THE ROYAL SOCIETY

Monocular focal retinal lesions induce short-term topographic plasticity in adult cat visual cortex

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Electrophysiological recording in primary visual cortex (V1) was performed both prior to and in the hours immediately following the creation of a discrete retinal lesion in one eye with an argon laser. Lesion projection zones (LPZs; 21–64 mm^2) were defined in the visual cortex by mapping the extent of the lesion onto the topographic representation in cortex. There was no effect on neuronal responses to the unlesioned eye or on its topographic representation. However, within hours of producing the retinal lesion, receptive fields obtained from stimulation of the lesioned eye were displaced onto areas surrounding the scotoma and were enlarged compared with the corresponding field obtained through the normal eye. The proportion of such responsive recording sites increased during the experiment such that 8–11 hours post-lesion, 56% of recording sites displayed neurons responsive to the lesioned eye. This is an equivalent proportion to that previously reported with long-term recovery (three weeks to three months). Responsive neurons were evident as far as 2.5 mm inside the border of the LPZ. The reorganization of the lesioned eye representation produced binocular disparities as great as 15°, suggesting interactions between sites in V1 up to 5.5 mm apart.

Keywords: deafferentation; area 17; striate cortex; scotoma

1. INTRODUCTION

For more than a decade, it has been known that the somatotopic map of the adult somatosensory cortex has a capacity for immediate reorganization. Changes in the receptive fields of neurons are present even in the first hours following an interruption of normal inputs to a region of somatosensory cortex (e.g. Merzenich *et al*. 1983; Kelahan & Doetsch 1984; Calford & Tweedale 1988; Turnbull & Rasmusson 1990; Byrne & Calford 1991; Calford & Tweedale 1991). However, reports of immediate topographic plasticity in adult visual cortex are limited, with most previous studies concentrating on reorganization following long recovery times after focal lesions of the retina (Kaas *et al*. 1990; Heinen & Skavenski 1991; Gilbert & Wiesel 1992; Chino *et al*. 1995; Darian-Smith & Gilbert 1995; Schmid *et al*. 1996). Previously, we have reported rapid and extensive topographic reorganization in cat primary visual cortex (V1) after induced retinal detachment (Schmid *et al*. 1995). However, less extensive, or no short-term reorganization has been reported by others using the retinal laser-lesion model. One study of monkey visual cortex reported that immediately following the creation of bilateral foveal lesions there was a silent zone in V1 (Heinen & Skavenski 1991). However, this may have been less extensive than would be predicted by the projection of the lesion. In another study, a limited degree of immediate reorganization was reported in macaque V1, following matched bilateral lesions of the retina (Gilbert & Wiesel 1992). Close to, but within the border of the cortical projection of the lesion, neurons were responsive to visual stimulation of retina surrounding the lesion and had enlarged receptive fields, with up to 1° shift in receptive field position. Neurons in the central region of the projection of the lesion were unresponsive to stimulation. In the cat, short-term reorganization of the cortical representation following monocular retinal lesions has been reported to occur only when the opposite eye is enucleated (Chino *et al*. 1992).

Since topographic plasticity in the lateral geniculate nucleus following retinal lesions is reported to be extremely limited (Eysel *et al*. 1981; Darian-Smith & Gilbert 1995) the phenomenon is considered to be generated in the cortex. This interpretation makes it difficult to reconcile the extensive topographic plasticity seen after monocular focal detachments with the limited plasticity reported after retinal lesions. It has been suggested by Horton & Hocking (1998), that the basis of visual cortex topographic plasticity may be retinal and that it is the greater preserved integrity of neural retina in the detachment model which allows for a more extensive expression than in the laser-lesion model. Here, however, we report that monocular focal laser-lesions in adult cat induce comparable, but slightly delayed, topographic reorganization in V1 to retinal detachments in the short-term (<12 hours).

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2. MATERIALS AND METHODS

Electrophysiological recording from primary visual cortex was performed in six adult cats. Each cat was initially anaesthetized with ketamine (40 mg kg^{-1} IM) and xylazine (4 mg kg^{-1} IM) and given dexamethasone (0.3 mg kg^{-1} IM) and atropine (0.1 mg kg^{-1} IM). The trachea was cannulated to allow for artificial ventilation. The cortical surface was exposed and the head held in place by means of a stainless-steel rod attached to the frontal region of the skull. An initial loading dose of pancuronium bromide (Pavulon 0.3 mg kg^{-1} IV) was given to minimize eye movements. After induction of paralysis the cat was artificially respirated with nitrous oxide and oxygen (70:30). Throughout the electrophysiological recording session anaesthesia was maintained with halothane (0.5–1.5%). The electrocardiogram (<180 beats per min) and peak expired CO_2 (*ca.* 4%) were monitored throughout the experiment. To maintain paralysis the cat received a continuous intravenous infusion of saline (2.6 ml kg^{-1} h^{-1}), dexamethasone (0.2 mg kg^{-1} h^{-1}), glucose (0.03 mg kg^{-1} h^{-1}) and pancuronium bromide (Pavulon, 0.15 mg kg^{-1} h^{-1}).

Atropine sulphate (1–2 drops, 1%) was applied to dilate the pupils and block accommodation, while phenylephrine hydrochloride (1–2 drops, 10%) was applied to retract the nictitating membranes. Retinoscopy was performed and the eyes focused at 1 m by means of appropriate contact lenses. The optic discs and major retinal blood vessels were projected onto a tangent screen using a fibre optic light source (Pettigrew *et al*. 1979).

Monocular retinal lesions were created using an argon laser with the laser spot focused to approximately 300 μm and an intensity of 500–700 mW. Electrophysiological recording was performed in the cortical hemisphere contralateral to the eye with the retinal lesion, prior to and up to 11 h following the generation of the retinal lesion.

The region of visual cortex that represents the lesioned area of retina in its normal retinotopic map is referred to as the lesion projection zone (LPZ). After the lesion, cortex within the LPZ still received a normal input from one retina (ipsilateral to the recording hemisphere) but no input from the lesioned region of the contralateral retina. The diameter of the lesions, and consquently the LPZ, was varied between cases, as presented in table 1. The shortest axis of the LPZ varied from 3.1 mm to 6.0 mm measured parallel to the surface of the cortex. In all cases except K26, one or two recording electrodes were moved by means of a microdrive (Narishige, Japan) in order to study the same region of cortex several times. Recordings were made every 250–500 μm along the track. In the remaining case (K26) four separate recording electrodes were held in place for the duration of the experiment. Responses were classified as cellular (single-unit or multi-unit with identifiable components) or clusters (without identifiable units) as described previously (Schmid *et al*. 1995). Receptive fields were defined by threshold boundaries to the monocular presentation of high contrast bars of preferred orientation (if present). Ocular dominance, based on the response strength to each eye with the other eye masked, was rated on a seven-point scale (Hubel & Wiesel 1962). Greater detail of recording and stimulus presentation procedures have been presented previously (Rosa *et al*. 1995; Schmid *et al*. 1996).

At the end of the recording session, cats were given an overdose of sodium pentobarbitone (90 mg kg^{-1} IV), perfused with 0.9% saline, 3% paraformaldehyde in 0.1 M phosphate buffer and then with 10% sucrose included in the fixative. The brains were cut coronally at 50 μm and alternate sections processed for Nissl substance using cresyl violet or for cytochrome oxidase (Wong-Riley 1979). Retinae were either whole-mounted and stained with cresyl violet (Stone 1965) or embedded in plastic, cross-sectioned (at 2 μm) and stained with luxol fast blue and cresyl violet.

Table 1. *Summary of lesion extent for all cases*

case	age (months)	retinal size (degrees)[a]	cortical size (mm)[b]
K25	17	9 × 11	3.7 × 6.0
K26	18	12 × 17	6.0 × 7.6
K30	19	10 × 14	4.7 × 7.8
K31	20	18 × 26	5.5 × 11.6
K32	25	14 × 17	4.7 × 9.7
K33	20	16 × 11	3.1 × 6.9

[a] The retinal size is given as the horizontal then the vertical dimension of the lesion.
[b] The cortical size is an approximation based upon projection onto published maps of V1 topography (Tusa *et al*. 1978). The shortest diameter through the centre of the lesion projection zone in visual cortex and its perpendicular dimension are shown.

3. RESULTS

The parameters of the laser-lesioning procedure match those used in a previous study of long-term effects (Schmid *et al*. 1996). In sectioned retinae, direct effects of the laser heating are evident in the outer retinal layers (figure 1). The outer segments of the photoreceptors and some elements of the photoreceptor cell body layer are completely destroyed. Remaining photoreceptor cell bodies show signs of damage in that they contain darkly staining masses and their usual columnar arrangement is disrupted. It is difficult to distinguish an outer plexiform layer. Unusual darkly staining cell bodies are also evident in the inner nuclear layer (INL). Whereas these may result from direct damage, it is also expected that cells with somae in this layer and with processes that extend into the outer retina will have secondary somal damage. The cells most likely to be so affected are the Müller-glia cells. The position of these darkly staining cells at the second most outer row of the INL is consistent with that of Müller-glia cells (Dreher *et al*. 1992). Ganglion cell numbers and staining densities, best examined in whole-mounted retinae, appear unaffected (as previously reported after long-term recovery (Schmid *et al*. 1996)). The damage to outer retinal layers was also clearly evident in the whole-mounted preparations.

The main findings of this study, from recording in V1 in the hours following placement of a monocular retinal lesion, are (i) neural receptive fields determined through photic stimulation of the lesioned eye avoided the portion of the visual field directly affected by the laser and were displaced so as to represent areas of the retina surrounding the scotoma; and (ii) the reorganization of the retinotopic map in the LPZ was not complete, with less vigorous responses obtained for stimulation of the lesioned eye. Within the LPZ no neural response could be elicited through stimulation of the lesioned eye in the first hour following the creation of the lesion. In the following

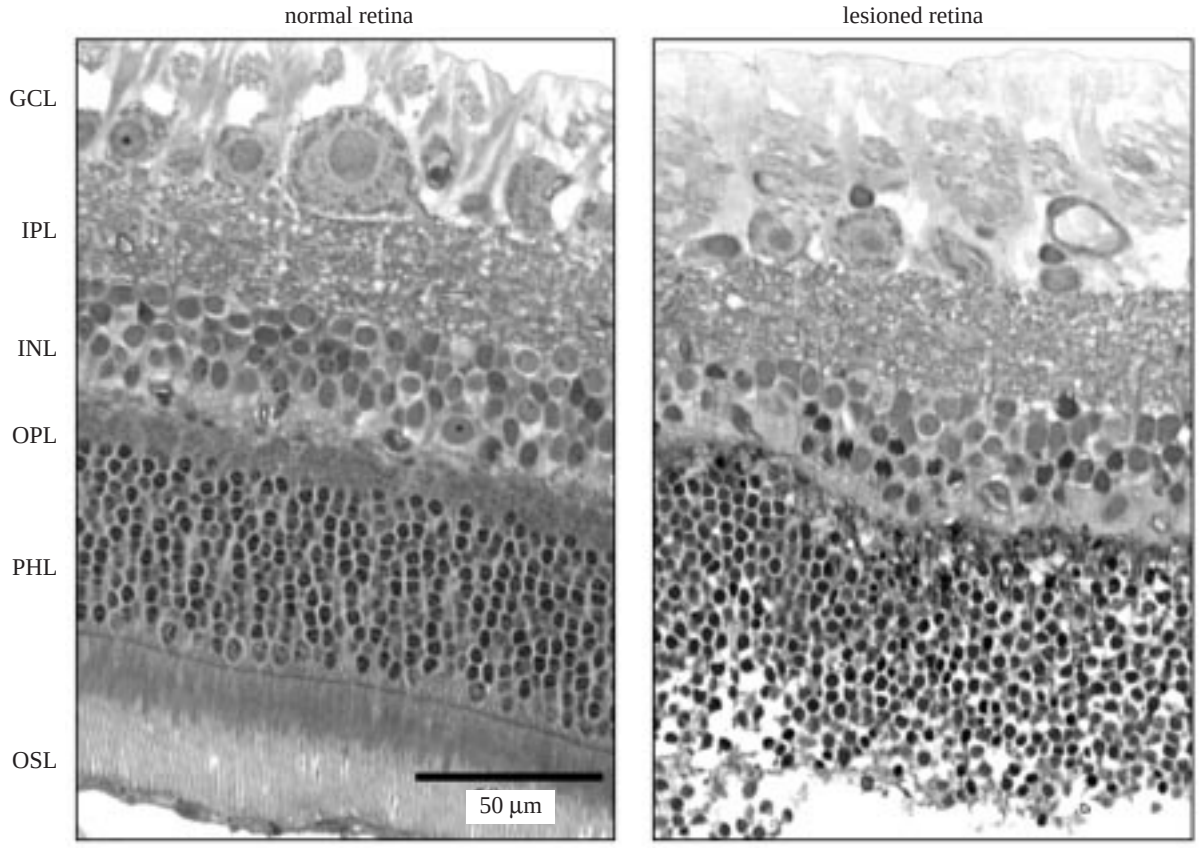

Figure 1. Micrographs of retinal cross-sections through normal and laser-lesioned retina from case K30. The retina was fixed 12 h after the creation of the lesion. Sections were 2 µm thick and stained with luxol fast blue and cresyl violet. GCL: ganglion cell layer; IPL: inner plexiform layer; INL: inner nuclear layer; OPL: outer plexiform layer; PHL: photoreceptor layer; OSL: outer segments of photoreceptor layer.

hours responses became evident. At all times the ocular dominance distribution of cortical neurons was shifted to favour the normal eye; and (iii) most of the receptive fields of neurons within the LPZ obtained from stimulation of the eye with the retinal lesion were enlarged compared to the corresponding field obtained from stimulation of the normal eye.

Figure 2 summarizes the data obtained from cat K25 in which a retinal lesion was centred at 15° eccentricity in the lower visual field. The electrode was moved throughout the track several times at the same trajectory. However, it is not claimed that recordings were made from exactly the same sites in the brain; pre- and post-lesion recordings are compared on a population rather than an individual basis. In addition, a direct comparison can be made between receptive fields obtained through stimulation of the two eyes at the same recording site. Reconstructions of multiple tracks based on recording depth showed that, for stimulation of the normal eye, the retinotopic progression was typical of that for normal animals (Tusa *et al*. 1978) and was unaffected by the lesion. Thus, as the electrode was advanced ventrally, parallel to the midline, receptive fields systematically progressed from the vertical meridian towards the periphery of the visual field. Prior to placement of the laser lesion, the neural receptive fields obtained in response to stimulation of each eye followed the same progression with the usual slight variation in binocular disparity. After placement of the lesion, receptive fields of neurons within the LPZ, which were responsive to stimulation of the lesioned eye were displaced onto normal retinal areas surrounding the lesion. At most recording sites within the LPZ cellular responses to stimulation of the lesioned eye only became apparent after a few hours: 1–3 h after creation of the lesion (figure 2*b*), neurons at six sites (out of a total of 14) were responsive to stimulation of the normal eye but showed no response to stimulation of the lesioned eye. After 4–8 h (figure 2*c*), 10 out of 12 sites sampled showed responsiveness to the lesioned eye and displaced receptive fields.

The extent of the shift in receptive field positions for neurons within the LPZ can also be presented by considering the projection of the visual field onto the surface of V1. Cellular responses with displaced receptive fields were observed up to 2.5 mm inside the zone of cortical projection of the lesion. When the displacement is considered in respect of the normal retinotopic map in V1, equivalent shifts of receptive field position of up to 5.5 mm are apparent when the position of the recording site is compared with the projection of the reorganized field (figure 3).

Prior to the creation of the retinal lesion, the sizes of cellular receptive fields obtained through stimulation of each eye were significantly correlated (slope = 1.01, $r^2 = 0.94$; figure 4). One to three hours after creation of the lesion, receptive fields of neurons within the LPZ were significantly larger when obtained through stimulation of

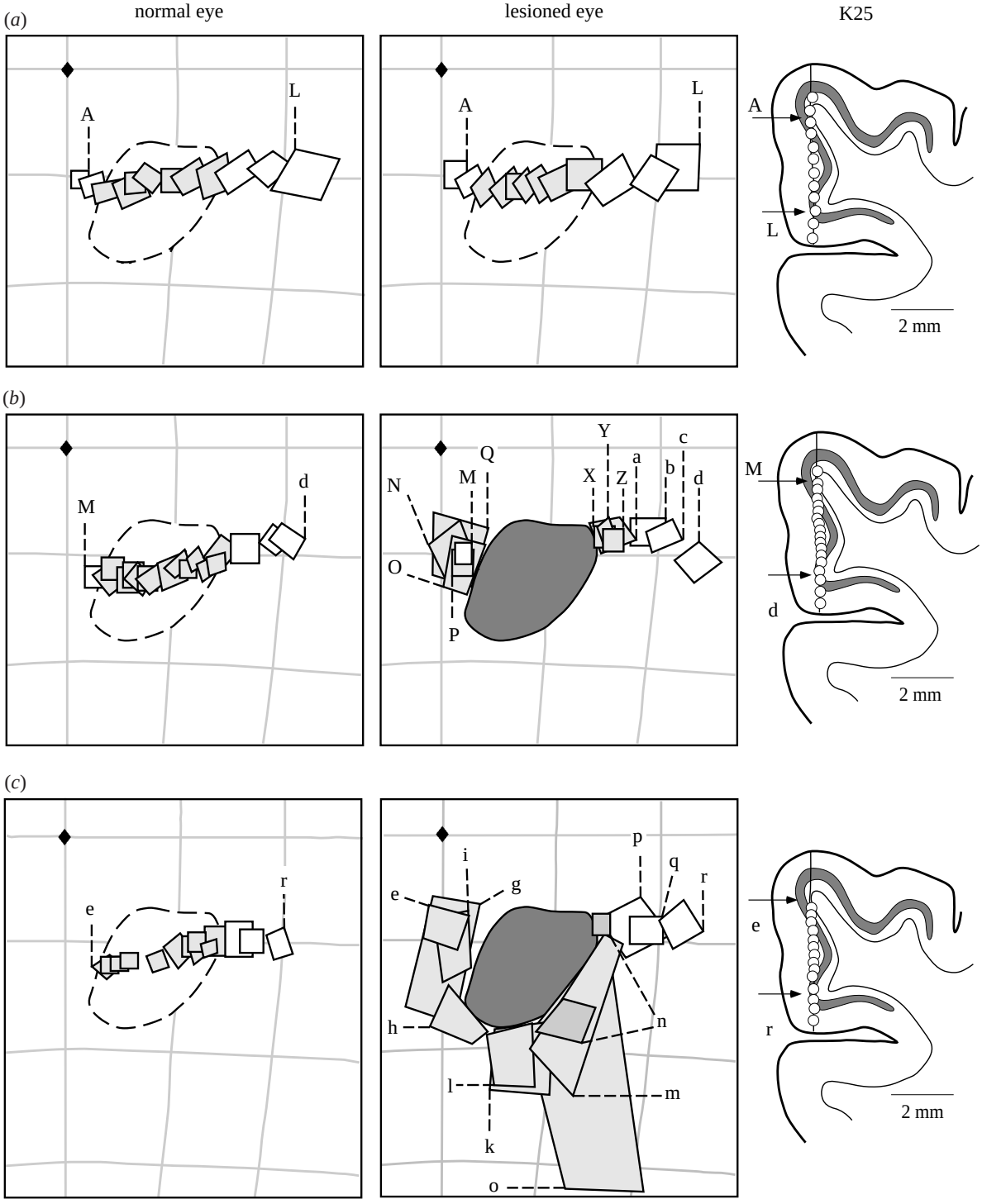

Figure 2. Neural receptive field boundaries and recording site positions in V1 from case K25. Receptive fields determined (*a*) prior to, (*b*) 1–3 h and (*c*) 4–8 h following the creation of the retinal lesion are illustrated. The receptive fields obtained from stimulation of the normal eye and the lesioned eye are shown in the left and middle columns, respectively. The background grid marks off 10°. The position of the area centralis is marked with a diamond. Recording sites are shown in the sections to the right. The arrows in the first section indicate the boundaries of the LPZ.

the lesioned eye as compared with stimulation of the normal eye (one-tailed, paired *t*-test; $t = 3.15$, $p < 0.002$). They remained significantly larger for the period of the experiment, up to 11 h (one-tailed, paired *t*-test; $t = 3.91$, $p < 0.001$). Neural receptive fields obtained from stimulation of the lesioned eye were up to five times larger (linearly) than the corresponding fields for the normal eye (figure 4).

Ocular dominance distributions of responses obtained prior to creating the retinal lesions resemble those reported by other investigators (e.g. Hubel & Wiesel 1962; Blakemore & Pettigrew 1970; Albus 1975). Most cellular and cluster responses were binocular, and the distributions on a seven-point scale were clustered around a dominance value of four, with a slight bias towards the contralateral eye. However, for recordings made after the

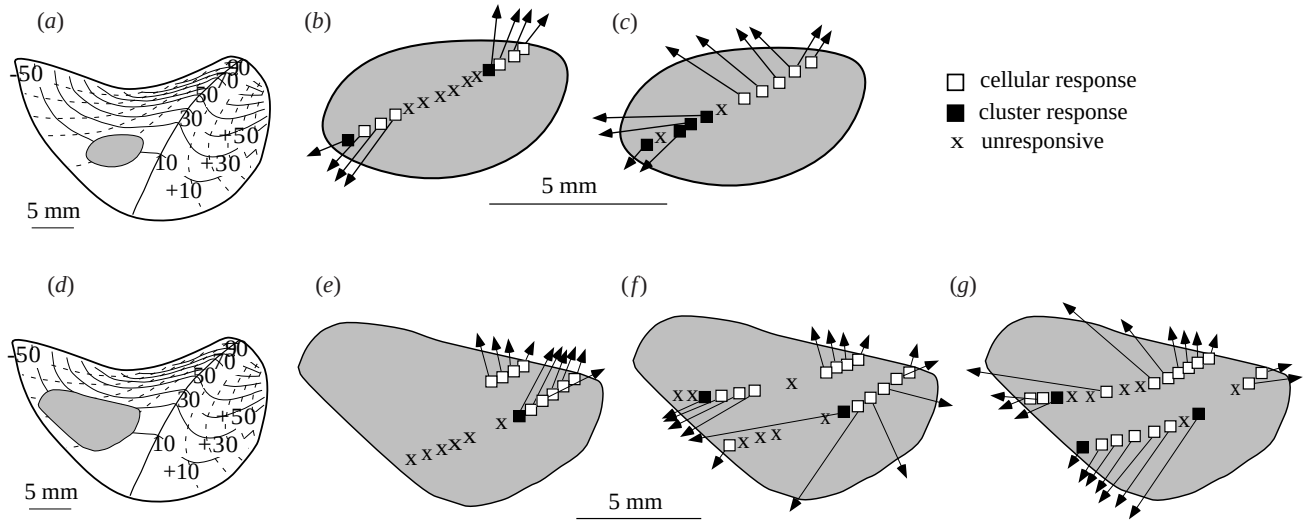

Figure 3. The extent of the LPZ was estimation by projection onto V1 using the reconstruction of Tusa *et al*. (1978). Projection of the lesion onto the V1 map for cases K25 and K31 are shown. (*a*,*d*). Recording sites examined in the periods (*b*) 1–3 h and (*c*) 4–8 h following the retinal lesion for case K25, and (*e*) 1–3 h, (*f*) 4–7 h and (*g*) 7–11 h following the retinal lesion for case K31, are illustrated with respect to the LPZ. To illustrate the extent of the reorganization in cortical dimensions, arrows extend from the centre of the receptive field obtained through stimulation of the normal eye to the centre of the receptive field determined through stimulation of the lesioned eye when these are projected onto the normal retinotopic map. Cellular responses are indicated by open squares while recording sites at which only cluster activity was obtained in response to stimulation of the lesioned eye are indicated by filled squares. Sites at which neurons remained unresponsive to stimulation of the lesioned eye are illustrated with crosses. Recording sites for which two separate receptive fields were obtained from stimulation of different units of a multi-unit pool are indicated by two arrows.

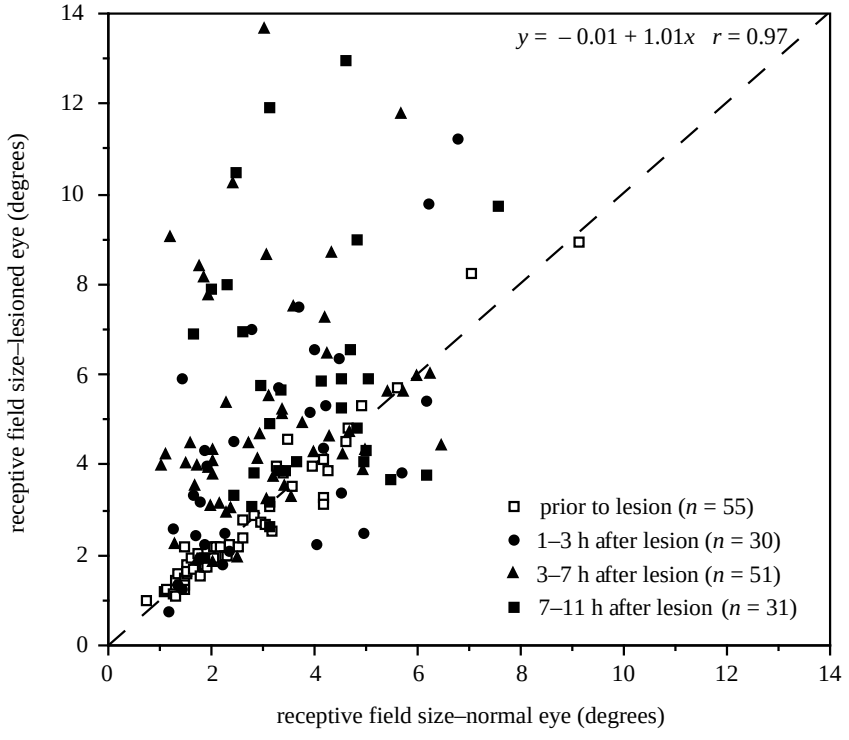

Figure 4. A plot of corresponding receptive field sizes (square-root of angular area, in degrees) for cellular responses obtained from stimulation of each eye is shown for before, 1–3 h, 3–7 h and 7–11 h after the creation of the retinal lesion, for all cases. The regression line is shown for receptive field sizes obtained prior to the retinal lesion.

laser lesion, from sites within the LPZ, there was a significant shift in the ocular dominance distribution towards the normal eye (figure 5; Mann–Whitney *U*-test, $z = 5.88$, $p < 0.0001$). The ocular dominance distribution did not change significantly with post-lesion recovery time (up to 11 h; Mann–Whitney *U*-test, $z = 0.86$, $p = 0.39$). For comparision, the ocular dominance distribution for responses from sites within the LPZ after long-term recovery, using data obtained in a previous study (Schmid *et al*. 1996), is also presented (figure 5*e*). This comparison reveals a significant shift in the distribution (Mann–Whitney *U*-test, $z = 3.68$, $p < 0.0002$), reflecting

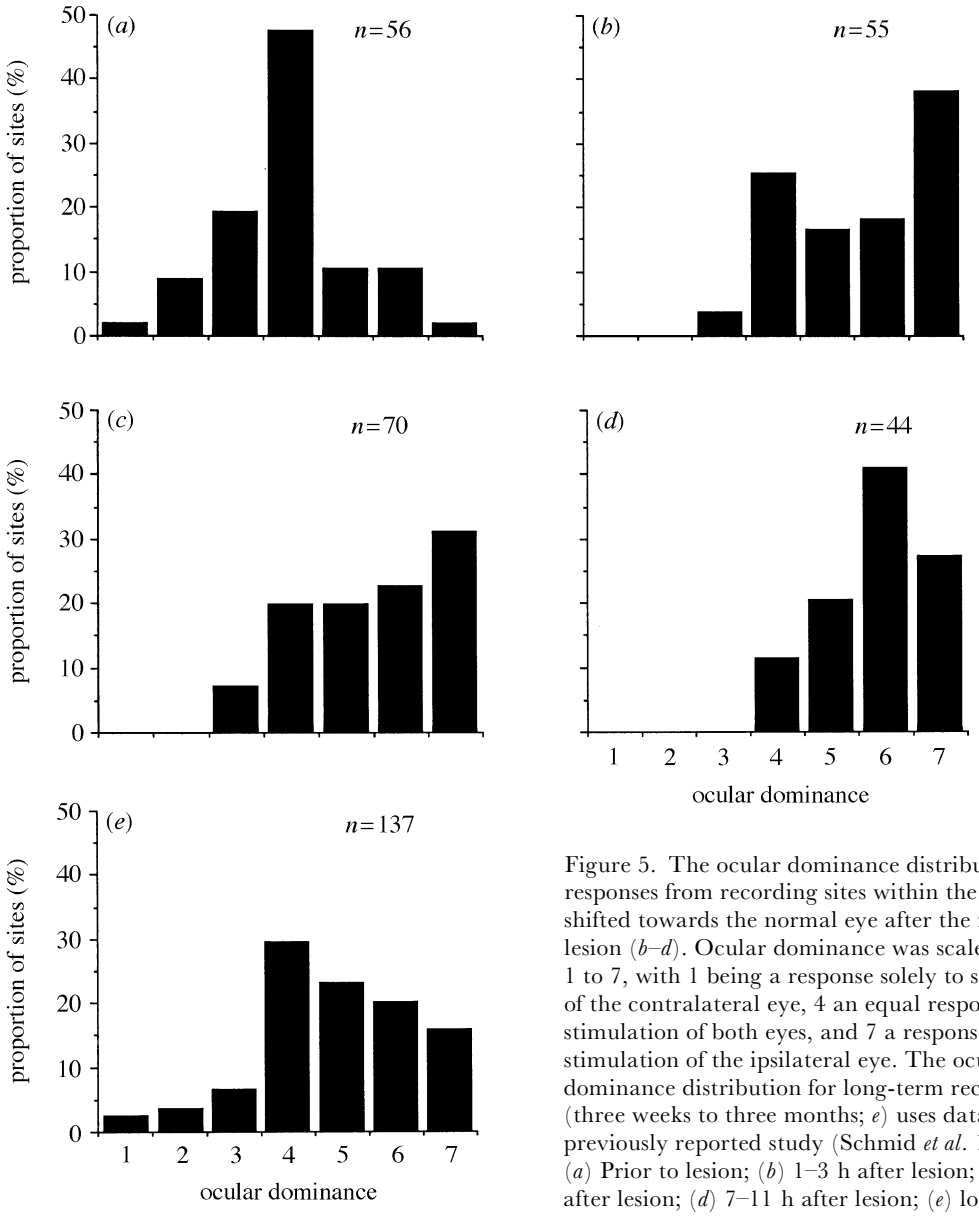

Figure 5. The ocular dominance distribution for responses from recording sites within the LPZ was shifted towards the normal eye after the retinal lesion (*b–d*). Ocular dominance was scaled from 1 to 7, with 1 being a response solely to stimulation of the contralateral eye, 4 an equal response to stimulation of both eyes, and 7 a response solely to stimulation of the ipsilateral eye. The ocular dominance distribution for long-term recovery (three weeks to three months; *e*) uses data from a previously reported study (Schmid *et al.* 1996). (*a*) Prior to lesion; (*b*) 1–3 h after lesion; (*c*) 3–7 h after lesion; (*d*) 7–11 h after lesion; (*e*) long-term.

increased relative responsivity to lesioned eye stimulation in the long-term recovery cases. For neurons outside the LPZ no difference in the ocular dominance distributions was evident prior to or following the creation of the laser lesion (Mann–Whitney *U*-test, $z=1.77$, $p=0.08$).

Since recordings were made at fixed intervals in electrode tracks (250 or 500 μm) the quality of the response encountered within the LPZ to stimulation of the lesioned eye becomes another metric of the degree of plasticity. Comparisons are presented between the proportions of recording sites yielding cellular responses, cluster activity or no response; prior to and at various times after the lesion, and also between the responses elicited through the normal and lesioned eyes (figure 6). For this analysis, sites were delineated according to the distance of the recording site from the boundary of the LPZ. Neurons at sites near the perimeter of the LPZ became responsive to photic stimulation of the lesioned eye sooner than those at sites towards the centre of the LPZ (figure 3). However, 11 h after placement of the lesion there were still some sites close to the boundary of the LPZ at which neurons were unresponsive to stimulation of the lesioned eye. To compare effects at the centre and at the periphery of the affected area a cut-off value of 1.4 mm was chosen. This corresponds to the radius of the point image size for cat V1 (Albus 1975). In the first 1–3 h after creation of the retinal lesion only 25% of recordings from sites greater than 1.4 mm from the edge of the lesion yielded a cellular response to stimulation of the lesioned eye (figure 6*b*). This increased to 60%, 3–7 h after creation of the lesion. At the completion of recording sessions, reorganization within the LPZ was not complete, with 50–60% of neurons responding to stimulation of the eye with the retinal scotoma compared with 90% for stimulation of the normal eye.

4. DISCUSSION

The results reported here indicate that the retinotopic map of V1 in adult cat can reorganize in the hours immediately following monocular retinal lesions. Reorganization takes the form of large, but defined,

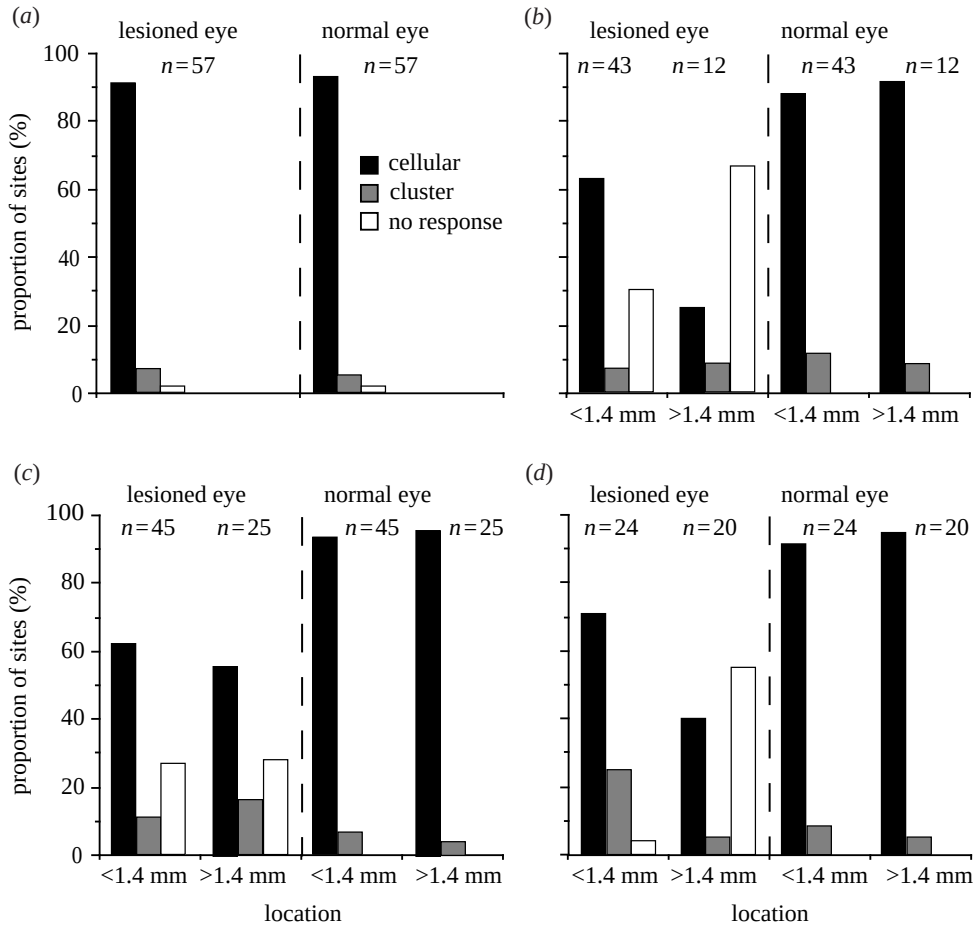

Figure 6. The positions of recording sites were classified according to position within the LPZ as determined from flattened reconstructions. Recording sites were divided into two groups, those less than 1.4 mm and those greater than 1.4 mm from the boundary of the LPZ. This was done for recording sites sampled (*a*) prior to lesioning and for post-lesion periods, (*b*) 1–3 h, (*c*) 3–7 h, (*d*) 7–11 h. Within these groups the percentages of cellular activity, cluster activity and no responses were determined for stimulation of each eye.

neural receptive fields displaced to normal retina, adjacent to the edge of the lesion. Neural receptive fields obtained in response to stimulation of the unaffected eye were of normal size and formed a normal topographic progression. Responses to stimulation within the reorganized receptive fields were generally weaker than those obtained prior to the lesion. This was judged by comparison of neural responses to the two eyes and there was a resultant shift in the distribution of ocular dominance scores to favour the normal eye. With an increase in time following the retinal lesion (0–11 h) the proportion of sites with cellular responses to stimulation of the lesioned eye increased. Towards the end of the experiments, 56% of recording sites in the LPZ revealed neurons responsive to stimulation of the eye with the retinal lesion. This compares with around 90% of cellular responses, obtained with these recording methods, to stimulation of either eye at recording sites in normal cortex.

In this study emphasis was placed on determining the time-course of the reorganized response, so extensive mapping of the LPZ was not performed. Thus, to determine the extent of the reorganization an approximation method was used: the limits of the retinal lesion and the position of individual recording sites were projected onto an 'unfolded' cortical map (Tusa *et al*. 1978). The extent of the jitter in the representation of visual space in V1 is represented as the point image size which varies little across the representation (Albus 1975). Thus, reorganization within 1.4 mm (radius of the point image) of the LPZ boundary may conceivably be a function of the normal variation of receptive field position for neurons at a given cortical location. However, the unusually large binocular disparities encountered (figures 2 and 3) provides strong evidence for reorganization, even in peripheral aspects of the LPZ. At the centre of the LPZ, neural responses to stimulation of the eye with a retinal lesion were evident as far as 2.5 mm from the nearest boundary of the LPZ and shifts of receptive field position (expected-normal to reorganized) as great as 5.5 mm across the representation in V1 were evident. These changes cannot be attributed to any normal variation in the representation in visual cortex.

For neurons within the LPZ, receptive field size was significantly larger when obtained from stimulation of the lesioned eye than with stimulation of the normal eye. While there was considerable variability, and some fields obtained through stimulation of each eye were comparable in size, receptive fields obtained from stimulation of the eye with the retinal lesion were as much as five times larger than the corresponding field obtained from stimulation of the normal eye. In cases with longer post-lesion recovery times (three weeks to three months) receptive field sizes obtained from stimulation of each eye were similar (Schmid *et al*. 1996). This result confirms a previous report of responsive neurons within the perimeter of the LPZ immediately following bilateral retinal lesions having enlarged receptive fields (Gilbert & Wiesel 1992).

Horton & Hocking (1998) have pointed out that the extent to which laser lesions affect the inner layers of retina, including the ganglion cells, is variable across

animals and in some cases across a given lesion. All retinae from both the present series, and the one previously reported for the effects of long-term recovery (Schmid *et al*. 1996), were examined either as thin sections or as whole-mounts. We have separately analysed cases with long-term recovery in which the lesion was seen to destroy the majority of ganglion cells and have presented a preliminary report (Calford *et al*. 1998). The lesions of the cases presented here were not seen to deplete the ganglion cell layer. Nevertheless, consideration of the morphological effects of the lesions (figure 1) suggests that, in affected retina, ganglion cells would have been inactive. It is known that inner retinal neurons depend totally upon adjacent Müller-glia cells for participation in the cycling of glutamate to and from glutamine (Pow & Robinson 1994). The interpretation, or expectation, that Müller-glia, which send major processes into the outer retina (end feet), would be damaged directly by (and later die as a result of) the laser heating, carries with it the conclusion that ganglion cells would become inactive.

The data presented here complement those of two previous studies, with equivalent recording methods, in which the short-term effects of monocular retinal detachments (Schmid *et al*. 1995) and the long-term effects of monocular retina lesions (Schmid *et al*. 1996) were examined. The degree of reorganization, in terms of the proportion of sites, within affected cortex, which revealed a cellular response with receptive fields displaced to unaffected retina, was equivalent for all three paradigms at 54–60%. The major difference found with the short-term lesion paradigm was that initially, the proportion of reorganized neural responses was far lower. While the increase in responsiveness is apparent in the collated data, it is instructive to examine individual cases where segments of an electrode track which were unresponsive to stimulation of the lesioned eye up to three hours after the lesion, became fully responsive after seven hours (compare lower electrode track figure 3*e*,*g*). The physiological consequences of induced retinal detachments differed from those of the present method in that the retina was unresponsive to photic stimulation (due to separation from the retinal pigment epithelium) but was intact and inherently viable. It is difficult to conceive of central consequences that would differ between the two paradigms over this time period. Thus, a retinal phenomenon must be sought for explanation of the delayed cortical reorganization after direct retinal lesioning. The most parsimonious explanation is that placement of the lesion temporarily affected the responsiveness of a region of retina surrounding the lesion. Simple light- or heat-induced pigment bleaching would be expected to play such a role and may explain the first period of total unresponsiveness. Subsequently, nuclear reactivity in regions surrounding the lesion may play a role in transient inactivity. Such areas show rapid expression of biochemical reactivity in a number of cell classes including photoreceptors, Müller cells and ganglion cells (Tassignon *et al*. 1991; Yamamoto *et al*. 1996; Humphrey *et al*. 1997; Chu *et al*. 1998). The degree to which surrounding retina is so affected would be expected to depend upon the intensity, wavelength, and dwell-time of the laser application—potentially accounting for some of the variability between previous reports (Heinen & Skavenski 1991; Chino *et al*. 1992; Gilbert & Wiesel 1992).

Darian-Smith & Gilbert (1994) have reported that axons of intrinsically projecting cells, entering the LPZ, undergo terminal sprouting in the months following binocular retinal lesions. The rapid occurrence of the topographic plasticity demonstrated in the present study suggests that such sprouting plays a role in consolidation of the reorganization (e.g. receptive field size and ocular dominance effects) rather than its initial expression. The fact that the terminal sprouting occurred within the geometric limits of normal intrinsic connections within V1 is consistent with this view. The rapidity of the reorganization seen in the present study requires existing viable circuits providing considerable cross-connectivity within the topographic representation. Since studies of the effects of retinal deactivation have failed to show reorganization of this scale in retina (Levick & Thibos 1993) or the dorsal lateral geniculate nucleus (Eysel *et al*. 1981; Darian-Smith & Gilbert 1995), it is most likely that the source of this cross-connectivity is existing intrinsic projections within cortex (Gilbert & Wiesel 1979, 1989; Luhmann *et al*. 1990*a*,*b*).

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