# Responsiveness of cat area 17 after monocular inactivation: limitation of topographic plasticity in adult cortex

Marcello G. P. Rosa, Leisa M. Schmid and Michael B. Calford

*Vision, Touch and Hearing Research Centre, Department of Physiology and Pharmacology, The University of Queensland, QLD 4072, Australia*

1. Recordings were made from neurones in the splenial sulcus of normal adult cats and adult cats which had one eye inactivated by enucleation or photocoagulation of the optic disc. Two visually responsive regions were observed, corresponding to the peripheral representation of visual area 1 (V1) and the splenial visual area. In normal animals, responses to the ipsilateral eye in V1 were restricted to the medial half of the splenial sulcus, up to 45–50 deg eccentricity. Thus, by inactivating the eye contralateral to the experimental hemisphere, we created a region in V1, 1–2 mm wide, that lacked normal inputs.

2. In contrast to results from previous experiments where lesions were placed in the central retina, neurones in the deprived peripheral representation remained unresponsive to light stimuli for up to 12 h after deactivation of the contralateral eye.

3. In animals that were allowed to recover from the monocular deactivation for periods of 2 days to 16 months, there was rearrangement of the retinotopic maps. Receptive fields in regions of cortex that normally represented the monocular crescent were displaced to the temporal border of the binocular field of vision. However, most neurones in the deprived peripheral representation remained unresponsive to visual stimuli even more than 1 year after treatment. This is also in marked contrast with the extensive reorganization that is observed in the central representation of V1 after restricted retinal lesions. Analysis of the cortical magnification factor demonstrates that the change in visual topography is local, and does not involve an overall centro-peripheral shift of the retinotopic map.

4. Among the neurones that did show displaced receptive fields, the response properties were clearly abnormal. They showed a notable lack of spontaneous activity, low firing rates and rapid habituation to repeated stimulation.

5. The low potential for reorganization of the monocular sector of V1 demonstrates that the capacity for plasticity of mature sensory representations varies with location in cortex. Even relatively small pieces of cortex, such as the monocular crescent representations, may not reorganize completely if certain conditions are not met. These results suggest the existence of natural boundaries that may limit the process of reorganization of sensory representations.

The topographical maps that exist in adult sensory cortex have a considerable plastic capacity. This was first observed in the somatosensory cortex, where immediate and chronic changes of receptive field size and location were described following amputation (e.g. Rasmusson, 1982; Merzenich, Nelson, Stryker, Cynader, Schoppmann & Zook, 1984; Calford & Tweedale, 1988), peripheral nerve section (e.g. Merzenich, Kaas, Wall, Sur, Nelson & Felleman, 1983) and training to perform tasks that put demand on specific body parts (Recanzone, Merzenich, Jenkins, Grajski & Dinse, 1992). Long-term changes were observed in primary auditory cortex as a result of cochlear lesions (Rajan, Irvine, Wise & Heil, 1993), and immediate changes were demonstrated in the same area as a result of learning (Weinberger, Hopkins & Diamond, 1984).

The visuotopic representations in the adult visual system are also plastic. The initial demonstration of this phenomenon was by Eysel and his colleagues, who used a xenon photocoagulator to generate lesions in circumscribed portions of retina and recorded visual responses in the lateral geniculate nucleus (LGN). In this structure, no evidence was found for immediate reorganization of the visuotopic map. Nonetheless, after a survival time of

1 month, neurones in a portion of the LGN that had been deactivated by destruction of the visuotopically matching region of the retina became responsive to stimulation of regions of the retina around the lesion (Eysel, Gonzalez-Aguilar & Mayer, 1980; Eysel, Gonzalez-Aguilar & Mayer, 1981). Considering this demonstration in the LGN and the changes observed in somatosensory and auditory cortices, it is not surprising that the adult visual cortex can also reorganize (Kaas, Krubitzer, Chino, Langston, Polley & Blair, 1990; Heinen & Skavenski, 1991; Gilbert & Wiesel, 1992; Schmid, Rosa & Calford, 1993). However, the changes in primary visual cortex (V1) are not simply a reflection of those occurring in the LGN, since extensive changes in receptive field position can be observed a few hours after retinal laser lesions (Gilbert & Wiesel, 1992; Schmid, Calford & Rosa, 1994). In addition, the reorganization in V1 can span larger distances in the visual field, in comparison with that in the LGN (Kaas *et al.* 1990; Gilbert & Wiesel, 1992).

The studies of visuotopic plasticity in adult brain cited above used photic lesions to deactivate portions of the retina. However, photic lesions (both with lasers and xenon photocoagulators) still pose specific problems to the interpretation of the results, as the exact delimitation of the affected area in the retina is difficult. For example, regions of the retina remote from the lesion may also be affected, although to a lesser degree (Eysel *et al.* 1981). In the study reported here we used the technique of deactivating the input of one of the retinae to the brain, by monocular enucleation or laser photocoagulation of the optic disc (Eysel, 1979), to induce the loss of vision in the monocular crescent of the peripheral visual field. In cats, this produces total loss of a sector of visual field that is 40–50 deg wide along the horizontal meridian (HM). In cortical terms this corresponds to an elongated strip only 1–2 mm wide (Tusa, Palmer & Rosenquist, 1978; Löwel and Singer, 1987), well within the limits of the reorganization observed by previous studies of visual cortex. With this method the location of the affected cortex is highly reproducible among different cases. In addition, the affected region is marked by sharp physiological transitions and after a few days, can be directly visualized by histochemical stains (Wong-Riley, 1979; Rosa, Gattass, Fiorani & Soares, 1992).

A second reason for examining the possibility of topographic reorganization in the representation of the monocular crescent is related to a more general question: once a piece of sensory cortex is deprived of its main normal afferents, can it be invaded by the inputs to (or from) *any* adjacent, unaffected representation? We reasoned that the monocular field is an interesting model to explore this question, as in the normal animal this portion of the visual field is never accessible to the ipsilateral eye. Thus, whereas with central laser lesions one observes displacement of receptive fields of the same eye to 'new' retinal regions, plastic changes in the monocular crescent representation would require the invasion of a domain of the contralateral eye by the ipsilateral eye. Previous work in adult rabbits (Clarke, Datskovsky, Grigonis & Hazel Murphy, 1992) reported that monocular enucleation results in a large silent region in striate cortex where the monocular crescent was originally represented, rather than showing evidence for the plastic arrangements that could be expected.

Our results demonstrate that, in the cat, some neurones located in the monocular sector of V1 remain responsive after enucleation of the contralateral eye. In a manner analogous to that observed with restricted laser lesions, these neurones respond to stimulation of portions of the visual field located around the scotoma (that is, in this case, at the edge of the binocular field of vision). However, the reorganization in these cases was not extensive, and, in contrast to results in somatosensory and auditory cortex and in the central representation of visual cortex, the large majority of neurones remained unresponsive even after more than 1 year survival. This result indicates the existence of natural boundaries that limit the reorganization of sensory maps in adults.

## METHODS

Ten adult domestic cats were used in acute electrophysiological extracellular recording experiments. Most of these animals received monocular treatment (either enucleation or laser photocoagulation) at different times before the recording session; one cat (FC4) was used as a control experiment in which the contralateral eye, rather than being deactivated, was simply occluded. All experiments were conducted following the ethical guidelines established by the Australian National Health and Medical Research Council, and were authorized and supervised by the University of Queensland's Animal Experimentation Ethics Committee.

### Monocular enucleations and laser photocoagulations

In order to produce unilateral loss of the peripheral visual field, four cats were monocularly enucleated, and six were treated with a high-power laser in and around the optic disc of one retina. All animals were initially anaesthetized with intramuscular (I.M.) injections of ketamine (30 mg $kg^{-1}$) and xylazine (3 mg $kg^{-1}$). Atropine (0·15 mg $kg^{-1}$) was given I.M. to reduce salivary secretions and in the case of the animals to be treated with the laser, to dilate the pupils. In one enucleated (FC7) and three laser-treated cats (FC5, FC6, FC11) the treatment was done during the recording session. For the enucleation, the anaesthesia was supplemented with intravenous sodium pentobarbitone (10 mg $kg^{-1}$) and 1–1·5% halothane for the duration of the procedure. In the laser-treated cases, only halothane (1–1·5%) was used. Monocular enucleations were done by the standard technique of cutting around the conjunctiva, sectioning the extraocular muscle insertions, then clamping and sectioning the optic nerve. After the surgical wound was cleaned, the orbit was treated with absorbent pads soaked with the long-lasting local anaesthetic Marcain (bupivacaine hydrochloride, 0·5%; Astra, North Ryde, NSW, Australia) and the eyelids were then sutured together. The photocoagulation treatment employed an argon laser projected through an optic fibre cable and focused by a dissecting

microscope and a plano-convex contact lens onto the retina. The laser and focusing system were designed and constructed by Lastek (Adelaide, SA, Australia), and allowed photocoagulation of the eyes with the animals held in the stereotaxic apparatus. A laser spot of 500 $\mu$m diameter, with an intensity of 1·0 W, was aimed at the optic disc and surrounding retina, severing the outgoing nerve fibres. In addition, the retinal blood circulation was interrupted by burning all the blood vessels leaving the optic disc. Thus, in the unlikely situation that some nerve fibres escaped direct thermal damage, the surviving ganglion cells would still be deactivated by anoxia (Eysel, 1979). The effectiveness of this treatment is illustrated by the complete absence of responses to this eye in the visual cortex immediately after the treatment. In addition, our previous studies demonstrated the absence of any activity elicited by this eye, as evaluated by visually evoked potentials, and that all ganglion cells in the treated retina degenerated within 1 week of the laser treatment (Schmid *et al.* 1993). Five experiments involved a survival time between the lesions and recordings (FC1, enucleation, 5 months recovery; FC2 and FC3, enucleation, 16 months recovery; FC8, photocoagulation, 1 month recovery; FC10, photocoagulation, 2 days recovery). During recovery, the animal was kept in a warm, dimly lit box under constant observation by the authors until upright posture was regained. It was then transferred to the cat colony, with normal visual stimulation, for the duration of the survival period under the supervision of qualified veterinary nurses. As the photocoagulation procedure is quick (15–30 min) and painless, the animals had recovered (i.e. were able to move around the cage, drink and eat) within 2 h of the end of the procedure. Due to the longer duration and different anaesthetic régime (as detailed above), in the cases where enucleation was used the recovery was slower (up to 6–8 h until the animal recovered full mobility). In these cases, the possibility of post-surgical pain was counteracted by intramuscular injections of a long-lasting analgesic (buprenorphine hydrochloride, 0·01 mg $kg^{-1}$; Temgesic, Reckitt & Colman, North Ryde, NSW, Australia), given as the animal showed the first signs of recovery from anaesthesia, and then every 8 h for the first 24 h.

**Electrophysiological experiments: animal preparation**

Each cat underwent a single non-recovery recording session. Initially, the animal was anaesthetized with a mixture of ketamine (50 mg $kg^{-1}$) and xylazine (5 mg $kg^{-1}$), and received I.M. injections of atropine (0·15 mg $kg^{-1}$) and dexamethasone (0·4 mg $kg^{-1}$). After the disappearance of withdrawal reflexes, the trachea was cannulated to allow for artificial ventilation. Throughout the subsequent surgical procedures and the experimental session, the animal lay on a thermostatically controlled soft heating pad, and the electrocardiogram was continuously monitored by means of a virtual oscilloscope system (MacLab 8, Analog Digital Systems, Castle Hill, NSW, Australia) run with the aid of an Apple Macintosh IIvx computer. The heart rate was kept under 175 beats $min^{-1}$ by small intravenous doses of anaesthetic (thiopentone sodium, 4 mg $kg^{-1}$). By using this criterion, we never observed spontaneous movements or changes in muscle tone during the surgical procedures. The skull midline was fitted with a stainless steel bolt and an acrylic well. A craniotomy 10 to 15 mm in diameter was made in the region circumscribed by the well, and the dura mater was resected. Once these procedures were completed the well was filled with silicone oil, and a picture of the cortical surface was taken; this was used as a reference for the placement of electrode penetrations. The eye, mouth and ear bars were removed, and the head was secured in position by the implanted bolt. Thus, an unobstructed field of vision was available. Finally, muscular paralysis was induced by the intravenous infusion of pancuronium bromide (Pavulon (Organon Australia, Lane Cove, NSW, Australia); 0·15 mg $kg^{-1}$, followed by 0·15 mg $kg^{-1}$ $hr^{-1}$) in a solution of sodium chloride (0·18%) and glucose (4%) with the addition of dexamethasone (0·4 mg $kg^{-1}$ $h^{-1}$). The total volume of fluids injected varied between 2·5 and 5 ml $hr^{-1}$, depending on the animal's weight. The cat was subsequently maintained under artificial ventilation with a gaseous mixture of $N_2O$–$O_2$ (7 : 3), and the respiratory volume and rate were adjusted to keep the percentage of $CO_2$ in the expired air between 3·5 and 4·5%. During paralysis, the depth of anaesthesia was assessed from the electrocardiogram (heart rate kept below 175 beats $min^{-1}$ as described above) and by the characteristics of the cortical activity: a superficial level of anaesthesia resulted in high spontaneous activity and burst-like, non-stimulus-locked firing by the cortical neurones. Anaesthetics (thiopentone sodium, 4 mg $kg^{-1}$ $h^{-1}$) were administered by slow I.V. infusion throughout the experiment and additional doses were given if judged necessary by the above criteria.

**Protection of the cornea, dioptric correction, control for eye movements and estimate of the field of vision**

Mydriasis, cycloplegia and retraction of the nictitating membranes were induced by the topical application of atropine (1%) and phenylephrine hydrochloride (10%) eye drops. Appropriate focus was achieved by means of hard contact lenses selected by streak retinoscopy. These lenses brought into focus the surface of a 40 cm diameter translucent hemispheric screen centred on the eye ipsilateral to the cerebral hemisphere to be studied. The lenses also protected the corneae from desiccation. The quality of the optic media, as evaluated by repeated ophthalmoscopic inspections, appeared stable throughout the experiment. In control recordings, as well as in the recordings in cases where acute deactivation was performed, the eyes could be alternately covered by opaque black plastic occluders. The positions of the optic discs, major blood vessels and if visible, the area centrales were projected onto the screen by the fibre optic method (Pettigrew, Cooper & Blasdel, 1979), and checked every 1–2 h during the experiment. In most animals no residual movement could be detected 4–6 h after the initial dose of pancuronium. The hemispheric screen was rotated to bring its horizon to coincide approximately with the horizontal meridian (HM), based on the position of the optic disc at the beginning of the experiment (Bishop, Kozak & Vakkur, 1962).

The extent of the monocular field of vision was estimated in each case by the corneal reflection method (Sousa, Gattass, Hokoç & Oswaldo-Cruz, 1978). A punctiform light source was moved onto the surface of the hemispheric screen, and its corneal reflection was monitored by an observer positioned along the optic axis of the eye. The transition points at which the corneal reflection was no longer observed were used to define the perimeter of the monocular field of vision. This method estimates the effective field of vision of the eye, corresponding to the optic field *minus* sectors obscured by ocular annexa and other head structures. In the cat, the effective field is a slight overestimate of the binocular field of vision, as a small sector of the optic field of the eye is not served by the temporal retina (Hughes, 1976). Thus, if any errors occurred, they resulted in underestimating, rather than

overestimating, the extent of the sector of cortex deprived of its inputs by monocular enucleation.

**Electrophysiological recordings: equipment and procedures**

Tungsten-in-glass microelectrodes with an exposed tip of 10–15 $\mu$m were inserted in vertical penetrations. The electrophysiological signal was amplified, fed into a waveform discrimination system (SPS-8701, Signal Processing Systems, Adelaide, Australia) run with the aid of an IBM-AT desktop computer, and monitored by means of a loud speaker with oscilloscopes. The minimum response fields of single units, small unit clusters or background were mapped by correlating the stimulation of specific portions of the visual field with the increments of the neural activity, as monitored both on an oscilloscope and through an audio system.

Visual stimuli comprised luminous white spots (1–10 deg in diameter) and bars (2–20 deg long) moved on the surface of the screen by means of a hand-held projector. The background illumination was around $1 \times 10^{-2}$ cd m$^2$, and stimuli were varied between $5 \times 10^{-1}$ and 5 cd m$^2$ according to the responses. These stimuli were invariably adequate to elicit responses of V1 neurones in the binocular representation. However, as will be described below, most neurones in the reorganized peripheral sector seemed unresponsive to visual stimuli. In order to ensure that this was actually the case, other tests were also applied if an unresponsive site was found. These included a small bar of light swept onto the animal's nose and diffuse illumination of the temporal retina with an ophthalmoscope. These tests were aimed at checking the possibility that the electrode might still be in a normally innervated portion of cortex, that responded to a portion of the visual field that was obscured by the nose. This was unlikely in view of the results of Hughes (1976) and those illustrated in Fig. 2; indeed, this was never observed. Receptive fields were drawn as rectangles parallel to the axis of preferred orientation, or ovals, if no orientation bias was apparent.

In all animals, the same region of V1 was studied, in order to reduce any differences that might result from sampling biases. As a routine, V1 was studied between anterior–posterior (A–P) levels −2 to +5, corresponding to elevations from the HM to approximately −30 deg. In order to avoid ambiguity in reconstructing the electrode tracks during the data analysis, electrode penetrations were separated by at least 400 $\mu$m. Small electrolytic lesions (4 $\mu$A, 10–15 s, electrode negative) were placed in several penetrations to aid in the postmortem track reconstruction and identification of the recording sites. The necessity to outline the region of cortex that was deprived of its normal input by the monocular treatment in animals with different postlesion times resulted in different strategies. In the cases involving recovery time after the monocular deactivation, the electrode penetrations were made blindly with respect to the limits of deprived and undeprived cortex in V1, and the distinction between recordings in these regions relied entirely on the examination of histological sections. With the cytochrome oxidase stain, alternately light and dark ocular dominance columns could be seen throughout the binocular sector, while the monocular field stained homogeneously light. A transition between binocular and monocular cortex (e.g. those shown by different tones of grey in Figs 4 and 5) could be observed even 3 days after photocoagulation. However, in cases in which the immediate effects of lesions were explored this was more difficult, as the postlesion times were not enough to produce differences in cytochrome oxidase stain intensity that were related to deprivation. Thus, in these experiments the monocular lesion was preceded by topographic mapping of the representations of the two eyes in the splenial sulcus. This precaution enabled us to determine the deprived sector with a precision of approximately 500 $\mu$m, the separation between electrode tracks. The deprived sector was then restudied, after the monocular enucleation or optic disc photocoagulation.

One of the main objectives of this study was to compare the responsiveness of neurones in the deprived cortex of the monocular crescent with that of neurones in other portions of V1 that still received normal input from one of the eyes. In order to avoid sampling bias in this analysis, we obtained recordings along the penetrations according to a predetermined grid, rather than specifically searching for responsive units. Thus, we analysed and compared responsiveness of cortex in terms of the probabilities of finding responsive neurones, based on a random sampling strategy. The responsive sites were classified as cellular (if the response consisted of clear neuronal spikes) or background (if a 'swish' response was audible but no clear spikes were observed). The separation between recording sites was 500 $\mu$m to 1 mm in penetrations along the medial wall of the cerebral hemispheres and 100 to 300 $\mu$m in the banks of the splenial sulci.

**Histology**

At the end of the experiment, the animal was given a lethal dose of sodium pentobarbitone (50 mg kg$^{-1}$) and transcardially perfused with 0·9% saline, followed by 3% paraformaldehyde in 0·1 M phosphate buffer and 3% paraformaldehyde–10% sucrose in phosphate buffer. The brain was removed from the skull, blocked according to stereotaxic co-ordinates and left overnight in fixative before being sectioned into 50 $\mu$m slices in the coronal plane with the aid of a freezing microtome. Every section was saved, and alternate sections were histochemically stained for cytochrome oxidase (Wong-Riley, 1979) or stained for cell bodies with Cresyl Violet. The reconstruction of the position of the recording sites was based on the electrode tracks and electrolytic lesions. An estimate of the shrinkage during the histological processing was obtained for each case by comparing the real distance between penetrations (based on the microdrive readings) with the distance measured in the sections. The laminar borders in V1 were identified by the criteria of Wong-Riley (1979).

## RESULTS

We begin by describing the normal visual topography in the splenial sulcus, together with the immediate effects of monocular deactivation on the responsiveness of peripheral V1. The partial recovery of responsiveness in the deprived region of peripheral V1 will be described next, in terms of the topographic organization and laminar distribution of the responsive neurones. Finally, the physiological transition between V1 and the splenial visual area will be demonstrated.

### Normal topography and immediate effects of enucleation and optic disc photocoagulation

In cats, the portion of V1 that normally represents the visual field periphery is located in the dorsal wall of the splenial sulcus. In this region, increasing eccentricities are represented in a medial-to-lateral gradient (Kalia &

Whitteridge, 1973; Tusa *et al.* 1978). Our recordings confirm these previous findings. Figure 1 shows an example of control recordings in one cat. These recordings were aimed at determining the border between the binocular and monocular sectors of V1, and at exploring the possibility of any responses to the ipsilateral eye in the representation of the monocular crescent in normal animals. Although this is *a priori* unlikely, responses to the ipsilateral eye in this sector could exist, with large binocular disparities between the receptive fields of the two eyes. However, this was never observed; instead, as expected, once one crossed the border between binocular and monocular V1 the neurones responded exclusively to the contralateral eye (Fig. 1). For most of the lower quadrant representation, the binocular–monocular border runs approximately half-way along the mediolateral extent of the sulcus. In coronal sections, the representation of the monocular crescent is widest around the level of the horizontal meridian (around the A–P level −2; Fig. 4*A*). At this level, the splenial sulcus bends laterally and ventrally, resulting in much of the monocular crescent being sectioned tangentially.

Based on the corneal reflection method, the binocular hemifields of cats are 45–52 deg wide in different animals. In Fig. 2, we compare this estimate with the sector of the visual field defined by the outer border of all binocular receptive fields recorded in one control and four acute deactivation cases. For this analysis, receptive field positions were corrected for horizontal and vertical disparity (Nikara, Bishop & Pettigrew, 1968). On average, there is a reasonable agreement between the two estimates. However, in some animals (e.g. Fig. 4) a mismatch of up to 7 deg was observed between the estimate based on the most peripheral receptive fields mapped through the

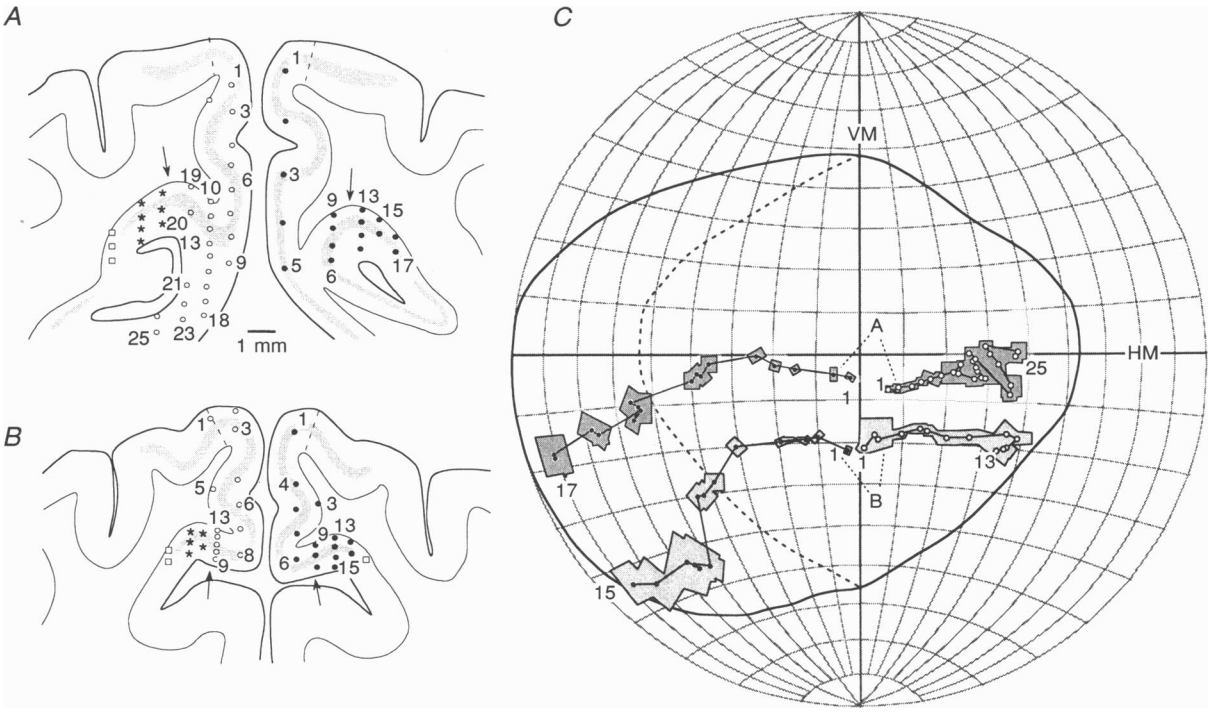


**Figure 1. Extent of the representation of the ipsilateral and contralateral eyes in V1 in cat FC4**

*A* and *B* are coronal sections at two different levels of V1, with the position of the recording sites indicated. Recording sites in V1 that yielded responsive neurones are shown by circles (○ and ●), unresponsive sites in V1 by ★, and recording sites with responsive neurones that were deemed to lie outside V1 based on receptive field size and topography are indicated by □. For clarity, only every second recording site is shown. Layer 4 is indicated in grey. *C*, equatorial–zenithal chart showing the extent and centre of receptive fields recorded at these sites; the separation of the grid lines indicates 10 deg in the visual field. All receptive fields were plotted via stimulation of the left eye. In the right hemisphere (●), the receptive field sequences extended all the way to the temporal border of the field of vision of the left eye (continuous black line), and responsive neurones were found throughout V1. In the left hemisphere (○), responses were found only up to the border of the binocular field of vision. Recording sites lateral to the monocular–binocular border (arrows in *A* and *B*) yielded normal spontaneous activity, but no visual responses to the left eye except when recordings extended into the splenial visual area (□). The uniocular field of vision of the cat actually extends beyond the 90 deg azimuth. VM, vertical meridian; HM, horizontal meridian.

ipsilateral eye and the estimate of binocular hemifield based on the corneal reflection (the latter being larger), thus supporting the results of Hughes (1976).

Immediately after monocular enucleation or laser deactivation, the region of V1 that previously corresponded to the monocular crescent representation was completely unresponsive. Thus, in all four acute experiments (one involving enucleation and three with photocoagulation), recordings up to 12 h after treatment revealed no evidence for reorganization of the visuotopic map. This is illustrated graphically in Fig. 3. In this figure, the probability of obtaining a neuronal response to the ipsilateral and contralateral eyes, based on random sampling, is compared before and after the monocular treatment. For the purposes of this analysis, four sub-regions of V1 are considered separately: central, binocular peripheral, monocular peripheral and transitional (the last one reflecting our error in the determination of the border between the binocular and monocular sectors of V1). As shown in Fig. 3*A*, the responsiveness to the contralateral eye before lesioning was uniformly high in V1. Under our recording conditions, 80–90% of the recording sites throughout this area yielded clearly responsive cells; the responses at the remaining sites constituted an unresolved background that may correspond to remote cell activity or fibres. In contrast, the responses to the ipsilateral eye were

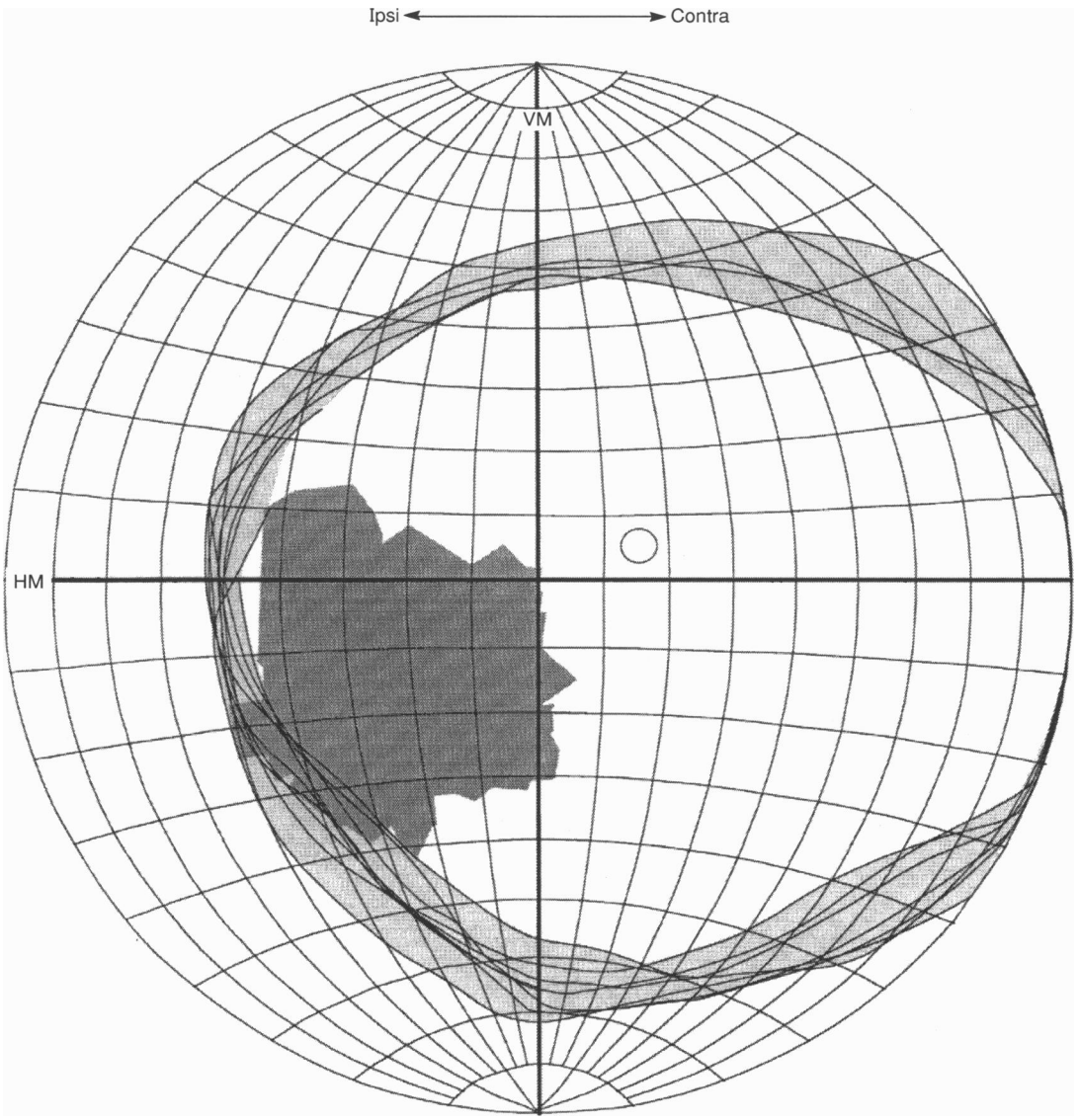


**Figure 2. Effective monocular field**

Extent of the effective monocular field of vision in all animals used in this study (thin lines are individual cases, light grey areas indicate the inter-animal variability) in comparison with the extent of the binocular representation in the region of primary visual cortex in this study (dark grey). The theoretical limit of the binocular field of vision extends slightly beyond the cortical binocular representation. The white circle represents the optic disc.

more variable with location (Fig. 3*B*). The percentage of neuronal responses to this eye decreased from the central to the peripheral regions of the binocular field. These control recordings found no evidence for responses to the ipsilateral eye in the monocular crescent sector. After enucleation of the contralateral eye, the same pattern remained (Fig. 3*C*) for at least 12 h. In spite of one responsive unit cluster, the probability of obtaining neurones responsive to the ipsilateral eye in the crescent representation did not differ before and after monocular deactivation ($\chi^2 = 1{\cdot}00$; $0{\cdot}6 < P < 0{\cdot}7$, d.f. = 2). Although a quantitative assessment was not attempted, the spontaneous activity of neurones in the deprived monocular crescent region was not obviously reduced in comparison with the control situation.

### Chronic recovery of responses

Figure 4 illustrates the recordings that were obtained in a cat during a session that extended from 48 to 72 h after monocular optic disc photocoagulation. During the 48 h recovery period, the animal had normal visual stimulation, as detailed in Methods. In contrast to the situation immediately following the monocular treatment, cellular responses to the stimulation of the remaining eye were observed at some sites in the deprived sector of the ipsilateral cortex (19·5% of the total sample). Nonetheless, the neurones in the large majority of the recording sites either remained unresponsive (55·3) or responded only with unresolved background (25·2%) that may correspond to remote cells or fibre activity. The receptive fields that could be plotted in the deprived peripheral sector of V1 now represented the nasal-most edge of the binocular field of vision, instead of the contralateral monocular crescent. For example, in section *A*, recordings down the medial bank of cortex (sites 1–15) and moving laterally in the splenial sulcus (16–28) resulted in receptive fields that moved towards the periphery of the visual field. At the transitional strip between the original binocular representation and the reorganized sector of peripheral V1, the edge of the binocular field of vision is represented (sites 29–36). Beyond this, moving into the reorganized sector, the proportion of cellular responses (filled circles) diminishes dramatically. Instead of moving further peripherally, all receptive fields concentrate at the edge of the binocular field, that is re-represented. Nonetheless, the visual topography is not completely eroded, as one can still observe the expected displacement of the receptive fields into the upper quadrant as recording sites move further lateral and ventrally (sites 37–52). The responsive cells tended to occur in clusters. For example, in one penetration (section *B*, sites 38–49) cellular responses were observed at virtually every site, whereas in several others only unresponsive sites and a few background responses could be found. We could not detect a relationship between the

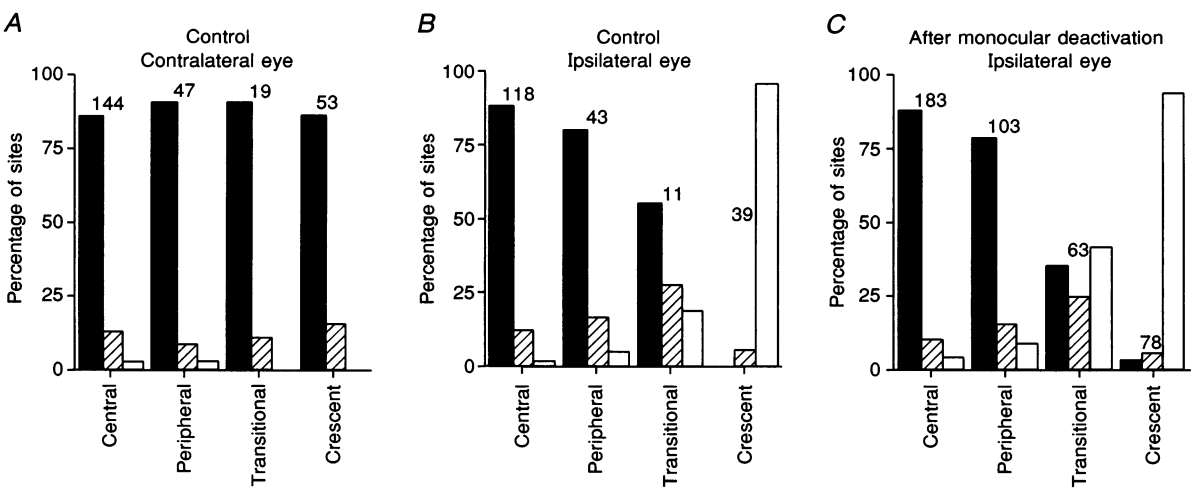


**Figure 3. Responses to the contralateral and ipsilateral eyes**

Responses to the contralateral and ipsilateral eyes in normal animals (*A* and *B*) and responses to the ipsilateral eye up to 12 h after inactivation of the contralateral eye (*C*), as a function of location in V1. The first two sub-regions correspond to sites recorded along the dorsal and medial hemispheric walls (Central) and along the portion of the splenial sulcus medial to the border between the binocular and monocular sectors (Peripheral). The fourth subdivision (Crescent) includes all sites in V1 lateral to this border, with the exception of an arbitrarily defined transitional strip 1 mm wide, centred on our estimate of the monocular–binocular border (Transitional). In view of the error involved in our method for determination of the binocular field extent, we analysed this transitional strip separately from the recording sites that are undoubtedly in the monocular crescent representation. ■, cellular response; ▨, background and □, unresponsive. The number of sites at which neuronal responses were recorded are given above the bars.

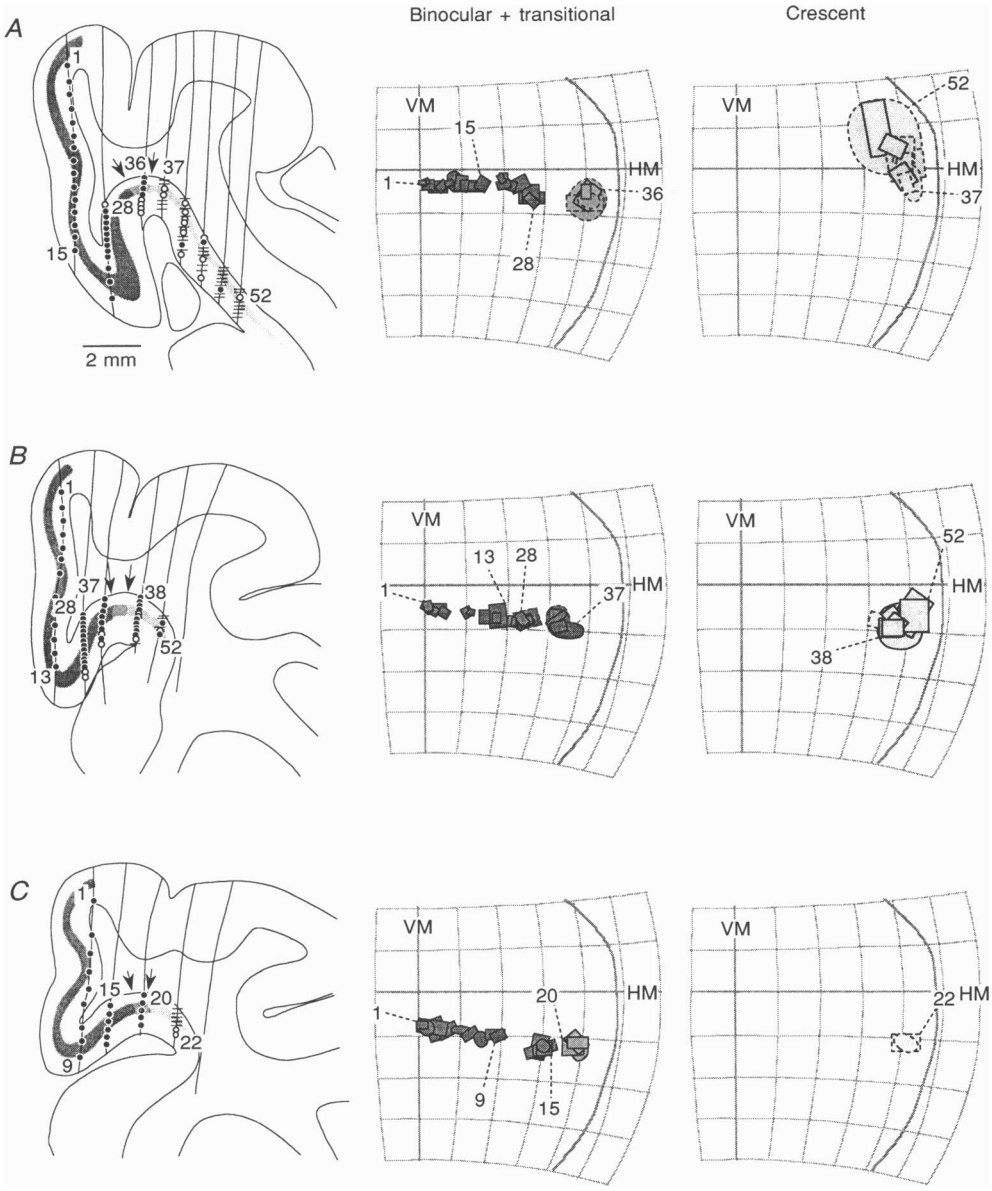


**Figure 4. Recording sites and receptive fields after inactivation of the contralateral eye**

Recording sites and receptive fields in V1 2–3 days after inactivation of the contralateral eye (case FC10). *A*–*F* are coronal sections, arranged from caudal (*A*) to rostral (*F*), indicating the recording sites. Sites with cellular responses are indicated ●, sites with background responses ○, and horizontal bars indicate sites that were completely unresponsive to visual stimulation. Layer 4 is indicated in different tones of grey. Dark grey indicates non-deprived cortex that originally contained representation of the two eyes, light grey indicates the deprived peripheral representation and intermediate grey (between arrows) indicates our uncertainty in the determination of the monocular–binocular border. These subdivisions were determined on the basis of the examination of cytochrome oxidase-stained sections (see Methods). The receptive fields recorded at those sites are shown in the middle and right columns. Receptive fields plotted based on definite cellular activity are indicated by a continuous outline and those plotted based on background 'swish' are indicated by dashed outline. The charts indicate 10 deg intervals in the visual field, and the outer border of the binocular field of vision of this animal.

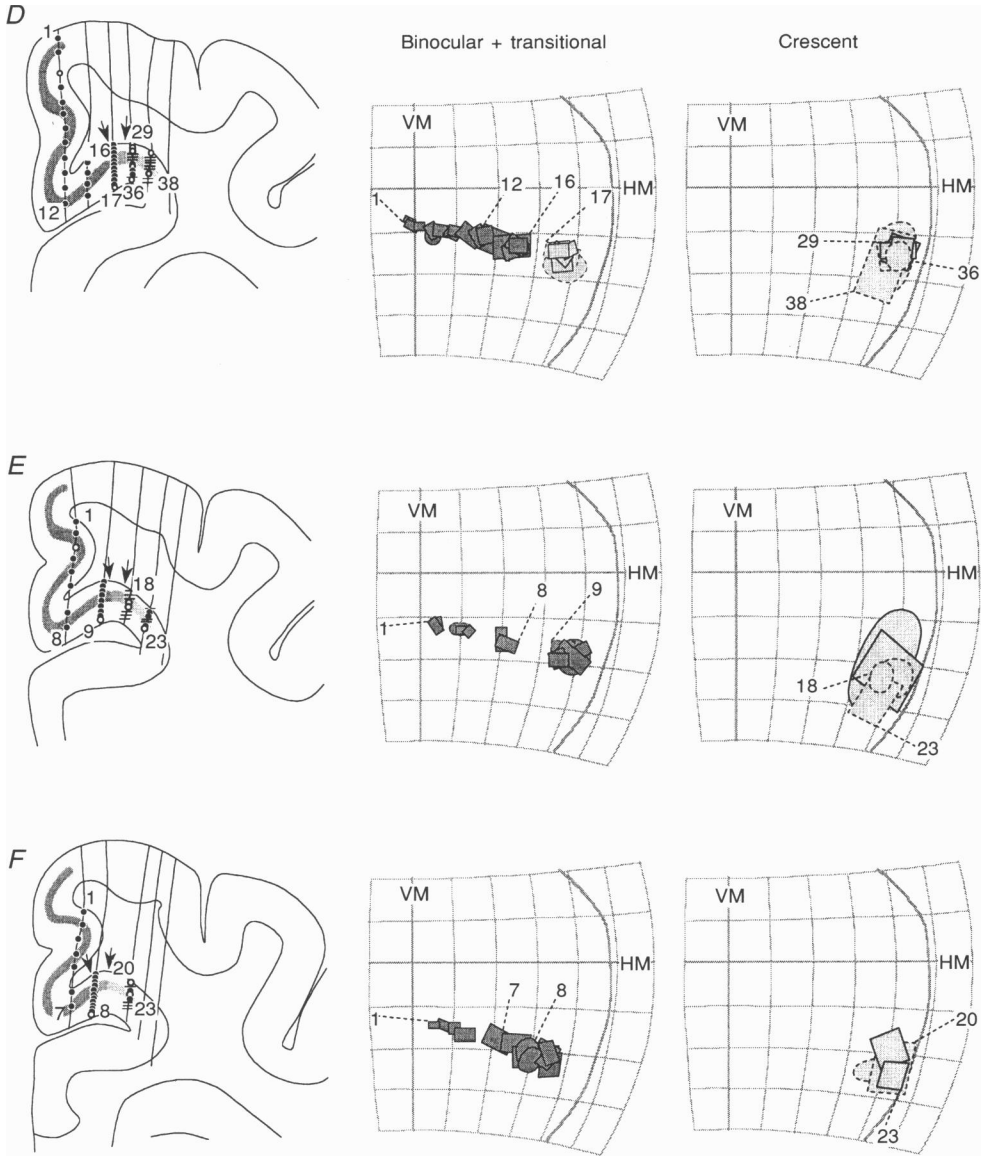


Figure 4 continued.

proportion of responsive cells in a given penetration and the distance from the medial border of the deprived sector. Sites with displaced receptive fields were observed up to the border of V1 (e.g. sections *B*, *E* and *F*), i.e. 2 mm away from their normal representation.

Similar recordings were obtained in cats 1, 5 and 16 months after monocular deactivation. Recordings from the cat with the longest survival time after monocular enucleation are illustrated in Fig. 5. As in the previous figure, most recording sites yielded no responsive cells, and the sites that yielded responsive neurones tended to cluster together. The data on recovery experiments are summarized graphically in Fig. 6. In spite of the long survival times after lesioning in some cases, there was no obvious change in the pattern of responsiveness of the deprived region. This impression was confirmed statistically: a $\chi^2$ test demonstrated that the proportions of neurone-responsive, background-responsive and unresponsive sites were not significantly different throughout the series of recovery experiments ($\chi^2 = 4{\cdot}97$; $0{\cdot}5 < P < 0{\cdot}6$, d.f. $= 6$). The only obvious difference that we could observe among these animals was in regard to the spontaneous activity of the cortex. Recordings in the

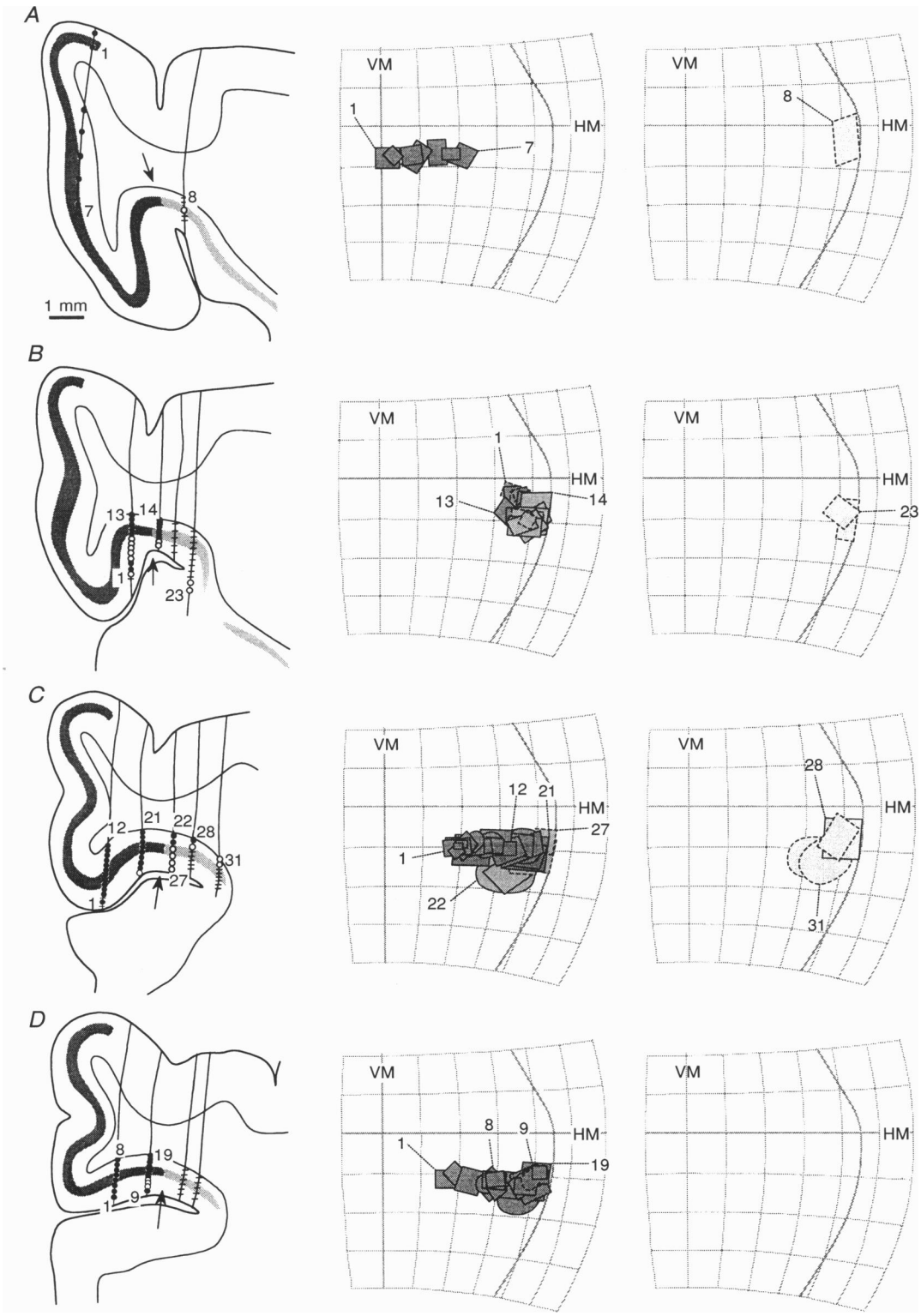


**Figure 5. Recording sites and receptive fields in one animal**

Recording sites and receptive fields in one animal that survived for 16 months after monocular enucleation (FC3). In this case, the border between monocular and binocular representations was sharply defined. Thus, a 'transitional' strip of layer 4 is not indicated in the figure. For other conventions, see legend to Fig. 4.

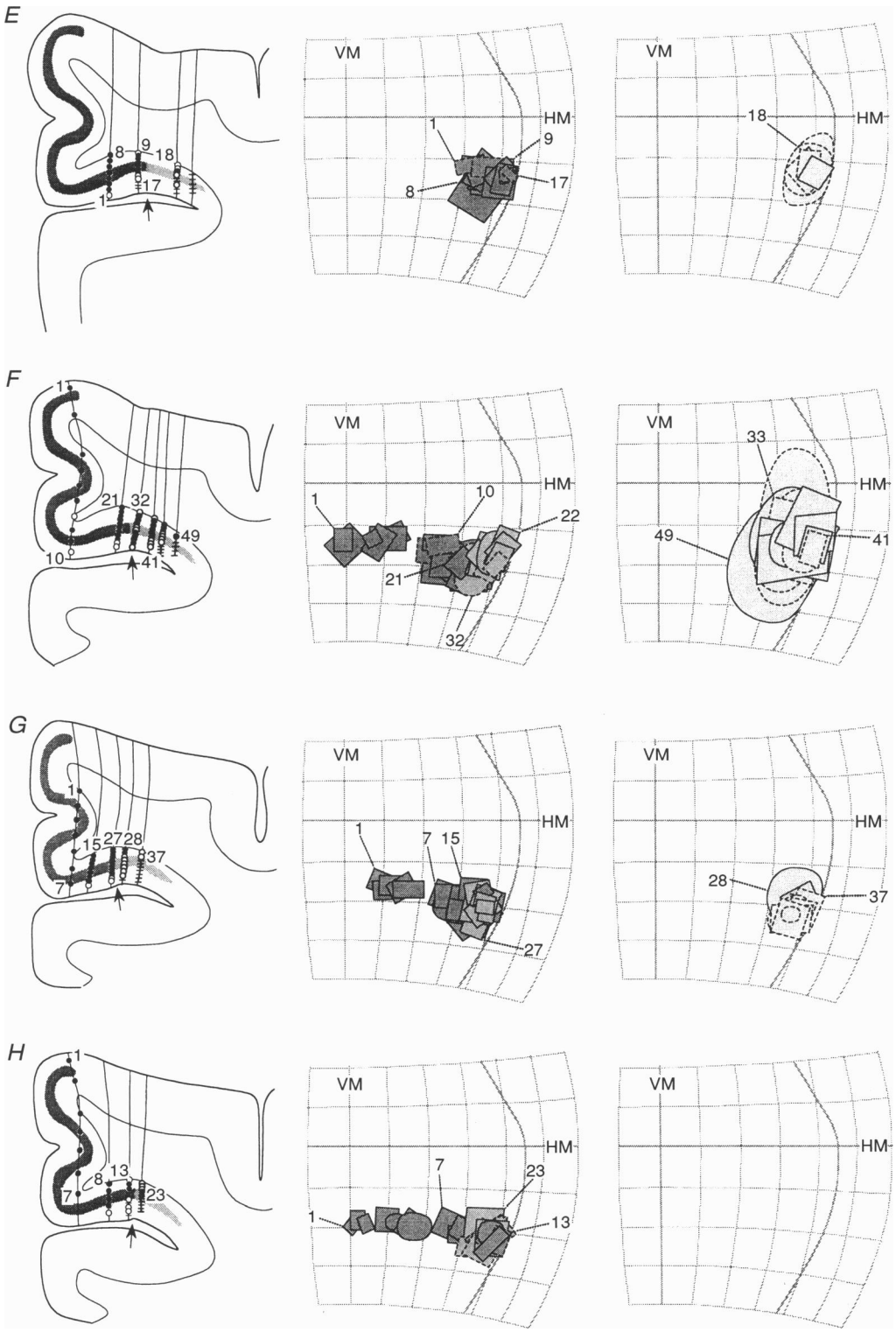


Figure 5 continued.

reorganized crescent sector of long-term recovery cats were notable for the lack of spontaneous activity. Whereas in the 48 h survival case (Fig. 4) the neurones in the deprived monocular crescent sector were still spontaneously active, this activity was almost entirely abolished for longer survival times. In these cases, whenever the electrode tip was positioned at a site in the deprived monocular crescent, there was little evidence of cellular activity apart from the injury discharges when the electrode was moved and for the occasional responsive units.

In addition to the patchy aspect, the distribution of responsive neurones in the reorganized peripheral sector was also characterized by a laminar preference. Since multiple lesions were not made along every penetration, the assignment of recording sites to specific layers also involved some interpolation between the transitions of activity (fibre–cellular–sulcus). Thus, for this analysis, we grouped the recording sites into only three groups (infragranular, granular and supragranular), and excluded recordings within 100 $\mu$m of the presumed white matter and layer 1. As illustrated in Fig. 7, the probability of obtaining responsive neurones was highest in the infragranular layers, and lowest in the supragranular layers. This difference between layers was significant ($P < 0{\cdot}05$, $\chi^2$ test) except for the 2–3 day recovery case. In pre- and postlesion recordings outside the peripheral representation there was no statistically significant difference in the proportion of unresponsive sites between layers.

## Response properties

In all recovery cases, the responsive neurones in the deprived monocular crescent sector had abnormal response properties. Although clearly defined single units could be observed in the oscilloscope trace and computer display, the responses to visual stimulation were not vigorous. In addition, the responses rapidly habituated to repetitive stimulation. Typically, intertrial intervals of 10 s or more were required to obtain consistent responses; otherwise, the response rapidly faded. On the other hand, orientation selectivity did not seem to be affected, as most neurones in

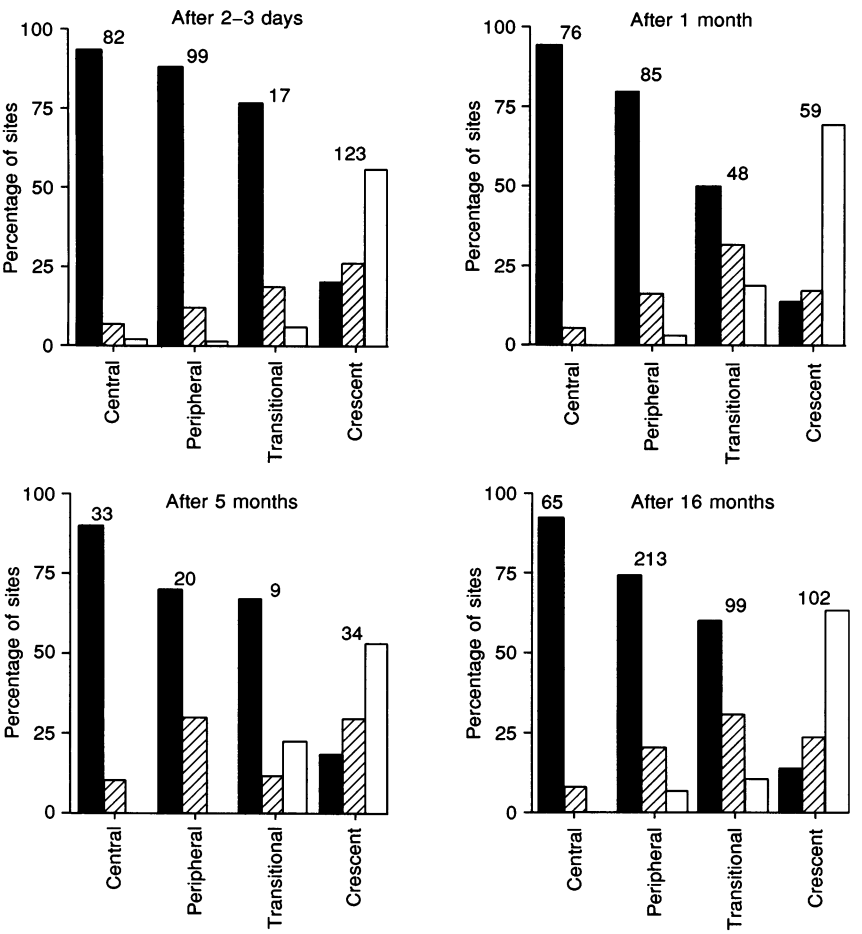


**Figure 6. Percentage of sites responsive to the ipsilateral eye**

Percentage of sites responsive to the ipsilateral eye in different portions of V1 as a function of survival time. 'Transitional' corresponds to a strip 1 mm wide centred on the transition between the monocular and binocular representations. ■, cellular response; ▨, background and □, unresponsive. For conventions, see legend to Fig. 3.

the reorganized peripheral sector were still tuned for this parameter. The low firing rate and rapid habituation that characterized the responses of neurones in the reorganized peripheral representation made a precise determination of the receptive field size difficult. For this reason, we did not try to compare quantitatively the sizes of receptive fields in the normal binocular periphery and in the reorganized peripheral representation.

In contrast to the changes observed in the reorganized peripheral sector between controls, immediate and recovery cases, the responses to the ipsilateral eye in the binocular sector of V1 showed little evidence of being disturbed by the treatment applied to the contralateral eye (Fig. 6). In all recovery cases neurones with sharp responses and normal visuotopic organization were found routinely just outside the reorganized peripheral sector.

### Magnification factor

The data illustrated in Figs 4 and 5 demonstrate that, in recovery cases, all receptive fields recorded from the portion of V1 that originally corresponded to the monocular crescent representation responded to stimulation at or close to the edge of the binocular field. This could result either from re-representations of the edge of the binocular field, or from an overall shift of the retinotopic map along the surface of V1. In order to explore these possibilities, we compared the cortical magnification factor (CMF) of V1 (Daniel & Whitteridge, 1961) in pretreatment controls and recovery experiments (Fig. 8). This figure demonstrates that, in contrast to the normal situation where the CMF decreases monotonically with eccentricity, in recovery cases there is a second peak and more variance in the values at the periphery of the binocular visual field. Thus, the destruction of normal afferent input to the peripheral sector of V1 merely causes a re-representation of a portion of the visual field that is already served by cortex elsewhere in the splenial sulcus, instead of an overall shift in the map.

### Splenial visual area

A representation of the peripheral visual field at the lateral extremity of the splenial sulcus, was described by Kalia & Whitteridge (1973) and termed the splenial visual area (SVA); however, the existence of this area could not be confirmed by subsequent studies (e.g. Tusa *et al.* 1978). In our material, we could routinely evoke cellular visual responses from the SVA. This visually responsive belt was only 1–2 mm wide (Fig. 9). Confirming the report of Kalia & Whitteridge, we found that the receptive fields in this area were invariably larger than those in V1 at the same eccentricity and that they tended to move towards the central visual field as the electrode was moved laterally. In monocularly deactivated animals, the visual responses in the SVA were unaffected except for the fact that the peripheral limit of the receptive fields corresponded to the edge of the binocular hemifield. Thus, electrode

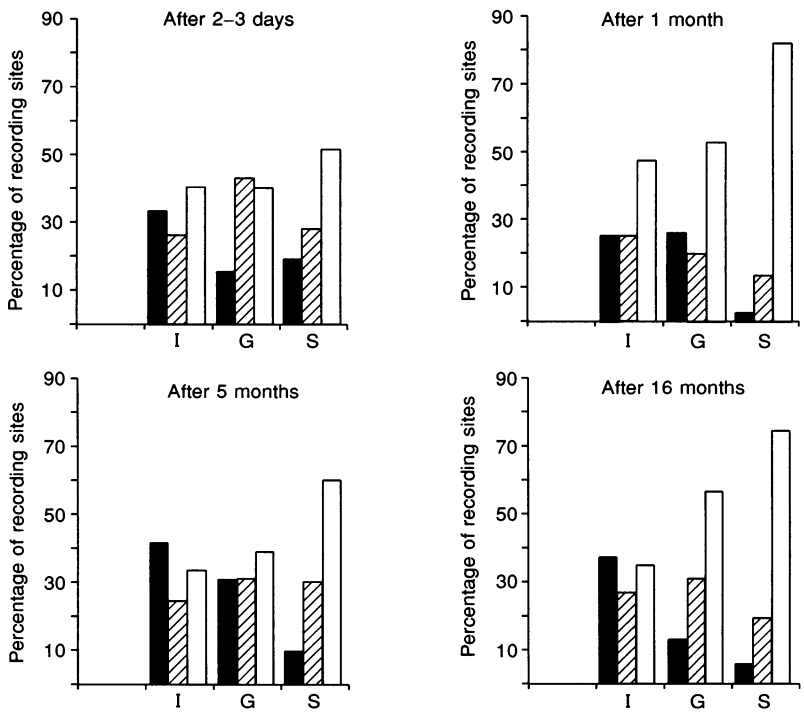


**Figure 7. Percentages of recording sites responsive to the ipsilateral eye in the deprived peripheral representation, as a function of depth in the cortex**

I indicates recordings in the infragranular, G, in the granular and S, in the supragranular layers. ■, cellular response; ▨, background and □, unresponsive.

penetrations crossing the splenial sulcus from medial to lateral would first find a sector of normal spontaneous activity and vigorous responses in small receptive fields (in V1), followed by a sector of low spontaneous and evoked activity (in the reorganized peripheral sector of V1), and finally a sector of high spontaneous activity and large receptive fields (the SVA). The physiological transitions between these sectors were sharp and were matched by histological transitions. Thus, it is unlikely that the evidence for reorganization we observed is due to sampling outside V1.

## DISCUSSION

We explored the possibility that reorganization of the map of the visual field periphery in adult cat striate cortex occurs after destruction of the inputs from one eye. In this situation, any reorganization of the visuotopic projection would require the ipsilateral eye to take over territory that is normally an exclusive domain of the contralateral eye. In contrast to observations in the somatosensory cortex (Calford & Tweedale, 1988) and in the central representation of visual cortex (Gilbert & Wiesel, 1992; Schmid *et al.* 1994), in the monocular crescent there is no immediate retinotopic reorganization. Within a few days of monocular deactivation, a fraction of neurones acquired receptive fields that were displaced relative to their expected location. Nonetheless, these responses were weak and most neurones remained unresponsive. In spite of survival times of more than a year, there was no evidence for a progressive 'filling in' of the deprived sector with a larger proportion of responsive neurones or neurones with stronger responses. Thus, our results indicate that there is a limit to the reorganization of the topographic map in monocular V1.

In view of the widespread evidence from recent studies of plasticity in the sensory representations in adult brains, perhaps the most remarkable aspect of our results is the low incidence of such effects. Thus, before discussing the implications of our observations, some methodological points must be addressed. The possibility that the lack of plastic effects may be due to over-anaesthetizing the

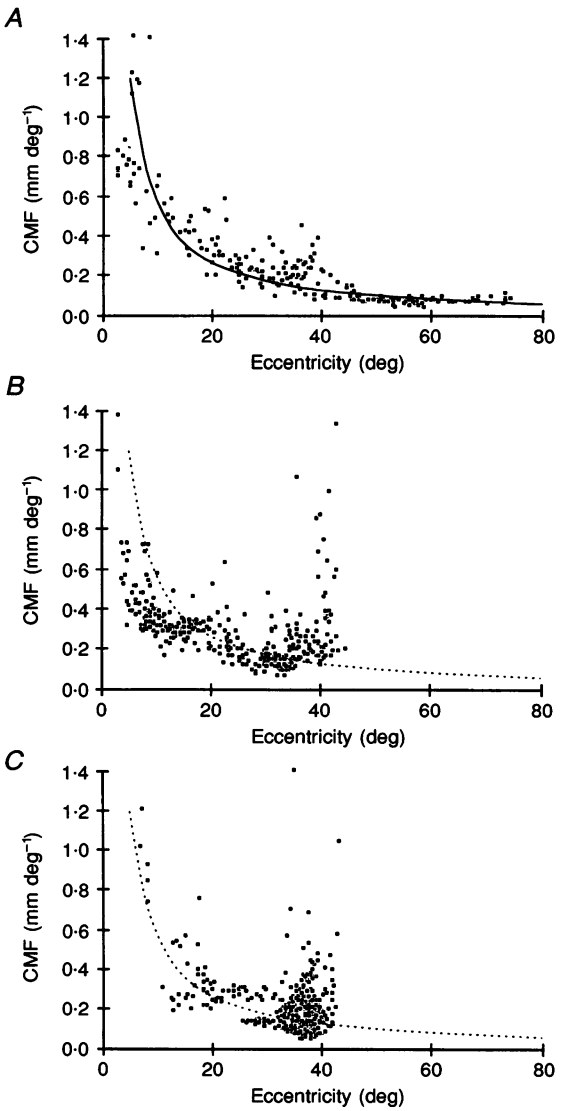


**Figure 8. Cortical magnification factor as a function of eccentricity in V1**

Cortical magnification factor (CMF; mm of cortex between two recording sites per angular distance between the centres of receptive fields recorded therein) as a function of eccentricity in V1. *A*, control animal FC4, showing a monotonic decrease of the CMF with eccentricity. A power function fitted to the data is shown by a continuous line. *B* and *C*, CMF in two animals that had the contralateral eye deactivated. *B*, FC10 after 2–3 days; *C*, FC3 after 16 months. The dashed line is the function fitted to the normal animal, drawn for comparison.

animals can be dismissed based on the fact that, in identical conditions, we observed immediate and chronic shifts in receptive field position in the binocular sector of V1 (Schmid *et al.* 1993, 1994). Moreover, we were consistently able to elicit vigorous visual responses from both the binocular portion of V1 and the splenial visual area (Kalia & Whitteridge, 1973). The latter area in particular may require optimal recording conditions, as other researchers have found the SVA to be unresponsive in preparations that were adequate for recordings in V1 (Tusa *et al.* 1978).

Our method for evaluating the incidence of plastic effects is based on the probability of obtaining a neuronal response at recording sites that follow a regular grid. We chose to report the incidence of background responses, that may have originated either in fibre tracts or in units that were far from the tip of the electrode; it is difficult to distinguish between these possibilities. Among the cases in which the cat was allowed to recover from the monocular treatment, 60–70% of the recording sites in the deprived peripheral representation were unresponsive to visual stimuli. In our

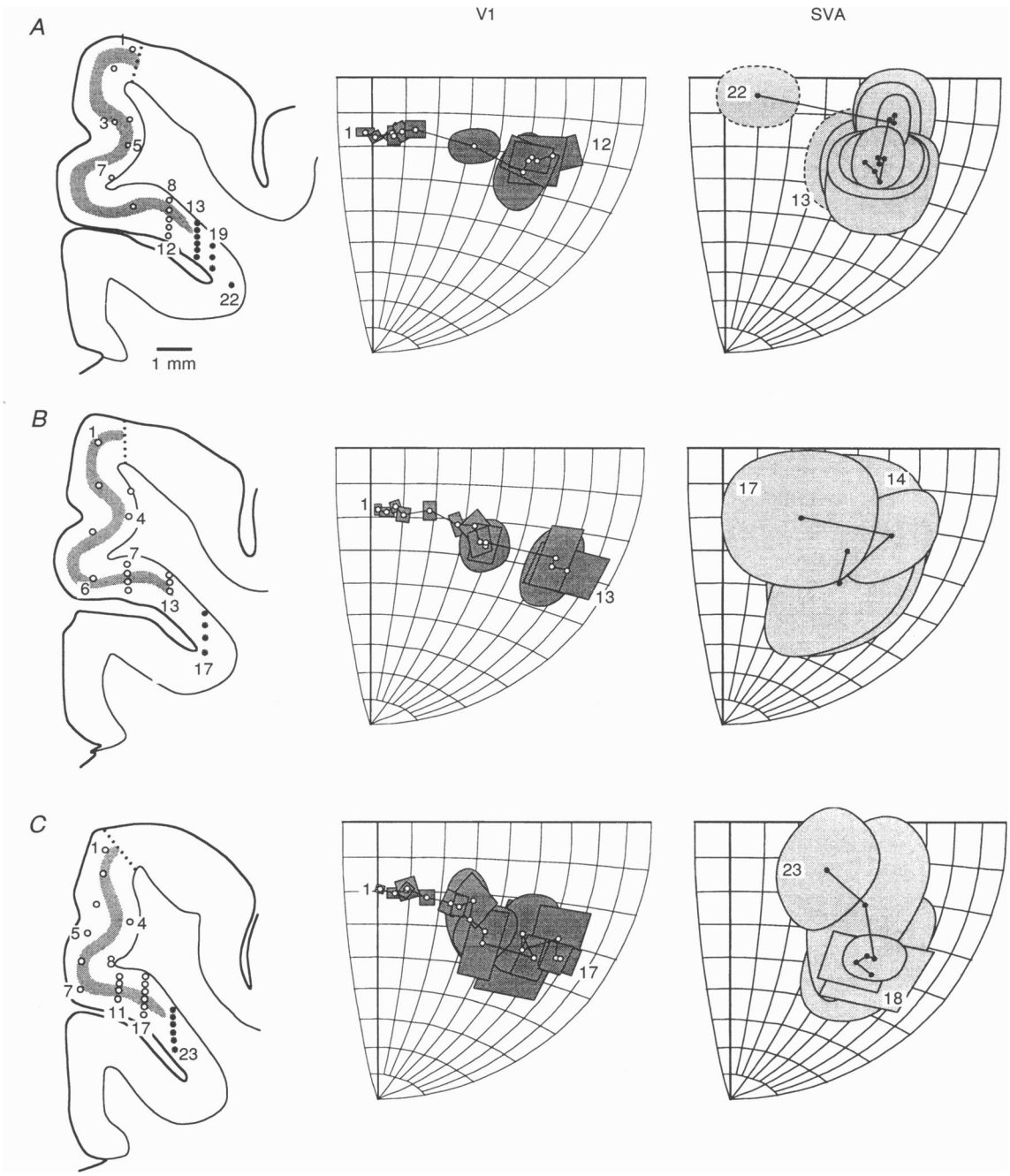


**Figure 9. Border area**

Border between V1 and the splenial visual area (SVA) in the hemisphere contralateral to the remaining eye of one animal (FC1). Conventions as in Fig. 4.

evaluation, among the remaining 30–40%, less than half can be counted as bona fide responses of neurones located close to the electrode tip. Nonetheless, we have reported the occurrence of background activity in order to cover the possibility that different researchers, using different criteria, may obtain a different proportion of responsive sites. Even if one considers this unreliable activity, the peripheral representation of V1 in monocularly deactivated cats shows an overall low responsiveness. In rabbits, Clarke and his collaborators (1992) reported that enucleation of one eye in adults produces a large unresponsive sector in the dorsomedial portion of V1. The difference between their result and ours (no responses *vs.* low responsiveness) is likely to be a reflection of the differences between these species, as in rabbits the uncrossed contingent of retinal ganglion cells forms less than 1% of the total population (Robinson, Sung, Dreher & Taylor, 1990).

**Retinotopic plasticity in the visual cortex**

The capacity of adult visual cortex to respond to inactivation of portions of the retina by reorganization of its visuotopic representation is now well established (Kaas *et al.* 1990; Heinen & Skavenski, 1991; Gilbert & Wiesel, 1992). After restricted laser lesions of the retina and recovery times ranging from 1 week to several months, most neurones in cat V1 that originally represented the lesioned retina acquire displaced receptive fields. Using restricted monocular laser lesions in cat central retina, Schmid and collaborators (1993) reported that neurones in the large majority of the recording sites in the deprived sector of V1 responded to the stimulation of visual field around the lesion. In their study, neurones at only 17% of the recording sites were unresponsive to the lesioned eye. A high proportion of responsive sites was also reported by Gilbert & Wiesel (1992), who used laser lesions in corresponding points of the two retinae. The contrast between these previous observations and those reported here is even more marked if one considers that the former studies employed lesions that caused deactivation of sectors of V1 up to 8–9 mm in diameter, while the representation of the monocular crescent in V1 is only 1–2 mm wide. In the central representation, lesion projection zones 2 mm in width are almost completely filled in by adjacent portions of the retina (Schmid *et al.* 1993). Thus, our results indicate that a region of cortex deprived of its normal inputs *will not necessarily be invaded by any normal inputs to* (*or from*) *adjacent cortex*. This suggests the existence of natural boundaries that limit the reorganization of adult topographic maps. While massive reorganization is possible in the range of 9–10 mm within the domains of the same eye in binocular V1, or across the entire forelimb representation in somatosensory cortex (Pons, Garraghty, Ommaya, Kaas, Taub & Mishkin, 1991), the degree to which the ipsilateral eye overtakes the 2 mm of the original monocular crescent representation is far more limited.

Although reorganization of the map in the LGN cannot explain all plastic effects that are observed in the adult visual cortex, due to its delayed time course (Eysel *et al.* 1980), it is possible that changes in this subcortical structure contribute to the limited plasticity we observed in the splenial sulcus. The magnification factor of the crescent in the LGN is so low that changes in topographic organization sufficient to bring about the changes we observed in V1 could have been missed between penetrations by previous studies. However, the progress of changes in spontaneous activity in the layers of the LGN affected by optic disc photocoagulation suggests that the activity of the reorganized peripheral V1 is not simply a reflection of the LGN. In the LGN, unlike in V1, there is an initial decrease in spontaneous activity, followed by recovery to near-normal levels after 10 weeks survival (Eysel, 1979).

Another indication of differences between regions of cortex is the absence of shifts in receptive field position in the deprived crescent sector immediately after monocular enucleation or deactivation. Such immediate effects of deafferentation were initially described in somatosensory cortex (e.g. Merzenich *et al.* 1983; Calford & Tweedale, 1988, 1991). In monkey area V1, Gilbert & Wiesel (1992) noticed a reorganization of receptive fields that originally represented the border of retinal lesions immediately after this treatment. More recently, we observed that extensive reorganization also occurs in V1 in the cat within 3–6 h of monocular retinal lesions (Schmid *et al.* 1994). In contrast in the present data, recordings extending up to 12 h after enucleation or deactivation revealed no evidence for short-term reorganization within the deprived crescent sector.

The determination of the reason for these different responses of the same cortical area to destruction of its normal afferents may provide information about the mechanism of retinotopic plasticity in adult animals. One possible reason for the limited capacity of reorganization of the monocular crescent representation is that activation by one eye would have to 'cross the border' and invade a territory that is normally the exclusive domain of the other eye. In cats, the horizontal intrinsic connections of V1 do not usually respect boundaries of ocular dominance columns; this only occurs if the animal is raised with strabismus (Löwel & Singer, 1992). The organization of intrinsic horizontal connections of cat V1 is well described in the central vision representation (e.g. Gilbert & Wiesel, 1983), but it is not known whether the intrinsic connections of peripheral V1 are similarly organized. The possibility thus exists that intrinsic connections of cells dominated by the ipsilateral eye avoid the monocular–binocular border. In this case, this border would effectively be a natural barrier to retinotopic reorganization, unless sprouting of new connections occurred.

In previous work, we demonstrated that retinotopic reorganization is eye-specific: in the central representation, it is possible to obtain displacement of receptive fields of one eye without disturbing the normal topography of the representation of the other eye (Schmid *et al.* 1993). In addition, the present results suggest that a retinotopic shift in adult visual cortex maps may be more difficult if ocular dominance borders have to be crossed. The possibility of reorganization within the domains of one eye poses difficulties for explanations based on sprouting of horizontal connections, as these would have to be highly specific. In the present study we observed weak cellular responses 2–3 days after treatment. These were scattered along the deprived crescent representation and were not restricted to the monocular–binocular border. In addition, we found no change in the spatial distribution or in the

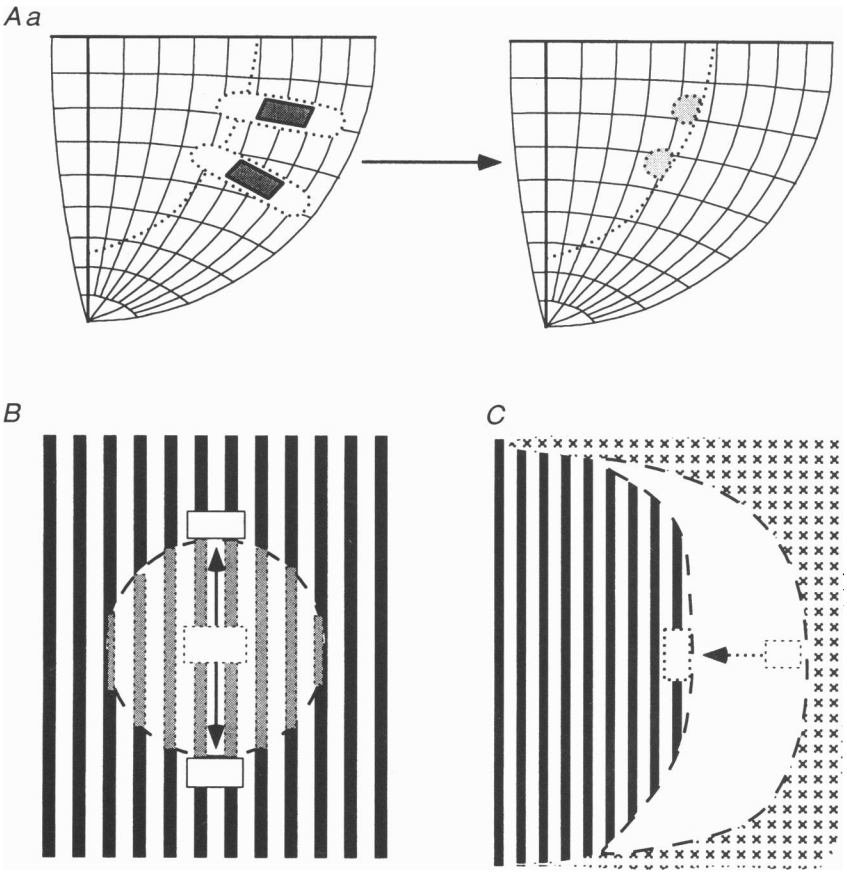


**Figure 10. Origin of reorganized receptive fields**

*A*, origin of reorganized receptive fields in the deprived peripheral sector based on unmasking and potentiation of active surrounds of receptive fields. *Aa*, two hypothetical receptive fields in monocular V1 (dark grey), with subthreshold, excitatory regions (dotted and dashed outlines). *Ab*, after inactivation of the contralateral eye, the active surround that originally carried subthreshold excitation from the ipsilateral eye, may potentiate and become responsive by itself. *B*, interpolation of receptive field responses to oriented contours within the cortical representation of retinal lesions is still possible if the neurone refers to portions of the visual field surrounding the scotoma. This diagram shows a portion of the visual field centred on a retinal lesion (dashed circle), in which a pattern of vertical stripes is present. The dotted rectangle is the original receptive field position in the cortex and the arrows indicate the receptive field displacement to either one or both borders of the lesion. By referring to receptive fields located around the perimeter of the lesion, the nervous system could still treat the lines as continuous (grey) even though there are no photoreceptors covering this portion of the retina. *C*, in the case of the monocular crescent this is not possible as this scotoma is bounded by portions of the space that are not served by the retina (crosses). Whatever the displacement of the receptive field into the binocular field, the activation of the new receptive field cannot interpolate continuity of contours across the scotoma.

proportion of sites with responsive neurones for periods of up to 16 months after lesioning. A gradual sprouting of axonal terminations is unlikely to explain this rapidly attained steady state.

We found no evidence for immediate rearrangement of the map. Thus, simple unmasking of previously existing, but inhibited, thalamocortical or corticocortical connections is also unlikely to explain our results. The possibility exists, however, that the manifestation of displaced receptive fields also depends on use-dependent potentiation of these previously existing synapses. This may be why we observed no displaced receptive fields immediately after inactivation, but were able to observe these responses after 2 days of normal visual stimulation. It is possible that the appearance of 'new' receptive fields along the temporal border of the binocular field merely reflects 'surfacing' of previously subthreshold flanks of these receptive fields (Fig. 10*A*). The existence of excitatory, subthreshold regions ('active surrounds') around a receptive field of the neurone was demonstrated in monkey V1 by masking the receptive field and its immediate surround (Fiorani, Rosa, Gattass & Rocha-Miranda, 1992). In the cat, enlargement of the excitatory receptive field by masking seems to depend on conditioning by repetitive stimulation of the surround (Pettet & Gilbert, 1992), again pointing to a use-dependent potentiation of synaptic strength. Some neurones of layer 6 in normal cat and monkey V1 form a subpopulation with large and elongated receptive fields (Gilbert, 1977), and these could be associated with the fact that the probability of finding responsive sites in the reorganized crescent is highest in the infragranular layers. Moreover, at least in monkey, neurones with active surrounds are seldom observed in the supragranular layers (Fiorani *et al.* 1992).

Another possibility is that the reorganization of retinotopic maps depends on projections from areas of the extrastriate cortex to V1. In the cat, all these areas have representations of central vision, but representations of peripheral vision are rare (Tusa, Palmer & Rosenquist, 1981). Thus, if the contribution of these areas was critical to the establishment of the new excitatory portions of the receptive fields, one could expect that the potential for shifts in the retinotopic map would decrease as one moved from the central into the peripheral representation of V1. Again, there is a dearth of data on connections of the peripheral representation of V1, in comparison with the central representations. Nonetheless, this hypothesis could be tested by observing the effect of deactivating extrastriate areas on the postlesion reorganization of the V1 retinotopic map.

Finally, there is the possibility that the shape and extent of the scotoma itself may pose problems for the mechanisms responsible for the retinotopic plasticity in V1. Retinotopic reorganization may play a role in establishing the image continuity across scotomata. If a restricted lesion occurs in the central or paracentral retina, humans have no conscious perception of its location even if they close the opposite eye, as the patterns that project to the surrounding retina 'fill in' the scotoma. This is the well-known completion phenomenon that also explains why we are unaware of the presence of the optic disc blind spots (Helmholtz, 1911). Fiorani and collaborators (1992) proposed that this phenomenon depends on neurones in V1 with split fields that span the scotoma and neurones that detect the coincidence of oriented patterns across the gap in the visual field (Fig. 10*B*). Neither of these receptive field classes would be functional in the case of the monocular crescent scotoma, because of its location at the edge of the visual field and its elongated shape (Fig. 10*C*). Thus, if the stabilization of 'new' receptive fields around the edge of a scotoma relies on an activity-dependent (Hebbian) mechanism and if the optimal stimuli for neurones in reorganized sectors are patterns that span the gaps in the visual field, then one could expect a much lesser degree of reorganization in peripheral V1, as compared with central V1.

### Splenial visual area

In the course of these experiments, we had the opportunity of recording from the visual area situated lateral to V1 in the splenial sulcus. This is, to our knowledge, the first confirmation of the results of Kalia & Whitteridge (1973) in cats. Recordings and tracer injections in mustelid carnivores (e.g. McConnell & Le Vay, 1986), rodents (Montero, Rojas & Torrealba, 1973) and flying foxes (M. G. P. Rosa & L. M. Schmid, unpublished observations) also suggested the existence of visually responsive cortex located between the peripheral representation of V1 and the limbic cortex; thus, this area may be a more general mammalian characteristic. At least in the cat, this area seems to be unique in that it does not emphasize the central visual field representation.

### Conclusion

Destruction of the projections that represent the monocular crescent to cat area V1 does not, as initially expected, result in a rapid and extensive reorganization of the visual representation in the splenial sulcus. Thus, the potential for plasticity of adult sensory maps in the cortex depends on the specific conditions of the region under study. While the somatosensory and auditory cortices together with the central representation of visual cortex have the potential for extensive reorganization in adults, a piece of cortex as small as the monocular crescent representation may remain largely silent. This paradox may result from the lack of intrinsic or extrinsic corticocortical connections that are critical for the establishment of reorganized maps, or may suggest an activity-dependent mechanism for establishment of retinotopic shifts. The special characteristics of the monocular crescent model, such as the constancy of its anatomical location and its histological distinctness may, in the future, provide an adequate test for some of these hypotheses.

BISHOP, P. O., KOZAK, W. & VAKKUR, G. J. (1962). Some quantitative aspects of the cat's eye: axis and plane of references, visual field co-ordinates and optics. *Journal of Physiology* **163**, 466–502.

CALFORD, M. B. & TWEEDALE, R. (1988). Immediate and chronic changes in responses of somatosensory cortex in adult flying-fox after digit amputation. *Nature* **332**, 446–448.

CALFORD, M. B. & TWEEDALE, R. (1991). Acute changes in cutaneous receptive fields in primary somatosensory cortex after digit denervation in adult flying fox. *Journal of Neurophysiology* **65**, 178–187.

CLARKE, R. J., DATSKOVSKY, B. W., GRIGONIS, A. M. & HAZEL MURPHY, E. (1992). The effects of monocular enucleation on visual topography in area 17 in the rabbit. *Experimental Brain Research* **91**, 303–310.

DANIEL, P. M. & WHITTERIDGE, D. (1961). The representation of the visual field on the cerebral cortex in monkeys. *Journal of Physiology* **159**, 203–221.

EYSEL, U. T. (1979). Maintained activity, excitation and inhibition of lateral geniculate neurons after monocular deafferentation in the adult cat. *Brain Research* **166**, 259–271.

EYSEL, U. T., GONZALEZ-AGUILAR, F. & MAYER, U. (1980). A functional sign of reorganization in the visual system of adult cats: lateral geniculate neurons with displaced receptive fields after lesions of the nasal retina. *Brain Research* **181**, 285–300.

EYSEL, U. T., GONZALEZ-AGUILAR, F. & MAYER, U. (1981). Time-dependent decrease in the extent of visual deafferentation in the lateral geniculate nucleus of adult cats with small retinal lesions. *Experimental Brain Research* **41**, 256–263.

FIORANI, M. JR, ROSA, M. G. P., GATTASS, R. & ROCHA-MIRANDA, C. E. (1992). Dynamic surrounds of receptive fields in primate striate cortex: A physiological basis for perceptual completion? *Proceedings of the National Academy of Sciences of the USA* **89**, 8547–8551.

GILBERT, C. D. (1977). Laminar differences in receptive field properties of cells in cat primary visual cortex. *Journal of Physiology* **268**, 381–421.

GILBERT, C. D. & WIESEL, T. N. (1983). Clustered intrinsic connections in cat visual cortex. *Journal of Neuroscience* **3**, 1116–1133.

GILBERT, C. D. & WIESEL, T. N. (1992). Receptive field dynamics in adult primary visual cortex. *Nature* **356**, 150–152.

HEINEN, S. J. & SKAVENSKI, A. A. (1991). Recovery of visual responses in foveal V1 neurons following bilateral foveal lesions in adult monkey. *Experimental Brain Research* **83**, 670–674.

HELMHOLTZ, H. (1911). *Treatise on Physiological Optics*, vol. 2 (translation of the 3rd German edition). Optical Society of America, Washington.

HUGHES, A. (1976). A supplement to the cat schematic eye. *Vision Research* **16**, 149–154.

KAAS, J. H., KRUBITZER, L. A., CHINO, Y. M., LANGSTON, A. L., POLLEY, E. H. & BLAIR, N. (1990). Reorganization of retinotopic cortical maps in adult mammals after lesions of the retina. *Science* **248**, 229–231.

KALIA, M. & WHITTERIDGE, D. (1973). The visual areas in the splenial sulcus of the cat. *Journal of Physiology* **232**, 275–283.

LÖWEL, S. & SINGER, W. (1987). The pattern of ocular dominance columns in flat-mounts of the cat visual cortex. *Experimental Brain Research* **68**, 661–666.

LÖWEL, S. & SINGER, W. (1992). Selection of intrinsic horizontal connections in the visual cortex by correlated neuronal activity. *Science* **255**, 209–212.

MCCONNELL, S. K. & LE VAY, S. (1986). Anatomical organization of the visual system of the mink, *Mustela vison*. *Journal of Comparative Neurology* **250**, 109–132.

MERZENICH, M. M., KAAS, J. H., WALL, J. T., SUR, M., NELSON, R. J. & FELLEMAN, D. J. (1983). Progression of change following median nerve section in the cortical representation of the hand in areas 3b and 1 in adult owl and squirrel monkeys. *Neuroscience* **10**, 639–665.

MERZENICH, M. M., NELSON, R. J., STRYKER, M. P., CYNADER, M. S., SCHOPPMANN, A. & ZOOK, J. M. (1984). Somatosensory cortical map changes following digit amputation in adult monkeys. *Journal of Comparative Neurology* **224**, 591–605.

MONTERO, V. M., ROJAS, A. & TORREALBA, F. (1973). Retinotopic organization of striate and prestriate visual cortex in the albino rat. *Brain Research* **53**, 202–207.

NIKARA, T., BISHOP, P. O. & PETTIGREW, J. D. (1968). Analysis of retinal correspondence by studying receptive fields of binocular single units in cat striate cortex. *Experimental Brain Research* **6**, 353–372.

PETTET, M. W. & GILBERT, C. D. (1992). Dynamic changes in receptive field size in cat primary visual cortex. *Proceedings of the National Academy of Sciences of the USA* **89**, 8366–8370.

PETTIGREW, J. D., COOPER, M. L. & BLASDEL, G. G. (1979). Improved use of tapetal reflection for eye-position monitoring. *Investigative Ophthalmology and Visual Science* **18**, 490–495.

PONS, T. P., GARRAGHTY, P. E., OMMAYA, A. K., KAAS, J. H., TAUB, E. & MISHKIN, M. (1991). Massive cortical reorganization after sensory deafferentation in adult macaques. *Science* **252**, 1857–1860.

RAJAN, R., IRVINE, D. R. F., WISE, L. Z. & HEIL, P. (1993). Effects of unilateral partial cochlear lesions in adult cats on the representation of lesioned and unlesioned cochleas in primary auditory cortex. *Journal of Comparative Neurology* **338**, 17–49.

RASMUSSON, D. D. (1982). Reorganization of racoon somatosensory cortex following removal of the fifth digit. *Journal of Comparative Neurology* **205**, 313–316.

RECANZONE, G. H., MERZENICH, M. M., JENKINS, W. M., GRAJSKI, K. A. & DINSE, H. R. (1992). Topographic reorganization of the hand representation in cortical area 3b of owl monkeys trained in a frequency-discrimination task. *Journal of Neurophysiology* **67**, 1031–1056.

ROBINSON, S. R., SUNG, L., DREHER, B. & TAYLOR, I. (1990). The distribution of ipsilaterally projecting retinal ganglion cells in adult and neonatal rabbits. *Proceedings of the Australian Physiological and Pharmacological Society* **21**, 28P.

ROSA, M. G. P., GATTASS, R., FIORANI, M. JR & SOARES, J. G. M. (1992). Laminar, columnar and topographic aspects of ocular dominance in the primary visual cortex of *Cebus* monkeys. *Experimental Brain Research* **88**, 249–264.

SCHMID, L. M., CALFORD, M. B. & ROSA, M. G. P. (1994). Reorganization in the primary visual cortex (V1) of adult cats occurs immediately following monocular retinal lesions. *Investigative Ophthalmology and Visual Science* **35**, 1774.

SCHMID, L. M., ROSA, M. G. P. & CALFORD, M. B. (1993). Reorganizational plasticity in the primary visual cortex (V1) of adult cats occurs following monocular retinal lesions. *Society for Neuroscience Abstracts* **19**, 1801.

SOUSA, A. P. B., GATTASS, R., HOKOÇ, J. N. & OSWALDO-CRUZ, E. (1978). The visual field of the opossum. In *Opossum Neurobiology*, ed. ROCHA-MIRANDA, C. E. & LENT, R., pp. 51–65. Academia Brasileira de Ciências, Rio de Janeiro.

Tusa, R. J., Palmer, L. A. & Rosenquist, A. C. (1978). The retinotopic organization of area 17 (striate cortex) in the cat. *Journal of Comparative Neurology* **177**, 213–236.

Tusa, R. J., Palmer, L. A. & Rosenquist, A. C. (1981). Multiple cortical visual areas: Visual field topography in the cat. In *Cortical Sensory Organization*, vol 2, *Multiple Visual Areas*, ed. Woolsey, C. N., pp. 1–31. Humana Press, Clifton, NJ, USA.

Weinberger, N. M., Hopkins, W. & Diamond, D. M. (1984). Physiological plasticity of single neurons in auditory cortex of the cat during acquisition of the pupillary conditioned response: I. Primary field (AI). *Behavioral Neuroscience* **98**, 171–188.

Wong-Riley, M. (1979). Changes in the visual system of monocularly sutured or enucleated cats demonstrable with cytochrome oxidase histochemistry. *Brain Research* **171**, 111–128.

## Acknowledgements

The authors would like to thank Ms Rowan Tweedale for comments that improved the manuscript, Ms Rita Collins for the histological work and Mr Guy Elston for photography. The acquisition of the argon laser was made possible by the Clive and Vera Ramaciotti Foundation. We were supported by a Special Research Centre grant from the Australian Research Council and by research grants from the National Health and Medical Research Council.

*Received 1 March 1994; accepted 29 June 1994.*