° x 20°) in the visual field contralateral to the lesion. This preference for larger visual stimuli may reflect the reduction of visual response strength in the field contralateral to the striate lesion as already described, or it might also reflect a diminished visual acuity in that field (see DISCUSSION).

Effects of superior colliculus lesions
To determine if the superior colliculus was the source of the visual activity in STP that survived striate removal, we destroyed the superior colliculus ipsilateral to the striate lesion in three monkeys and then studied STP activity in the absence of both structures. We also studied the effects of unilateral superior colliculus lesions alone in three additional monkeys.

HISTOLOGICAL VERIFICATION. Examination of the stained sections confirmed that the left superior colliculus was completely destroyed in all six cases (Fig. 1). These lesions were accompanied by involvement of neighboring structures in the midbrain and thalamus. In two monkeys the pretectal area was largely destroyed. In all but one monkey there was damage to the posterior aspect of the medial pulvinar. For all six monkeys there was some damage in the inferior colliculus, the accessory optic nuclei, and the central grey beneath the superior colliculus.

EFFECTS OF COMBINED LESIONS OF STRIATE CORTEX AND THE SUPERIOR COLLICULUS. After a unilateral superior colliculus lesion was made in the same hemisphere as the striate cortex lesion, STP cells no longer responded to visual stimuli in the contralateral hemifield. Eighty percent of the cells were visually responsive (Table 2), but only to stimuli in the hemifield ipsilateral to the combined lesions (Table 3). Figure 5 illustrates typical visual receptive fields after this combined lesion. Almost every cell's contralateral receptive field border coincided with the vertical meridian, and usually there was a sharp response when a stimulus crossed the vertical meridian into the intact visual field, as illustrated in Fig. 7. The other receptive-field borders were similar to those in the intact monkey: The median ipsilateral border was 56° from the vertical meridian, and median upper border was +41°, and the median lower border was -55°.

FIG. 7. Responses of 4 superior temporal polysensory area units recorded in monkeys with combined unilateral lesions of striate cortex and superior colliculus in the same hemisphere. Histograms show the response to a stimulus swept from the contralateral into the ipsilateral visual field. All 4 units responded only after the stimulus crossed the vertical meridian into the intact visual field.
The superior colliculus, therefore, was crucial for the visually driven activity in STP that survived striate cortex removal. Only 1 of 176 STP cells responded in the contralateral visual field of the two monkeys (S8, S14) with complete unilateral lesions of both striate cortex and superior colliculus. Complete striate lesions were necessary to eliminate visual responses in the contralateral visual field as 11 of 82 cells from the monkey (S9) with striate cortex remaining in the calcarine fissure still responded in the contralateral field following the additional lesion of the superior colliculus. These responses appeared to be confined to the portion of the visual field represented in the spared cortex, although most of these 11 cells had weak responses, and it was difficult to map their receptive fields. For one cell we verified a response "window" with histograms made with computer-controlled presentations of visual stimuli. The cell responded to stimuli located between 60° and 80° from the vertical meridian, but not to more central stimuli. It should be noted that until this monkey received a collicular lesion we did not suspect that its striate lesion was incomplete.

Effects of Superior Colliculus Lesions Alone. Lesions of the superior colliculus alone had only minimal effects on visual responses in STP. Furthermore, even these small effects might be transient as we completed recordings from these monkeys within 2–3 wk after making the collicular lesions. Nearly all (97%) visual receptive fields were bilateral, and the proportions of bilateral, contralateral, and ipsilateral receptive fields (see Table 3) did not differ significantly from the proportions found in intact monkeys (chi-square = 3.3, df = 2, P > 0.1). Figure 5 shows typical bilateral receptive fields of several neurons recorded following removal of the superior colliculus ipsilateral to the recording sites. The median receptive-field borders in this group were 62° contralateral, 55° ipsilateral, 48° up, and 4° down. The corresponding borders in the intact monkey were 80, 70, 50, and 55°; therefore, receptive fields after collicular lesions were slightly smaller in all directions, with the largest decrement in the contralateral borders.

Unlike lesions of striate cortex, lesions of the superior colliculus did not render the contralateral portion of bilateral receptive fields less responsive. The proportions of cells most responsive in the different zones of the bilateral receptive fields (see Table 4) did not differ significantly from the proportions found for intact monkeys (chi-square = 5.35, df = 2, P > 0.05). However, STP cells in monkeys with superior colliculus lesions were often difficult to drive, regardless of stimulus location or modality. Furthermore, 17% of the STP cells in monkeys with colliculus lesions were unresponsive, as compared with only 2% in intact monkeys (see Table 2).

In contrast to the dramatic effects of striate cortex lesions, superior colliculus lesions did not disrupt the selectivities of STP neurons. Cells selective for stimulus form or direction of motion in the intact hemifield had the same selectivity in the hemifield contralateral to the collicular lesion. We were especially interested in movement-in-depth selectivity as some cells in the deep lamina of the superior colliculus are selective for such motion (58), much as some STP cells are. Figure 8 illustrates such a movement preference of an STP cell recorded after a lesion of the superior colliculus. This cell was selective for motion directly toward the monkey. As we found for such cells in the intact monkey (6), this movement selectivity was evident over most of the visual field, including the hemifield contralateral to the lesion.

Effects of Lesions of the Superior Colliculus on Nonvisual Responses. Because cells in the deep lamina of the monkey colliculus may have auditory and somatosensory responses in addition to visual activity (22, 26, 58), we were interested in the incidence of polymodal cells in STP following lesions of the superior colliculus. However, we found no effect of collicular lesions on the incidence of polymodal STP neurons, based on both the monkeys with collicular lesions alone and the monkeys with combined lesions of the superior colliculus and striate cortex. As in normal monkeys, over half the cells in these monkeys were responsive to somesthetic or auditory stimuli (Table 2), and such responses usually were elicited with either contralateral or ipsilateral stimulation (6).

Responses of neurons in other cortical areas. Inferior temporal cortex. The largest sample of cells outside of STP was located in the ventral bank of the superior temporal sulcus. Following striate lesions, visual receptive
fields of 48 cells in this region were mapped. Forty cells (80%) had visual responses confined to the hemifield ipsilateral to the striate lesion, but the remaining eight cells were bilateral, responding to visual stimuli in both hemifields. According to the map of Desimone and Gross (15), four of these eight cells with bilateral receptive fields were located in inferior temporal cortex, but the other four were too near the border between inferior temporal cortex and STP to be definitively assigned. All eight cells responded only to visual stimuli, as is characteristic of cells in inferior temporal cortex (14,15), and their visual receptive fields were smaller than typical STP receptive fields.

**SUPERIOR COLLICULUS.** In three monkeys with lesions of striate cortex, visual responses in the superior colliculus ipsilateral to the striate lesion were studied in the course of locating the colliculus for radiofrequency lesions (see METHODS). These responses are of interest because the superior colliculus was responsible for the visual responses in STP that survived striate lesions. In all three monkeys superficial collicular cells were easily driven by visual stimuli and had precise visual receptive fields. In two monkeys (S8 and S9), we studied activity in deeper laminae as well. In both monkeys responses to visual stimulation of the hemifield contralateral to the striate lesion were observed at recording sites as far as 3 mm below the collicular surface. Although the subsequent radiofrequency lesions precluded histological verification that these sites were...
in deeper lamina, the large visual receptive fields and the presence of nonvisual responses, both somesthetic and auditory, are physiological indicators that these recordings were from the deeper layers of the superior colliculus (e.g., 22, 26, 58).

**Behavioral observations**

Three monkeys were tested for orientation toward visual stimuli in the fields contralateral and ipsilateral to their lesions. They sat in a monkey chair without head restraint, facing a large black screen with a small hole in the center. An observer hid behind the screen and engaged the monkey's attention by showing small objects or pieces of fruit in the hole. When he signaled that the monkey was fixating the hole, another person moved a white card (5-cm square) through the visual field along a circular perimeter (57-cm radius), beginning in the far periphery and moving toward the fixation point. The observer recorded the eccentricity of the stimulus' leading edge at the moment the monkey broke fixation to look at it. Each session had 20 tests of each visual hemifield interspersed in a pseudorandom order; however, trials were discarded if the monkey did not first orient toward the target after breaking fixation.

Table 5 summarizes representative sessions of a monkey with a complete lesion of striate cortex (S12) and a monkey with a complete lesion of the superior colliculus (S10). Both monkeys consistently oriented, with eye and head movements, to stimuli in either visual hemifield. Therefore these results confirm previous reports that monkeys with striate cortex lesions orient to stimuli in their hemianopic field and indicate that our recording subjects had at least some visual function in that hemifield.

Although these two monkeys consistently oriented to targets in both hemifields, there was a quantitative effect of the lesions in that both monkeys oriented at significantly smaller eccentricities in the visual hemifield contralateral to their lesion in comparison with orientation in the ipsilateral hemifield. The monkey with a complete striate lesion oriented to targets in the contralateral field at a median eccentricity of 50°, whereas it oriented to targets in the ipsilateral field at a median eccentricity of 88°. This difference was highly significant by a rank-sum test \([U(20,20) = 43, P < 0.001,\) one tailed\]. The monkey with the superior colliculus lesion showed a similar, although smaller, effect: It oriented to stimuli in the contralateral field at 74° and to stimuli in the ipsilateral field at 88°. Again, the two hemifields differed significantly by a rank-sum test \([U(15,14) = 28, P < 0.001,\) one tailed\]. The monkey with the striate lesion was tested 55 days after receiving his lesion. The monkey with the collicular lesion was tested 5 days after the lesion was made.

We tested a third monkey (S9) 80 days after an incomplete left striate cortex lesion was made, and retested this monkey 5 days after the ipsilateral superior colliculus was destroyed. After the incomplete striate lesion the monkey oriented at similar eccentricities in both visual hemifields. After the additional lesion of the ipsilateral superior colliculus the monkey often failed to detect the target being swept through the affected hemifield, but on other trials he oriented to locations in the contralateral periphery. As described earlier, the spared striate cortex in this monkey represented the peripheral part of the visual field.

**DISCUSSION**

The present study demonstrates that STP, the polysensory cortex in the fundus and upper bank of the superior temporal sulcus of the macaque monkey, receives visual inputs ultimately deriving from both striate cortex and the superior colliculus. Because neither striate cortex nor superior colliculus lesions alone abolished visual responses of STP cells, but combined lesions did, we conclude that both of these structures contribute to visual responses in STP. We consider below the possible anatomical basis of the visual responsiveness in STP that survives striate lesions and the relationship of our results to the phenomenon of "blindsight" and the concept of "two visual systems."

<table>
<thead>
<tr>
<th>Table 5. Median visual orientation, degrees eccentricity</th>
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<tr>
<td>Left Visual Field</td>
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<td>Monkey S12: Left striate lesion</td>
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<td>Monkey S10: Left sup col lesion</td>
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Anatomical basis of visual responses in STP

Although neither striate cortex nor striate-recipient areas, such as V2, project directly to STP, indirect pathways from striate cortex to STP have been described. Jones and Powell (27) originally described a sequence of corticocortical projections originating in striate cortex and culminating in a projection into STP from neighboring inferior temporal cortex. However, this is only one of several cortical routes over which visual information could reach STP. For example, posterior parietal cortex, which has visual activity related to spatial orientation (45, 61), projects to STP (27, 53). We have injected STP with horse-radish peroxidase (HRP) (unpublished data) and identified additional cortical regions projecting to STP: dorsolateral prefrontal cortex, the cingulate gyrus, the parahippocampal gyrus, and area MST in the superior temporal sulcus posterior to STP. There is evidence that cells in each of these cortical areas are visually responsive, and most appear to be polymodal like STP (3, 7, 15, 16, 45).

Similarly, although the superior colliculus does not project directly to STP (or to any cortex), there are several multisynaptic routes from the colliculus to STP. Cells in deep collicular laminae project to the medial pulvinar nucleus (4, 5, 24), which in turn projects to STP (9). Our HRP injections of STP confirm the connection between STP and the medial pulvinar and show additional thalamic inputs from the suprageniculate nucleus and the magnocellular division of the medial geniculate. Like the medial pulvinar, these thalamic nuclei also receive projections from deep lamina of the superior colliculus (4, 5, 24).

Additional routes that would allow visual inputs from the superior colliculus to reach STP could involve the lateral and inferior divisions of the pulvinar, which receive projections from both the superficial and deep superior colliculus (4, 5, 24). The inferior and lateral divisions of the pulvinar in turn project to extrastriate cortical areas which could influence STP over both direct and indirect corticocortical pathways, as discussed above with respect to geniculostriate influences. The deep colliculus also projects to the lateral aspect of the mediodorsal nucleus (24), which projects to regions of prefrontal cortex projecting to STP, as also discussed above.

It is unclear which of these indirect pathways from the superior colliculus to STP remain physiologically viable following striate cortex lesions, and thus are capable providing STP with visual inputs independent of the geniculostriate system. For example, Schiller and his colleagues (50) recorded in the superior colliculus after ablating or cooling striate cortex and found that, although visual responses in the superficial laminae remained, visual responses in the deeper laminae were abolished. This argues against pathways involving thalamic targets of the deep colliculus. On the other hand, we observed cells in the deep superior colliculus with visual activity that survived lesions of ipsilateral striate cortex, as did Marrocco (33). Furthermore, the ability of monkeys to make accurate saccadic eye movements to visual targets in the absence of striate cortex (21, 36, 52) strongly suggests that visual information from the retinal map in the superficial layers of the colliculus (12) must somehow reach the oculomotor map in the deeper collicular laminae (44).

The fate of visual activity in the thalamus following lesions of visual cortex is largely unknown. Visual activity in the lateral pulvinar is substantially reduced by striate cortex lesions; however, ~15% of the cells remain visually responsive (2, 8). Unfortunately, activity in other thalamic nuclei, including the medial pulvinar, has not been studied following damage to the geniculostriate system.

Visual activity in two extrastriate areas, namely V2 (49) and inferior temporal cortex (46), appears to depend critically on striate cortex, and thus these areas could not relay the visual activity in STP that survives lesions of striate cortex. However, it was recently found that some visual activity in extrastriate area MT survives striate removal (47). Interestingly, MT projects to area MST (56), which in turn projects to STP.

Comparisons with inferior temporal cortex

Although inferior temporal (IT) cortex projects to STP (27), the distinctive response properties of single cells in these two areas (14, 23) indicates that STP, for the most part, does not elaborate the visual processing found in IT cortex. The present study provides additional evidence that STP is functionally different from the neighboring IT cortex. It was previously reported that IT cells do not respond to visual stimuli in the hemifield contralateral to the striate lesions (46). Conse-
quently, the visual activity in STP that survives striate lesions must derive from sources other than IT.

On the other hand, the loss of visual inputs from IT might affect the minority of STP cells that are selective for visual form. In fact, no STP cell was selective for stimulus form in the visual field contralateral to a striate cortex lesion. This observation is consistent with the possibility that form-selective cells in STP receive their visual inputs primarily from IT cortex, and that this IT input is silenced by lesions of striate cortex.

We were surprised to find, in the small population of IT cells sampled, that at least 10% responded to visual stimulation in the hemifield contralateral to the striate cortex lesions. This disagrees with the more extensive study cited above (46), which reported that virtually no visual activity in IT could be elicited from the hemianopic field. A crucial difference between the previous study and the present one concerns which part of the IT cortex was recorded: The previous study sampled IT cortex on the lateral surface of the inferior temporal gyrus, whereas the present study sampled IT units in the ventral bank of the superior temporal sulcus (see Fig. 1). Desimone and Gross (15) found that these two regions of IT cortex were different in that receptive fields of cells in the ventral bank of the superior temporal sulcus were larger than receptive fields of cells in the adjacent inferior temporal gyrus. The differing effects of striate cortex lesions on visual activity in these two regions further support this functional parcellation of IT cortex.

Two visual systems

Schneider (51) and Trevarthen (55) originally emphasized the distinction between pattern and spatial visual functions (or "focal" vs. "ambient" vision). They suggested that this dichotomy paralleled the two principal pathways from the eye to the brain: the geniculo-striate system served pattern vision, whereas the tectofugal system served spatial vision. Subsequent studies indicate a more complex relationship between function and anatomy, particularly in the primate; Ungerleider and Mishkin (57) have argued that both pattern and spatial vision are well represented in primate neocortex, with a dorsal, occipitoparietal system serving spatial vision and a ventral, occipitotemporal system serving pattern vision.

The present findings suggest an integration of the original tectal-cortical hypothesis with the two cortical systems that Ungerleider and Mishkin (57) describe. We have previously argued that neuronal response properties in STP reflect spatial visual functions (6, 23). To summarize this evidence, STP units 1) have visual receptive fields that often encompass virtually the entire visual field, 2) often respond strongly to stimuli in the periphery, and are sometimes inhibited by stimuli on the fovea, and 3) are more often selective for visual motion than for stimulus size or form. Furthermore, lesions that include STP produce impairments in spatial orientation without causing impairments on pattern-discrimination tasks (31, 41, 42). The present study shows that visual inputs from the superior colliculus ultimately reach STP, even though stimulus selectivities of STP units depend on striate cortex. Therefore, we suggest that the cortical areas primarily concerned with spatial vision, such as STP, serve to integrate tectofugal with geniculostriate information. Conversely, cortical areas primarily concerned with pattern vision, such as IT cortex, may process visual information derived more exclusively from the geniculostriate system. In other words, the superior colliculus may principally influence cortical areas having what Trevarthen characterized as tectal ambient functions as opposed to cortical-focal functions (55). Moreover, the reciprocal relationship also holds in that cortical areas with spatial functions (e.g., posterior parietal cortex, the frontal eye fields, and STP) project strongly back to the superior colliculus, specifically to the deeper layers (11, 29, 30).

Parallels between neuronal activity and behavior

The effects of lesions of striate cortex and the superior colliculus on the visual responses of STP neurons have several parallels with the effects of such lesions on the visually guided behavior of monkeys and humans. Considerable visual abilities survive removal of striate cortex in primates; these spared abilities have been collectively termed blindsight. Performance of visually guided eye and hand movements is especially immune to striate removal in monkeys (20, 21, 25, 28, 36, 52, 59, 60), and most of the monkeys' blindsight abilities also have been found in the anopic field of humans with geniculostriate damage (1, 39). The major parallel between the responses
of STP cells and visual behavior is that just as a monkey does not lose its ability to use visual stimuli after lesions of striate cortex, STP neurons do not lose their responsiveness to visual stimuli. Moreover, there are parallels between the impairments of the monkey’s visual capacities after striate removal and the abnormalities of the visual activity in STP. In both cases visual sensitivity is reduced by the lesion: Monkeys with striate lesions fail to reach for small stimuli (20), fail to make saccades to dim spots (36), and fail to detect gratings of high spatial frequencies (34). In the present study, half the STP cells did not respond in the affected hemifield, and the remaining responsive cells had weak responses in comparison with either responses in the intact monkey or responses in the intact hemifield in the monkeys with striate lesions. Furthermore, large stimuli were usually necessary or optimal for STP visual responses in monkeys with striate lesions, whereas in the intact monkey STP cells usually respond well even to very small stimuli (6). Another parallel involves form perception. Form vision is severely disturbed by striate lesions, although rudimentary pattern and color perception survive (19, 48). No STP cell was selective for particular stimulus forms or shapes in the contralateral hemifield, including cells with such specificity in the intact hemifield.

An intriguing finding of the present study was that the directional selectivities of STP neurons were severely disrupted in the hemifield contralateral to striate cortex lesions. Little direction selectivity was seen in the blind hemifield, and even cells with strong directional preferences in the intact hemifield either did not respond in the blind hemifield or responded weakly regardless of the direction of stimulus movement. Unfortunately, it is not known how much the perception of stimulus motion depends on striate cortex. Keating (28) found that monkeys with striate lesions could detect movement and even discriminate between two different angular velocities. However, it was recently discovered that moving visual targets confined to the contralateral hemifield of monkeys with unilateral striate lesions do not elicit smooth pursuit eye movements; such targets are tracked with saccadic eye movements (21). Furthermore, the monkeys make inaccurate saccades toward targets moving in the contralateral hemifield (52). As STP neurons lose their movement selectivity after striate lesions, it would be interesting to test destriate monkeys on tasks that emphasize the complex movements for which STP neurons in the intact monkey are selective, that is, motion in depth and optical flow patterns.

A further parallel between unit activity in STP and behavior is suggested by our visual perimetry testing. In the intact monkey many STP cells respond to virtually anything the monkey can see, and plotting the receptive fields of such cells is almost equivalent to measuring the monkey’s visual field. This equivalence may also apply to monkeys with lesions of striate cortex. The monkey with a complete striate cortex lesion failed to reliably orient to stimuli >50° eccentric in the hemifield contralateral to the lesion, and the median STP receptive field in monkeys with striate lesions extended 50° into this hemifield. Equivalently, intact monkeys orient to stimuli at the periphery of their visual field, which is nearly the median STP receptive field in the intact monkey (80° eccentric contralateral to the recording hemisphere, see Refs. 6 and 23).

The effects of superior colliculus lesions on STP visual activity also parallel the effects of such lesions on visual behavior. Lasting effects of collicular lesions alone on visual behavior are relatively minor, and what deficits remain often reflect inadvertent damage of neighboring structures (37, 38, 54). Likewise, we found only small effects of superior colliculus lesions on the visual responses of STP cells. However, in sharp contrast to collicular lesions alone, a superior colliculus lesion in combination with a striate cortex lesion profoundly affects both visual behavior and STP neuronal activity. After combined partial lesions of striate cortex and the superior colliculus, monkeys cannot saccade to targets located in the region of the visual field affected by both lesions (36). Likewise, monkeys with complete bilateral lesions of striate cortex and the superior colliculus cannot make visually guided arm movements (54). Similarly, we found that complete removal of striate cortex and the superior colliculus in one hemisphere renders STP neurons visually unresponsive in the contralateral hemifield.

There are two general interpretations of these parallels between the visual capabilities of monkeys with lesions of striate cortex and the superior colliculus and the visual activity of STP cells after such lesions. A strong interpretation is that STP has a role in blindsight.
A weak one is that S1P activity reflects the monkey's surviving visual abilities, but is not crucial for them. For many behaviors the weaker interpretation is more plausible. For example, it is unlikely that the large receptive weaker interpretation is more plausible. For crucial for them. For many behaviors the fields of cells in S1P could guide the accurate visually guided eye and hand movements that survive striate cortex lesions. On the other hand, the neglect syndrome caused by S1P lesions (31, 41, 42) does indicate a role in attention and orientation to visual stimuli. Because S1P remains visually active after striate lesions, it would be interesting to investigate the effects of S1P lesions on visual abilities that survive striate lesions.

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