The effect of discrete monocular retinal lesions on the representation of the visual field in the primary visual area (V1) was investigated in adult cats. Lesions were created using argon lasers, 8 d to 4½ months prior to electrophysiological recording. This produced lesion projection zones (LPZs) in V1, 1.6-9.5 mm wide, that were deprived of their normal input from one eye, but that received a normal input from the other eye. Nevertheless, at the majority of recording sites within these zones neuronal responses were elicited by stimulation of the lesioned eye, with receptive fields being displaced onto regions of retina surrounding the lesion, while receptive fields determined through stimulation of the normal eye followed the normal visuotopic organization of V1. However, neuronal responses to stimulation of the lesioned eye within the LPZs were characterized by rapid habituation and unusually low firing rates in comparison with responses to stimulation of the normal eye. Stimulation of the normal eye temporarily masked the responsiveness of neurons within the LPZ to stimulation of the lesioned eye. The proportion of neurons responsive to stimulation of the lesioned eye was higher just inside the borders of the LPZs than at the centers of these zones. However, neurons responsive to stimulation of the test eye were found up to 3.6 mm from the perimeter of the LPZs, and therefore the shifts in the visuotopic map caused by retinal lesions cannot be explained solely on the basis of the normal scatter of receptive fields and point-image size in V1. The proportion of cells responsive to stimulation of the lesioned eye was highest in the infragranular layers, and lowest in the supragranular layers. By combining a restricted lesion of one eye with laser photocoagulation of the optic disc of the other eye, the effects of deactivation of the normal eye on the lesion-induced visuotopic reorganization were also investigated. Neither chronic nor acute deactivation produced any discernible further changes in visuotopy or in the characteristics of neuronal responses to stimulation of the eye with the discrete lesions. Our findings show that the representations of the two eyes in adult visual cortex are capable of independent reorganization. These findings parallel those of work in auditory cortex, suggesting that topographic reorganization in primary sensory areas of adult cortex may be governed by similar mechanisms.

A capacity for topographic reorganization of the adult cortical somatosensory representations has been well known for over a decade. Early studies demonstrated that the interruption of inputs to the brain from a given body part resulted in a “displacement,” onto adjacent unaffected body parts, of the receptive fields of cortical cells that originally represented the deafferentated area (e.g., Franch, 1980; Rasmussen, 1982; Merzenich et al., 1983). Since then, there has been a substantial amount of research on topics such as the time course of the topographical changes induced by deafferentation (e.g., Kolak and Dobetsch, 1984; Calford and Tweedale, 1988, 1991; Kolak et al., 1994), the extent of the reorganization in the cortex (e.g., Rasmussen et al., 1985; Pons et al., 1991; Lund et al., 1994), the pathways involved in the generation of reorganized maps (e.g., Rasmussen and Nance, 1991; Rasmussen, 1988; Calford and Tweedale, 1990; Nicolelis et al., 1993), and the behavioral significance of the cortical reorganization (Jenkins et al., 1990; Ramachandran et al., 1992; Recanzone et al., 1992; Xerri et al., 1994). In contrast, demonstrations of changes in the visuotopic maps of adult visual cortex are more recent (Kaas et al., 1990; Heinen and Skavenski, 1991), and many basic questions are yet to be answered.

The major issue addressed by the present study is whether binocular lesions are a prerequisite of the reorganization of visual cortical maps. Early work on topographic reorganization in the adult brain used the visual projection onto the lateral geniculate nucleus (LGN) of the cat as a model (Bysell et al., 1980, 1981). The visual representations in the main laminae of the LGN are monocular, and a retinal lesion restricted to one eye was found to be sufficient to produce changes in the visuotopic representation. In the primary visual area (V1), however, most neurons respond to the stimulation of both eyes (Hubel and Wiesel, 1962). Perhaps for this reason, most of the studies of topographic reorganization in adult visual cortex have employed either homotopic lesions of the two retinae or a monocular lesion combined with enucleation of the other eye, to produce complete deaferentation of a portion of V1. An exception was the study of Chino and colleagues (1992), who compared the cortical maps obtained through the stimulation of one retina (containing the lesion) before and after the enucleation of the nontreated eye. It was reported that, as long as this eye was kept intact, no responses to the stimulation of the lesion-affected retina could be recorded in the region of cortex that corresponded to the visuotopic representation of the lesion (henceforth referred to as the “lesion projection zone” or LPZ). However, once the influence of the other eye was eliminated by enucleation, responses to visual stimulation became evident within a few hours. These responses were elicited by photic stimulation of retinal regions located around the edges of the lesion. The results of Chino and collaborators (1992) appear to contrast with those obtained in analogous experiments in the primary auditory cortex, where lesions of portions of one cochlea induced changes in its cortical tonotopic representation, even though the map of the other cochlea remained normal (Rajan et al., 1993). Thus, in view of the possibility that the mechanisms for reorganization differ among cortical areas, we considered it important to reassess the effects of monocular lesions of the retina on the visuotopic organization of V1. We also sought to compare directly the effects of unilateral versus bilateral deactivation. This is additionally relevant, since under many clinical conditions it is unlikely that retinal pathologies will affect the homotopic regions of the two retinae. For example, would reorganization occur in cases of industrial accidents involving lasers (Boldrey et al., 1981), in which typically only one of the eyes is affected? To address such questions, we compared the proportions of responsive cells within the LPZs in cats that received monocular lesions and cats that received monocular lesions coupled with inactivation of the opposite retina by laser photocoagulation of the optic disc.
Table 1
Summary of cases

<table>
<thead>
<tr>
<th>Case</th>
<th>Age (months)</th>
<th>Test eye</th>
<th>Retinal lesion size (degrees)</th>
<th>LPZ size (mm)</th>
<th>Recovery period (days)</th>
<th>Recording hemisphere</th>
</tr>
</thead>
<tbody>
<tr>
<td>K13C</td>
<td>9</td>
<td>right</td>
<td>5° x 10°</td>
<td>4.4 x 1.6 mm</td>
<td>13</td>
<td>left</td>
</tr>
<tr>
<td>K15C</td>
<td>10</td>
<td>right</td>
<td>10° x 13°</td>
<td>6.4 x 2.5 mm</td>
<td>13</td>
<td>left</td>
</tr>
<tr>
<td>K16C</td>
<td>7</td>
<td>right</td>
<td>15° x 13°</td>
<td>3.3 x 4.0 mm</td>
<td>27</td>
<td>right</td>
</tr>
<tr>
<td>K18C</td>
<td>8</td>
<td>left</td>
<td>5° x 7°</td>
<td></td>
<td>132</td>
<td>right</td>
</tr>
<tr>
<td>K22C</td>
<td>8</td>
<td>left</td>
<td>22° x 13°</td>
<td>16.0 x 9.5 mm</td>
<td>33</td>
<td>right</td>
</tr>
<tr>
<td>K18F</td>
<td>7</td>
<td>right</td>
<td>10° x 9°</td>
<td>3.1 x 5.8 mm</td>
<td>27</td>
<td>right</td>
</tr>
<tr>
<td>K17F</td>
<td>36</td>
<td>right</td>
<td>17° x 13°</td>
<td>5.3 x 5.6 mm</td>
<td>48</td>
<td>right</td>
</tr>
<tr>
<td>K20D</td>
<td>14</td>
<td>right</td>
<td>8° x 12°; 3° x 12°*</td>
<td>5.8 x 2.6; 7.5 x 2.5 mm*</td>
<td>21</td>
<td>left</td>
</tr>
<tr>
<td>K24H</td>
<td>24</td>
<td>left</td>
<td>15° x 12°</td>
<td>10.5 x 5.3 mm</td>
<td>103</td>
<td>left</td>
</tr>
<tr>
<td>K19D</td>
<td>13</td>
<td>left</td>
<td>8° x 13°</td>
<td>6.4 x 5.8 mm</td>
<td>8</td>
<td>right</td>
</tr>
<tr>
<td>K21D</td>
<td>16</td>
<td>left</td>
<td>12° x 15°</td>
<td>9.1 x 8.4 mm</td>
<td>33</td>
<td>right</td>
</tr>
<tr>
<td>K21H</td>
<td>16</td>
<td>left</td>
<td>12° x 20°</td>
<td>11.1 x 6.6 mm</td>
<td>33</td>
<td>left</td>
</tr>
</tbody>
</table>

* Lesion projection zone.
* Insufficient data were obtained to establish fully or to estimate the borders of the lesion projection zone.
* Cases in which the normal eye had the optic disc photocoagulated during the recording session.
* Parts of two lesions were represented in the experimental hemisphere in this case.
* Cases in which one of the eyes had the optic disc photocoagulated at the same time as the retina of the other eye received a laser lesion.

Materials and Methods
The chronic effects of retinal laser lesions on the visual topography of V1 were evaluated in 10 adult domestic cats (Table 1). All animals were treated with laser lesions of restricted portions of the retina of one eye (henceforth referred to as the "test eye"). In six cases the other eye (the "normal eye") was left intact, and in four it was deactivated by high-intensity laser photocoagulation of the optic disc and surrounding retina.

Retinal Lesions and Optic Disc Photocoagulations
Lesions were created using an argon laser adapted to operate with the animal held in the stereotaxic frame (LasTek, Adelaide). Cats were anesthetized with ketamine (30 mg/kg, i.m.) and xylazine (3 mg/kg, i.m.). Atropine (0.1 mg/kg, i.m.) was given to reduce tracheobronchial secretions, and ophthalmic atropine eye drops were used to dilate the pupils. Imaging of the retina was achieved by the fitting of a planoconcave contact lens (back central optic radius, 8.6 mm) to the cornea. In all cases a spot size of approximately 300 μm with an intensity of 500–700 mW was used. In each animal two lesions were created, one in the nasal and one in the temporal hemiretina. In some experiments the effects of deactivation of the other eye on the responsiveness of neurons within the LPZ were assessed. Deactivation was achieved by repeatedly applying a laser beam (1 W) to the optic disc and the surrounding retinal area (Rosa et al., 1995). In two animals deactivation of the normal eye occurred during the electrophysiological recording session, following a preliminary exploration of the LPZ. In a further two animals the deactivation of the normal eye was performed in the same session as the creation of the discrete laser lesions of the test eye, thus allowing survival times of 8 and 33 d, during which the only visual input came from the eye with a retinal lesion. Application of a laser around and within the optic disc is less traumatic than surgical enucleation, and can be carried out during a recording session without disturbing the recording apparatus or changing the anesthetic conditions (Rosa et al., 1995). Apart from damaging the retina and severing nerve fibers, this procedure closed down all retinal blood vessels, compromising the supply of oxygen to the inner retina. Following optic disc photocoagulation, no neural responses could be elicited in V1 by stimulation of the deactivated eye, and no visual cortical evoked potential (VEP) to a flash stimulus could be elicited (Fig. 1).

Animal Preparation and Maintenance
On the day of recording the cat was anesthetized as described above, given dexamethasone (0.3 mg/kg, i.m.) and atropine (0.1 mg/kg, i.m.), and intubated with a cuffed pediatric endotracheal tube. Additional doses of anesthetic (ketamine 20 mg/kg, i.m.) were given as needed. Dental acrylic was used to attach a stainless steel rod to the frontal region of the skull; this was used to support the head during the recording session. A craniotomy was made over both left and right occipital cortices, the dura mater was removed, and the brain was covered with silicone oil. Throughout the electrophysiological recording session anesthesia was maintained with intravenous injection of thiopentone sodium (2–4 mg/kg/hr), and eye movements were minimized by the intravenous injection of pancuronium bromide (Pavulon, 0.15 mg/kg/hr), diluted in a solution consisting of saline (2.6 ml/kg/hr), dexamethasone (0.2 mg/kg/hr), and glucose (0.05 mg/kg/hr). The cat was artificially respired with a gaseous mixture of nitrous oxide, oxygen, and carbon dioxide (70:28.5:1.5%). Peak expired CO₂ was monitored, and maintained at 3.8–4.2% by adjusting the stroke volume and rate of the ventilator. In control experiments with nonparalyzed animals, this regime proved adequate to maintain the animal anesthetized for the duration of the recording session (24–48 hr). The electrocardiogram was monitored, and the heart rate was maintained below 150 beats/min by adjusting the rate of anesthetic infusion. The anesthesia was considered adequate if
were no changes in the electrocardiographic trace in response to noxious stimuli (pinches to the paw) and if the spontaneous activity of V1 cells was low. Atropine sulphate (1%) and phenylephrine hydrochloride (10%) were applied topically to dilate the pupils, to block accommodation, and to retract the nictitating membranes. Following cycloplegia, retinoscopy was performed and the eyes focused in three experiments with monocular lesions (K16, K18, and K23), the chronic inactivation of the opposite eye forced us to use published maps of the normal topographic representation of V1 (Tusa et al., 1978; Luhmann et al., 1990b). Finally, in two animals (K19 and K21) the chronic inactivation of the opposite eye forced us to use published maps of the normal topographic representation of V1.

**Histological Procedures**

At the end of the electrophysiological recording sessions cats were given an overdose of sodium pentobarbitone (90 mg/kg i.v.) and perfused through the heart with 3% paraformaldehyde in 0.1 M phosphate buffer with 10% sucrose. The brain was postfixed for 24-48 hr, and cut coronally at 50 μm on a freezing microtome. Alternate sections were stained either for Nissl substance or for cytochrome oxidase, and used to locate the electrode penetrations. The microdrive readings, the positions of the microlesions, the transitions between gray and white matter and the sulci were used to determine the position of recording sites in the sections. The position of the boundary between V1 and the second visual area (V2) was determined histologically (Price, 1985) and physiologically, by considering the progression and size of receptive fields (Tusa et al., 1978). Flat reconstructions of the surface were prepared for each case by aligning the contours of layer 4 (e.g., Rosa et al., 1993), and the recording sites were projected radially onto the reconstructions. The retinae were either whole mounted and stained with cresyl violet (Stone, 1965) or embedded in plastic, cross-sectioned (2 μm), and stained with Luxol fast blue and cresyl violet.

**Correction Factors and Delimitation of the Lesion Projection Zones**

Receptive fields for both eyes were corrected for eye movements and brought into alignment based on the binocular disparities. For this analysis, the vertical and horizontal disparities of the receptive fields recorded in regions of cortex away from the LPZ were determined, and correction factors were applied to all fields recorded through the normal eye. Since the binocular disparity varies as a function of azimuth, a single correction factor based on the average disparities was not adequate (Joshua and Bishop, 1970); thus, multiple regressions were calculated to correlate the vertical and horizontal disparities to the receptive field azimuths.

The projection of the tapetal reflection onto the tangent screen allowed a delimitation of the portion of the retina that was directly affected by the laser. There was a precise correlation (within 1°-2°) between the aggregate extent of the receptive fields recorded via stimulation of the test eye and the image of the retinal lesion thus obtained (Fig. 2). The cortical extent of the LPZ was primarily defined by comparing the receptive field sequences obtained through stimulation of the normal eye with the extent of the lesion in the visual field. For example, in Figure 24, all of the recording sites in penetration 1 were considered to fall outside the LPZ. For penetration 3, recording sites D and M were considered to be just within the LPZ. In three experiments with published maps of the normal topographic representation of V1 (Tusa et al., 1978; Luhmann et al., 1990b). Finally, in two animals (K19 and K21) the chronic inactivation of the opposite eye forced us to estimate the extent of the LPZ by comparing the receptive field positions with the normal topography of V1. Receptive fields recorded from neurons in recording sites that were far from the borders of the lesions were used as reference points to align and scale the topography of V1, in these cases to the maps of Tusa et al. (1978) and Luhmann et al. (1990b). Using the criteria described above, the extent of the LPZ was drawn on the flat reconstruction of each case (e.g., Figs. 4, 7; Table 1), and this was used to estimate the spatial extent of the reorganization.

**Results**

The main findings were independent of whether the hemisphere studied was ipsilateral or contralateral to the test eye, and can be summarized as follows.

1. In cases with well circumscribed lesions, receptive fields mapped through stimulation of the test eye were not found in the portion of the retina directly affected by the laser. However, some of the neurons within the LPZ had receptive fields...
displaced to the portions of the retina surrounding the laser lesion. At the same recording sites, the receptive fields mapped through the normal eye retained the normal visual topography. Thus, the visuotopic reorganization of the map of the test retina occurred even in the presence of an unaffected representation of the other retina in V1.

The organization of the visuotopic map in the LPZ was not complete. Within this zone, cellular responses to stimulation of the test eye were less vigorous than responses to stimulation of the normal eye. In addition, in the LPZ, a significantly larger number of neurons and multiunit clusters responded only to stimulation of the normal eye, in comparison with cortex outside the LPZ.

In the following paragraphs, we will substantiate these claims by illustrating data from individual cases, followed by statistical analyses of the influence of retinal lesions on responsiveness of neurons in the LPZ. Finally, histological controls on the extent of retinal lesions and effectiveness of the photoocoagulation procedure will be presented.

**Topographic Representation**

Whether or not the other eye was inactivated, the same trends in the displacement of receptive fields of neurons in the LPZ were observed. Receptive fields mapped through the normal eye always followed the visuotopic progression typical for normal animals (Tusa et al., 1978): as recordings were made along a penetration parallel to the midline, receptive field positions moved systematically towards the periphery of the visual field. At some of these sites, the receptive fields mapped via stimulation of the test eye were displaced into portions of the visual field that were served by regions of the retina not directly affected by the laser lesion. Figure 2A illustrates five of the penetrations made in case K171, in which we studied the hemisphere ipsilateral to the test eye. In penetration 1, caudal to the LPZ, the receptive fields mapped through the two eyes were in close register, and no topographic abnormalities were observed. However, in some of the penetrations made further anteriorly (Fig. 2C, penetrations 3 and 4), distortions of the visuotopic map were observed. In some cases, the sequence of receptive fields plotted through stimulation of the test eye bowed out markedly so as to represent the perimeter of the lesion (e.g., penetration 2), while those plotted through the normal eye crossed the region of space corresponding to the lesion. In other cases, a smooth centroperipheral sequence of receptive fields mapped through the normal eye was matched by groups of receptive fields on either side of the lesion, as mapped via the test eye (e.g., Fig. 2A, penetrations 3 and 4). Finally, some penetrations yielded little evidence of topographic reorganization. For example, in penetration 5 (Fig. 2A) the recording sites corresponding to receptive fields F-K mapped through the normal eye yielded no cellular response to the test eye, although a background response could be elicited at most of these sites; only one displaced receptive field (E) was observed within the LPZ in this penetration.

The effect of the removal of input from the normal eye was investigated in four animals (e.g., Fig. 2B, C). In the case illustrated in Figure 2B (K20C), inactivation of the other eye was performed during the recording session. The preinactivation recordings (penetrations 1 and 2 of Fig. 2B are illustrated as examples) show signs of visuotopic reorganization similar to those illustrated in Figure 2A. After inactivation of the other eye, a pair of penetrations (3 and 4) was placed between penetrations 1 and 2. The neuronal receptive fields obtained in the course of these postinactivation recordings represented the same portion of the visual field, and, similar to the preinactivation condition, there were several sites at which we were unable to obtain responses to the test eye (dashes).

Figure 2C illustrates some of the data obtained in case K21. In this case, we could not establish the control situation of the visuotopic map, since the other eye was inactivated prior to the recordings. However, we found evidence for deviations from the normal topographic representation of V1 that were qualitatively similar to those observed in cats with monocular lesions or monocular lesions combined with short-term inactivation of the other eye. These included sequences of receptive fields that curved so as to follow the perimeter of the lesion (e.g., penetrations 1 and 4 in Fig. 2C) and split receptive fields (penetrations 2 and 3 in Fig. 2C). As in the cases illustrated in Figure 2A and B, there were recording sites with unresponsive neurons scattered along the penetrations.

At a number of recording sites we observed responses to more than one portion of the space around the lesion (highlighted in gray in Fig. 2A-O). Some of these "split" receptive fields were formed by a stronger, cellular response in one subfield and a weaker, background response in the other subfield (e.g., Fig. 2A, penetration 3, field E). The background subfields may represent an excitatory contribution of intracortical fibers (Luhmann et al., 1990b). However, there were some examples of cellular responses on both sides of the scotoma (e.g., Fig. 2A, fields 2H and 4K). Although "ectopic" receptive fields have been reported previously, both in young and mature cats, in adult animals they are rare (Luhmann et al., 1990b) and may require pharmacological manipulations to become more evident. In our sample, the number of split fields as a percentage of the total number of receptive fields in the LPZ varied from zero to 29% in different animals. However, if one considers only those split fields formed by clear cellular responses on both sides of the lesion, they form less than 3% of the total. Although in our sample there were no clear examples of single isolated neurons that responded to both subfields, the presence of nonoverlapping receptive fields for adjacent cells still indicates a local disturbance of the visuotopic map.

**Responsiveness of Neurons in Cortex Inside and Outside LPZs**

Within the limitations of the hand-held stimulus presentation method and qualitative evaluation of the responses, we observed that the neurons and multiunits within the LPZs had abnormal spontaneous activity and response properties. Responsive neurons in the LPZ were characterized by low firing rates to stimulation of the test eye, as compared with the normal eye. Also, responses to stimulation of the test eye tended to habituate rapidly to repetitive stimulation. For these reasons, a precise determination of the borders of the neural receptive fields obtained through stimulation of the test eye in the LPZ demanded patient study, including repeated presentations of stimuli with long intertrial intervals. Although orientation selectivity was observed at a few sites, this property was not studied systematically, as a spot stimulus of optimal size usually yielded responses that were as strong as those to a bar. The optimal stimulus size varied, with central receptive fields usually requiring smaller stimuli than peripheral ones to yield optimal neuronal responses. In addition, in animals with monocular lesions only, presentation of stimuli to the normal eye invariably masked the neuronal response to subsequent stimulation of the test eye. This masking effect lasted for a minimum of 10 sec, but for some cells the response to the test eye was masked for up to 30 sec. However,
Figure 2. Examples of electrode penetrations and receptive fields recorded in three animals. A, case K17; B, K20; C, K21. In A, the right column illustrates coronal sections through cat V1, with electrode penetrations and recording sites indicated. Recording sites are coded according to the responsiveness to the test eye: black circles indicate cellular responses, white circles "background" responses, and dashes unresponsive sites. The cytochrome-rich layer 4 is indicated in darker gray. The left and middle columns illustrate receptive fields recorded through stimulation of the normal eye and the test eye, respectively. In these panels, the area centralis is indicated by a diamond, the grid lines (light gray) indicate intervals of 10° in the visual field, the retinal lesion is indicated in dark gray, and the corresponding portion of the visual field of the normal eye in dashed outline. Receptive fields and recording sites are named alphabetically from the surface of the brain. The receptive fields of neurons are indicated in continuous outline and those plotted based on unresolved background in dotted outline. "Split" receptive fields are highlighted in gray. In C, the optic disc contralateral to the test eye was photoagulated 33 d before the recordings, and therefore that eye yielded no receptive fields. Scale bars (to the right of each section) equal to 2 mm.
B

1  normal eye  test eye

2

3 (after inactivation)

4

C

1

2

3

4

K20

K21

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the responses to stimulation of the test eye improved with time, as long as the normal eye was kept occluded and long intertrial intervals were used. The spontaneous activity of neurons within the LPZs was generally reduced in comparison with cortex located outside these zones (Rosa et al., 1995). This was particularly clear in the cases with long-term inactivation of the other eye, in which little or no spontaneous activity was observed within the LPZs.

**Cats with Discrete Monocular Lesions Only**

Comparisons between the proportion of recording sites yielding neurons responsive to stimulation of the test eye and to the normal eye, within and outside the LPZs, are illustrated in Figure 3. We analyzed data from recording sites located within 1.4 mm of the border of the LPZ separately from those located more than 1.4 mm inside this border. The cutoff value (1.4 mm) represents the radius of the area of representation of a point in the visual field in cat V1 (Albus, 1975). Thus, some of the receptive fields recorded from neurons within 1.4 mm of the perimeter of the LPZ may simply reflect the normal scatter of receptive field positions in V1. Within this region, the proportion of recording sites that yielded cellular responses to stimulation of the test eye was 70% for contralateral cases (40 single units, 37 multiunits) and 62% for ipsilateral cases (29 single units, 7 multiunits). A further 12–15% of the recording sites yielded only background activity. In comparison, outside the LPZ, 84–89% of the sites yielded single neurons or multiunits that responded to stimulation of the test eye. Thus, even within 1.4 mm of the border of the LPZs there was a significant difference between the proportions of neurons responsive to stimulation of the test eye and the normal eye ($\chi^2 = 10.0, df = 2, 0.005 < p < 0.01$ contralateral to

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Figure 3. Comparison of the proportions of recording sites yielding neurons responsive to stimulation of the test eye (left column) and the normal eye (right column), in the hemisphere contralateral (top row) and ipsilateral (bottom row) to the test eye, in cats that received monocular lesions only. In each panel, the data are shown separately for recordings outside the LPZ, less than 1.4 mm inside the perimeter of the LPZ and more than 1.4 mm from this perimeter. Black bars represent the proportion of recording sites yielding responsive neurons, gray bars represent the proportion of sites yielding responsive background recordings, and white bars the proportion of sites in which neurons were unresponsive.

Figure 4. Flat reconstructions of portions of the rostral half of cat V1, showing the size and shape of the LPZs and the recording sites in five animals. Two reconstructions are shown for case K24 (middle row), corresponding to recordings obtained before (left) and after (right) photocoagulation of the optic disc of the contralateral eye. All reconstructions are shown as if they were of a right hemisphere, as seen from the midline (arrows illustrating the rostral [r], caudal [c], dorsal [d], and ventral [v] aspects of each reconstruction are shown at the top left of each panel). Thick continuous lines indicate the borders of V1, and dashed lines indicate the fundus of the suprasplenial sulcus (ss) and the tip of the splenial sulcus (ssl). The estimates of the LPZs are shown in shades of gray: cortex less than 1.4 mm inside the estimated border of the LPZ is shown in light gray and cortex more than 1.4 mm inside this border in dark gray. Recording sites are coded according to the responsiveness of neurons to stimulation of the test eye: black circles represent cellular responses, white circles background responses, and crosses represent no responses. mw, medial wall.
the test eye; \( \chi^2 = 22.2, df = 2, p < 0.0001 \) ipsilateral to the test eye). Outside the LPZs there was no difference between the proportions of neurons responsive to the two eyes. There were fewer recording sites more than 1.4 mm inside the border of the LPZs. Although the sample is small, the data demonstrated that some neurons in this region did respond to stimulation of the test eye (Figs. 3, 4). The trend in the data suggest, however, that these neurons were scattered amongst a higher proportion of unresponsive ones (neurons at 40–50% of the recording sites located more than 1.4 mm inside the perimeter of the LPZ were completely unresponsive to the test eye). Nonetheless, clear neuronal responses to stimulation of the test eye (including five single units) were observed up to a maximum of 3.4 mm inside the LPZ (case K20C; Fig. 4).

Although a relatively high proportion of neurons within the LPZs remained responsive to stimulation of the test eye after monocular retinal lesions (Fig. 3), the responses to stimulation of this eye tended to be weaker than those to stimulation of the normal eye. Among the binocular cells and multunit located within 1.4 mm of the border of the LPZs, 58% yielded stronger responses to the normal eye in comparison with the test eye, 30% yielded responses of equal strength to both the test and the normal eye, and only 12% yielded stronger responses to the test eye (only recording sites that yielded cellular responses to stimulation of both eyes were considered in this analysis). In all animals with monocular lesions, there were only three cases that were submitted to monocular lesions (case K24I), the proportion of cellular responses before and after photocoagulation were not different within 1.4 mm of the border of the LPZ (\( \chi^2 = 0.9, df = 2, 0.6 < p < 0.7; \) Figs. 4, 5). Although the number of recordings obtained before and after photocoagulation was insufficient for statistical analysis, there was no enhancement of the neuronal responsiveness to the test eye after inactivation of the other eye (Fig. 4, middle row); while before the inactivation 6 out of 9 recording sites in this zone yielded no responses to stimulation of the test eye, after the inactivation neurons at 2 out of 12 sites responded to this eye.

Two other animals were examined 8 d (K19Q) or 33 d (K21C) after a treatment consisting of a monocular lesion combined with photocoagulation of the opposite optic disc. As shown in Figure 6, only in the contralateral hemisphere of K21 was the proportion of cellular responses in the LPZ (within 1.4 mm of the border) similar to that outside this zone. Given the scatter of receptive field positions and the larger margin of error in determining the exact border of the LPZ in animals with long-term inactivation of the other eye, it is unclear whether this result can be interpreted as a sign of a more robust reorganization after binocular inactivation, especially as there was no indication of a large proportion of responsive cells more than 1.4 mm from the border. Moreover, in no case were the neuronal responses normal; as in the cases that were treated only with monocular lesions, low fir-
Retinal Histology

The ganglion cell layer was largely undamaged; however, in some areas, there were scattered patches where the damage was more severe, and the bipolar and ganglion cell layers were also affected. There appears to have been little damage to the nerve fibers crossing the lesion. The optic disc photocoagulation was found to deactivate the eye by severing nerve fibers and coagulating blood vessels. Retinal flat mounts and cross-sections (Fig. 9B) showed that the ganglion and bipolar cells had degenerated within 8 d of the optic disc photocoagulation.

Discussion

Electrophysiological recordings were performed in V1 of adult cats following the placement of laser lesions in the retina of one eye. By stimulating this eye, we found that the receptive fields of neurons in the cortical LPZ were displaced onto regions of retina surrounding the retinal lesion, corresponding to topographic shifts of more than 3 mm parallel to the surface of V1. The topographic shifts observed in these animals were similar to those observed in animals in which the other eye was deactivated, either acutely or chronically. These results show that the topographic representation in V1 of adult cats can reorganize following monocular lesions of the retina, even in the presence of a normal representation of the other eye. At least within the tested period, many neurons in the LPZs failed to respond to the test eye, and those that did respond to this eye had low firing rates and rapidly habituated to repetitive stimulation. Thus, the results also indicate that the capacity for compensatory rearrangement of the visuotopic map in the adult cat is not extensive.

The Possible Basis for Reorganization

Any given column in V1 receives connections from many sources, including subcortical nuclei, other columns of V1 located up to 6–8 mm away, and other cortical areas (e.g., Garey and Powell, 1967; Miller et al., 1980; Gilbert and Wiesel, 1983; Raczkowski and Rosenquist, 1983; Salin et al., 1992). Therefore, there is potential for extensive reorganization of the visuotopic map without anatomical changes. For example, considering the system of intrinsic horizontal axons, any given column in cat V1 receives inputs from over 100 mm², or one-third of the surface of this area. If all these inputs were excitatory and suprathreshold, this would result in very large receptive fields in V1; based on the data of Tusa et al. (1978), some of the receptive fields centered at the horizontal meridian at 10° eccentricity would be 30° in diameter. Even larger...
receptive fields would be expected from the projections from extrastriate areas to V1. However, such large receptive fields are not normally observed in V1 (e.g., Albus, 1975; Luhmann et al., 1990b; Grieve and Sillito, 1991). Normally, the point-image size in V1 is a fraction of that expected based on its anatomical structure, a fact that could be explained based on the activity of inhibitory interneurons (e.g., Jones, 1993; Kisvárday and Eysel, 1993; Albus and Wahle, 1994) or on the assumption that many of the extrastriate long-range connections normally convey subthreshold signals.

Albus (1975) calculated that any given point of the visual field projects to a cylindrical compartment of cat V1 that is 2.5–2.8 mm in diameter. These observations illustrate the fact that the local disorder in the representation of the visual field in the cortex imposes constraints on the interpretation of the data. For example, as a step in the analysis, we adopted the concept of "lesion projection zones" that were defined on the basis of the map of the visual field as seen through the intact eye. Since most neurons in cat V1 are responsive to the stimulation of either eye, and since the optimal binocular alignment for most of these neurons corresponds to zero disparity (Pettigrew and Dreher, 1987), the design of receptive fields recorded through the non-injured and that recorded through the test eye is a valid measurement of the actual displacement that occurred as a result of the retinal lesion. Nonetheless, the LPZs are unlikely to have sharply defined boundaries, as factors such as the normal receptive field scatter in V1 may result in gradient-like borders. Crossing the borders of the LPZs and moving up to 1.4 mm towards the center of the lesion, one would find progressively fewer neurons responsive to the test eye, even if no topographic reorganization had occurred. This normal property of cortex may be sufficient to explain the results we obtained in the animals with small retinal lesions. However, our data deviate in two ways from the predictions of a model based solely on the point-image size: first, we recorded neuronal responses from sites located more than 1.4 mm inside the border of the LPZs, and second, the vertical and horizontal binocular disparities of receptive fields of many single neurons and multiunits were unusually high. The horizontal and vertical binocular disparities of receptive fields of V1 neurons do not usually exceed 3°–4° (Pettigrew and Dreher, 1987), whereas in the LPZs, disparities of more than 10° were observed (Fig. 11). We therefore conclude that within a few weeks of a retinal lesion there were already physiological signs of reorganization in the topographic map of V1.

An important question is whether this reorganization occurs in the cortex itself or whether it merely reflects changes that occur at subcortical levels. The first demonstration of reorganization of visual maps in adult animals was by Eysel and colleagues (1980, 1981), who studied the effects of photocoagulator lesions of the retina on the topographic organization of the LGN. These studies demonstrated that, after retinal lesions, the corresponding sector of the LGN was completely unresponsive. However, starting about 4 weeks after the retinal lesion, neurons in this sector became responsive to the portions of the visual field around the edge of the lesion. It was calculated that the limit of reorganization in the LGN corresponds to a circular defect 250–300 μm in diameter, measured parallel to lamina A. This translates into displacements of receptive field positions in V1 of up to 1.2 mm. The maximal receptive field displacement observed in the LGN was 5°, with lesions placed at 20° horizontal eccentricity, corresponding to about 1 mm in the topographic map of V1. Thus, even assuming that the reorganization proceeds from the entire perimeter of the LPZ to its center, the plasticity in the LGN cannot account for reorganization across more than 2 mm of cortex. Combined with the lateral spread of individual thalamocortical afferents (Humphrey et al., 1985), the reorganization within the LGN is sufficient to cause the activation of neurons across approximately 4 mm of V1. However, this model based solely on LGN reorganization and spread of geniculocortical axons predicts a very low proportion of responsive neurons 2 mm from the border of the LPZ. Thus, to account for the observed reorganization in four of the cases reported here (K17, K21, K23, K24), it is necessary to invoke the contribution of mechanisms other than the reorganization of the geniculocortical projections from laminae A and A1. Further evidence pointing to the contribution of other pathways includes the occurrence of large shifts of receptive field position in V1 within hours of a retinal detachment (Schmid et al., 1995), and the demonstration that some neurons in the representation of the monocular crescent become responsive to the ipsilateral eye within 48 hr of inactivation of the contralateral eye (Rosa et al., 1995). These survival times are too short for an explanation based on the transsferal of topographic reorganization from the LGN to the cortex.

Some of the mechanisms that can plausibly explain the long-range visuotopic reorganization in V1 require little or no anatomical change. Among these are the potentiation of previously existing synapses of excitatory connections (Hirsch and Gilbert, 1993) and the disinhibition of previously masked portions of the receptive field by a decrease in GABAergic inhibition (Dykes et al., 1984; Calford and Tweedale 1991; Jones, 1993). However, support for a mechanism based on anatomical changes was obtained by Darian-Smith and Gilbert (1994). These authors observed that 9 months after the placement of binocular lesions the horizontal axons projecting into the LPZs, mainly in the supragranular layers, had a more elaborate arborization than those projecting outside these zones. This model is attractive because the spatial extent of these axons is sufficient to cover LPZs, resulting from even very large scotomata. Yet, in light of the present results, it will be necessary to study the time course of the sprouting of new axonal branches, with emphasis on the first few weeks after generation of the retinal lesions. If sprouting of new axonal branches is the main mechanism for reorganization of the visuotopic map in V1, then one would expect a significant enrichment of the axonal branching pattern of intrinsic pyramidal neurons in the first 6 weeks posttreatment. If the anatomical changes are delayed relative to the physiologically determined topographical changes, they may represent a mechanism for consolidation of the reorganized topographical map.

Within the range of postlesion recovery times covered by our experiments, the proportion of unresponsive neurons is...
highest in the supragranular layers and lowest in the infragranular layers (Fig. 8). Similar observations were obtained in a study of the responsiveness of the peripheral representation of V1 after inactivation of the contralateral eye (Rosa et al., 1995). At present, the significance of this observation to the determination of the mechanism of topographic reorganization is not clear. Luhmann et al. (1990a) reported that the horizontal intrinsic axons in cat V1 are longest in the supragranular layers, while the data of Darian-Smith and Gilbert (1994) indicate that the horizontal connections are normally longest in the infragranular layers. If the observations of the first study were correct, our data would point away from a model based solely on the extent of horizontal intrinsic connections. If the conclusions of the second study were correct, then our observations could simply reflect the extent of the excitatory horizontal axons; in this case, one would also expect that the largest change in the proportion of responsive neurons would occur in the supragranular layers, after long survival times.

Another possibility is that the reorganized receptive fields of neurons in V1 depend on projections from other visual areas or nuclei with larger receptive fields. For example, Salin et al. (1992) demonstrated that any point in cat V1 receives projections from a sector of the second visual area (V2) that represents a region of the visual field at least three times as large as its receptive fields. Direct or indirect projections from other thalamic nuclei in which neurons have large receptive fields (such as the lateral posterior-pulvinar complex; Miller et al., 1980; Raczkowski and Rosenquist, 1983; Hutchins and Updyke, 1989; Chalupa and Abramson, 1989) also cannot be ruled out. Although under normal conditions the excitatory contribution of these pathways to the activity of V1 neurons may be minor, the response properties of neurons within the LPZs are abnormal in many ways. It is possible that once the main input via the LGN is silenced an excitatory contribution of projections from extrastriate cortical and/or extrageniculate visual thalamic nuclei to V1 becomes evident.

One last point to be considered is whether the responses we observed within the LPZs are simply a reflection of the "periphery effect" (McIlwain, 1964), a normal response property of retinal ganglion cells. The characteristics of the response of LPZ neurons argue against this. First, the periphery effect-related discharge of retinal ganglion cells is not in phase with the stimulus movement; instead, it reflects a sustained modulation of firing, and has a long decay time. Second, this effect is optimally elicited by repetitive movement of large stimuli at relatively high frequencies. These two characteristics contrast with the low spontaneous discharges and rapid response habituation of LPZ neurons, which are reflected in the long intertrial intervals that are required to optimize their responses. Third, the periphery effect is substantially weaker at the level of the LGN, in comparison with optic tract fibers. Finally, the periphery effect is abolished or substantially weakened by barbiturate anesthesia similar to that used in our experiments (McIlwain, 1964). Thus, although we cannot exclude the possibility that reorganization of retinal synaptic circuits may occur after retinal lesions, and that this may include the same neurons that normally underlie the periphery
Relation to Previous Studies

It remains to be established, however, if the quality of the neuronal responses and the proportion of responsive neurons change with longer survival times. In our experience, the responses of V1 neurons within the LPZ are abnormal up to 4 months after a retinal lesion. Given their low firing rates and rapid habituation, it was difficult for us to establish to what extent the neuronal responses to the test eye within the LPZs resembled those of normal V1 neurons in terms of selectivity to the orientation, direction, spatial frequency, contrast, and velocity of the stimuli. Addressing these questions will require experiments employing controlled stimulus presentation, and probably the averaging of a large number of stimulus presentations.

The main difference between our results and those of previous studies refers to the question of whether or not neuronal responses to stimulation of the test eye can be evoked within the LPZ if only a monocular lesion is created. While our results suggest that this is possible, Chino et al. (1992) reported a different result, namely, that with the normal eye intact, a "cortical scotoma" (i.e., a region of cortex in which there were no neuronal responses to the stimulation of the test eye) existed. After enucleation of the normal eye, the "cortical scotoma" disappeared, and neurons responding to the stimulation of the edges of the retinal lesion immediately became evident. At present, the reason for this discrepancy is unclear. However, as we have emphasized in this article, the visual responses of LPZ neurons to the photic stimulation via the test eye lacked the vigor of the normal responses of V1 neurons. These responses may be dependent on the level of maintained anesthesia, and it is possible that small differences in the protocol may lead to the impression of unresponsive neurons close to the electrode tip. Procedural differences in the presentation of visual stimuli could also provide an explanation for the different results. The results of our study and those of Chino and collaborators (1992) converge in their implication that the normal eye does inhibit or mask the cortical responsiveness to the test eye. In our experiments, we found that stimulation of the normal eye masked responsiveness to the test eye for at least 10 sec, but the responses to this eye gradually improved over the following minutes. The study of Chino et al. (1992) did not report this effect. It is possible that if stimulus presentation was rapidly interleaved between the two eyes the impression of an absence of response to stimulation of the test eye would occur. After enucleation, without the masking effect of the normal eye, these responses may have become evident. The ability of the normal eye to depress the responses of the test eye may be important in counteracting the effects of dysfunctionally high binocular disparities. Thus, the reorganized cortex seems to have the ability to use two mutually exclusive topographic maps, with the dominant (nonlesioned) eye suppressing the weaker (lesioned) eye map when both eyes are stimulated. This phenomenon may be related to the binocular occlusion of responses in V1 when stimuli are presented simultaneously to the two eyes with high binocular disparities (Pettigrew et al., 1968). In summary, we suggest that occluding the normal eye may be nearly as effective as enucleation in bringing out the responses to the test eye. One corollary of these results is that in the behaving animal the reorganized map remains to a large extent inhibited. Still, they demonstrate that the mechanism that brings about changes in receptive field position can express itself within a time frame incompatible with models based on anatomical reorganization of the cortex (in agreement with Chino et al., 1992).

Heinen and Skavenski (1991) found in macaques that 75% of cells in the LPZ responded to visual stimulation. However, the majority of these neurons had abnormal response properties. By contrast, Kaas et al. (1990),

effect (Barlow et al., 1977), it is unlikely that the reorganization we observed in V1 is merely a reflection of the large modulatory peripheries that are normally observed in receptive fields of retinal ganglion cells.

In conclusion, one may say that it is unlikely that a single mechanism can explain all the observations on visuotopic reorganization in V1 that are presently found in the literature. Particularly close to the perimeter of the LPZ, a series of mechanisms including the normal scatter of receptive field position, reorganization at subcortical levels, spread of thalamocortical afferents and corticocortical connections may contribute to the expression of visual responses to stimulation of the visual field around the edges of the retinal lesions.

Relation to Previous Studies

Our findings confirm and extend those of other studies on reorganization in V1 where both eyes received laser lesions at homologous retinal locations (Heinen and Skavenski, 1991; Gilbert and Wiesel, 1992), or one eye received a laser lesion and the other eye was enucleated (Kaas et al., 1990; Chino et al., 1992). It has been claimed that portions of the visuotopic map up to 4 mm (Kaas et al., 1990) or 2-3 mm (Darian-Smith and Gilbert, 1994) inside the LPZs may completely reorganize after survival times of 6 to 9 months. The present results demonstrate that cells with reorganized receptive fields responding to the test eye are already evident over comparable distances within the LPZs in the first weeks after retinal lesions. It remains to be established, however, if the quality of the

Figure 9. Effect of laser treatments on histology of the retinal layers. In A, a cross-section through a retinal lesion is shown. The large arrow indicates the edge of the retinal lesion. The small arrow points to a ganglion cell. B shows a cross-section of retina from an optic disc photocoagulated eye, where no surviving ganglion cells can be observed. Both sections were stained with Luxol fast blue and cresyl violet. Scale bars, 100 μm.
following monocular lesions (5–10° within 10° of area centrals) and enucleation of the other eye in cats, reported cells which responded in a normal manner in terms of receptive field size, shape, and responsiveness except that the receptive fields were displaced. Although this could suggest a difference between cat and monkey, it could also simply reflect smaller LPZs in the study of Kaas et al. (1990) as compared to that of Heinen and Skavenski (1991). Although Kaas and collaborators (1990) claim to have generated "cortical scotomas" 4–8 mm in diameter, the illustrated retinal lesions are only slightly larger than the V1 cortical receptive fields themselves (e.g., their Fig. 1). In a subsequent study from the same group, lesions of similar size were demonstrated, by histological reconstruction, to generate reorganized regions of V1 1.6–2.7 mm in diameter (Chino et al., 1992). These values contrast with the reorganized regions of approximately 5 mm in the study of Heinen and Skavenski (1991). In the present study we observed, similar to Heinen and Skavenski, weak responses to the stimulation of the test eye for most of the neurons in the LPZ, and a significant increase in the proportion of unresponsive neurons. As explained above, however, we cannot rule out the existence of a "ring" of neurons up to 1.4 mm wide inside the perimeter of LPZs, in which some neurons had nearly normal responses. In our opinion, these may to a large extent reflect the normal receptive field scatter in V1, perhaps reinforced, in cases with long survival times, by the plastic changes that occur at the level of the LGN.

Studies in other sensory systems also indicate that the topographical maps in the cortex can be modified in adult animals. Our results are consistent with findings in both the auditory (Robertson and Irvine, 1989; Rajan et al., 1993) and somatosensory (Merzenich et al., 1984; Calford and Tweedale, 1988, 1991) systems. Studies in the auditory cortex have shown that following monaural lesions of the cochlea there are shifts in the frequency selectivity of primary auditory cortex neurons to the stimulation of the affected ear (Robertson and Irvine, 1989), and that the tonotopic map for the nonlesioned ear remains normal (Rajan et al., 1993). These results suggest that reorganization in primary auditory cortex is possible in the presence of a competing, normal input. This consistency of results across the auditory and visual cortices suggests that the processes involved in topographical reorganization follow similar rules in different systems. In the somatosensory cortex, it has been shown that initially there is an expansion and displacement of neural receptive fields following peripheral deafferentation, but in the week following treatment there is a contraction back to normal receptive field sizes (Calford and Tweedale, 1988, 1990). In the present

Figure 10. A shows a cresyl violet-stained whole mount of a retinal lesion in case K13. The arrow points to a surviving ganglion cell, and several others can be seen across the lesion. Scale bar, 0.25 mm. B is the corresponding density map of alpha (large) ganglion cells in the vicinity of the retinal lesion shown in A. The perimeter of the retinal lesion shown in A is indicated by the thick outline, and some blood vessels by the thin outlines. The numbers inside each zone indicate the total number of alpha ganglion cells in that region of retina. S, superior; I, inferior; N, nasal; T, temporal. Scale bar, 0.5 mm.
study, given the difference in the firing rates of neurons in response to stimulation the test eye and to the normal eye, it was hard to ascertain whether there was a significant change of receptive field size. Yet, by adopting a "maximal response area" criterion, we could detect no clear difference between the sizes of receptive fields plotted through each eye. Gilbert and Wiesel (1992) reported that in the monkey, immediately following the creation of matched binocular lesions some displaced receptive fields of neurons recorded from sites located around the edges of the LPZs were five times normal size, but a similar study in the cat (Chino et al., 1992) reported no similar effect.

The classical "critical period," during which the cat visual cortex shows maximal capacity to compensate for the effects of manipulations such as monocular deprivation, extends approximately to the end of the fourth month of postnatal life (Olson and Freeman, 1980). However, some susceptibility to this treatment remains up to the end of the first year of life, in the extragranular layers of V1 (Daw et al., 1992). Although some of the animals we used were less than 1 year old, it is unlikely that this will have influenced the main conclusions of our study. Some of the best evidence for receptive field shifts over distances of more than 1.4 mm was obtained in animals older than 1 year (K17, K21, and K24). In addition, few responsive neurons were located in the supragranular layers, confirming our previous work (Rosa et al., 1995). Finally, it is unlikely that the age of the animals is the main reason for differences between our results and those of previous reports. The illustrations of Kaas and collaborators (1990) and Chino and collaborators (1992) suggest that most (if not all) recording sites yield "reorganized" receptive fields shortly after a retinal lesion combined with contralateral enucleation, whereas Gilbert and Wiesel (1992) and Darian-Smith and Gilbert (1994) suggest that most of the LPZ is silent until months after the generation of the lesion. Our results, showing some examples of cellular responses that cannot be attributed to the normal scatter in the cortical representation but emphasizing their relative rarity, lie between these extremes. Thus, unless all of the cases used by Chino and collaborators were much younger than ours, and those used by Gilbert and collaborators much older, the differences in results cannot be explained by a model emphasizing protracted critical periods. This is the case especially if one assumes that the shifts of receptive field position are linked to the intrinsic horizontal axons of V1, since these connections mature by the second postnatal month (Luhmann et al., 1990; Albus and Wahle, 1994).

Conclusions

The study of topographic reorganization in the visual cortex poses specific problems to the interpretation of the results, related to the relatively large point-image size and to the presence of two interlaced maps of the visual field, each representing one of the eyes. Our results demonstrate that shifts in receptive field position occur even a few weeks after retinal lesions, that monocular lesions are sufficient to promote topographic reorganization, and that this reorganization occurs over distances larger than those that could be explained based on reorganization of the geniculocortical projection. Considering that monocular scotomata such as those caused by retinal degeneration, detachment, or vascular disease affect a large proportion of the mature human population, our results suggest that plastic phenomena similar to those described here may be relatively common in human primary visual cortex.

Notes

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