Responses of Neurons in the Middle Temporal Visual Area After Long-Standing Lesions of the Primary Visual Cortex in Adult New World Monkeys

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The retinotopic organization of the middle temporal visual area (MT) was determined in six adult owl monkeys and one adult marmoset 69 d to 10 months after lesions of the dorsolateral primary visual cortex (V1). The lesions removed were limited to extensive parts of the representation of the lower visual quadrant in V1. Microelectrodes were used to record from neurons at numerous sites in MT to determine whether parts of MT normally devoted to the lower visual quadrant (1) were unresponsive to visual stimuli, (2) acquired responsiveness to inputs from intact portions of V1, or (3) became responsive to some other visually driven input such as a relay from the superior colliculus via the pulvinar to MT. All monkeys (n = 6) with moderate to moderately large lesions had unresponsive portions of MT even after 10 months of recovery. These unresponsive regions were retinotopically equivalent to the removed parts of V1 in normal animals. Thus, there was no evidence for an alternative source of activation. In addition, these results indicate that any retinotopic reorganization of MT based on inputs from intact portions of V1 was not extensive, yet neurons near the margins of responsive cortex may have acquired new receptive fields, and the smallest 5° lesion of V1 failed to produce an unresponsive zone. Deprived portions of MT were not remarkably changed in histological appearance in cytochrome oxidase, Nissl, and *Wisteria floribunda* agglutinin preparations. Nevertheless, some reduction in myelin staining and other histological changes were suggested. We conclude that MT is highly dependent on V1 for activation in these monkeys, and alternative sources do not become effective over months when normal activation is absent. Additionally, remaining V1 inputs have only a limited capacity to expand their activation territory into deprived portions of MT.

Key words: **area 17; V1; extrastriate cortex; lesion; MT; reorganization; plasticity**

Introduction

In the present study, we sought to determine the effects of long-standing lesions of the primary visual cortex (V1) on the responsiveness of neurons in the middle temporal visual area (MT) of adult monkeys. Extrastriate visual areas were long thought to be totally dependent on V1 for activation, because lesions of V1 in humans produce a scotoma for visual stimuli that has been referred to as cortical blindness (Weiskrantz, 1986). When it appeared that some individuals could perform simple visual tasks without awareness of the stimuli in parts of the field made blind by cortical lesions (Weiskrantz et al., 1974; Weiskrantz, 1986), it became important to consider the possibility that some regions of extrastriate cortex can be activated by sources of visual input other than V1.

In some mammals, including tree shrews (Killackey et al., 1971) and cats (Winans, 1967; Doty, 1971), considerable vision is preserved after V1 lesions, and much of extrastriate cortex remains responsive to visual stimuli as a result of significant extrastriate projections from the lateral geniculate nucleus (LGN) and possibly from regions of the pulvinar with visual inputs from the superior colliculus (for review, see Funk and Rosa, 1998). However, in primates, nearly all of the projections of the LGN terminate in V1 (for review, see Stepniewska et al., 1999), and much or most of extrastriate cortex appears to depend completely on V1 for activation. In early landmark studies in macaque monkeys, the responsiveness of neurons in inferotemporal cortex (Rocha-Miranda et al., 1975) and V2 (Schiller and Malpeli, 1977; Girard and Bullier, 1989) to visual stimuli was found to depend on V1. Later, Girard et al. (1991a,b) provided evidence that the responsiveness of neurons in the third visual area, V3, and the fourth visual area, V4 (dorsolateral area, DL), also depend on V1.

Although the results of these studies suggest that neurons in much of extrastriate cortex depend on V1 for activation in macaque monkeys, there is evidence for two exceptional cortical areas. Girard et al. (1991b) found that some neurons in V3a (dorsomedial area, DM) continued to respond to visual stimuli after the relevant portion of V1 was inactivated by cooling, and Rodman et al. (1989) found that some neurons in MT retained responsiveness after lesions of V1 (for related results, see Girard et al., 1992). Because neurons in MT failed to respond to visual stimuli in monkeys with both V1 and superior colliculus (SC) lesions, a relay of visual information from the SC to the pulvinar and then to extrastriate cortex was postulated as the source of the visual activation of MT in the absence of V1 (Rodman et al., 1990). More recently, Rosa et al. (2000) reported that V1 lesions in New World marmoset monkeys failed to completely deactivate neurons in MT.

The present study was motivated by the quite different results that were obtained from MT after V1 lesions in owl monkeys (Kaas and Krubitzer, 1992). Owl monkeys are New World mon-

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keys with smaller brains and few brain fissures. Thus, MT is nearly completely exposed on the surface of the upper temporal lobe, where it can be systematically explored with microelectrodes for responsiveness after V1 lesions. Because both V1 and MT contain retinotopic representations of the contralateral visual hemifield, a lesion of part of the V1 representation would deprive neurons of this source of visual activation in a retinotopically corresponding part of MT. When the part of V1 representing the lower visual quadrant in owl monkeys was ablated, neurons in the corresponding part of MT were totally unresponsive to visual stimuli, whereas neurons in the nondeprived portion of MT were normally responsive. Although some neurons along the margin of the deprived zone in MT may have acquired slightly displaced receptive fields mediated by remaining V1 inputs, there was no evidence for a relay of visual activation to MT from the superior colliculus. Comparable results were obtained by Maunsell et al. (1990) in macaque monkeys. When V1 was deactivated by blocking activity in the LGN, there was a complete lack of responsiveness of MT neurons.

To explain these differing results, it may be useful to consider species differences, recording densities and conditions, and other experimental procedures and to collect more data. Collectively, the studies seem to indicate that the responsiveness of neurons in MT to visual stimuli is at least considerably reduced after V1 loss. Possibly a preserved responsiveness to visual stimuli via a superior colliculus relay is expressed under some conditions or in some species and not others. Although most of the recordings in the above studies were immediately obtained after a V1 lesion or inactivation, some recordings by Rodman et al. (1989) were obtained after weeks of recovery, during which sources of weak activation could have been potentiated. A long postlesion recovery period could lead to expression of a previously unexpressed source of activation from the superior colliculus (or another source outside V1) or possibly expansion of the portion of MT activated by intact parts of V1. Partial lesions of the retina deactivate neurons in part of V1, but these neurons recover responsiveness to visual stimuli over time (for review, see Kaas et al., 2001). After a partial loss of sensory afferents in the somatosensory system of monkeys, remaining inputs typically expand their territories of activation in cortex, and previously ineffective pathways acquire activation strengths above threshold (Jain et al., 1997, 2001). In the present study, we lesioned part of V1 in six adult owl monkeys and one adult marmoset and recorded from MT after ≥69 d of recovery.

Materials and Methods

The effects of removing a portion of V1 on the responsiveness of neurons in MT were investigated in six adult owl monkeys and one adult marmoset monkey. All surgical procedures were performed in accordance with the *Guidelines for the Care and Use of Laboratory Animals* published by the National Institutes of Health (publication 86-23) and the Vanderbilt University Animal Care and Use Committee.

V1 lesions. Under aseptic conditions, a part of V1 was removed by aspiration. For surgery, animals were anesthetized with either 2% isoflurane inhalant anesthetic or with ketamine hydrochloride (30 mg/kg, i.m.) and xylazine (0.5–1.0 mg/kg, i.m.), with supplementary doses administered as needed to maintain a surgical level of anesthesia. Animals were placed on a heating pad and secured in a stereotaxic apparatus. The dorsolateral occipital cortex was exposed, and the dura mater was cut and retracted to uncover visual cortex. Lesions were made by aspiration using cortical landmarks to restrict lesions to V1. The margins of the cavity created by the lesion were covered with Gelfoam. The retracted dura mater was laid back over the area, and the opening in the cranium was closed with a cap of dental cement. The skin incision was closed with sutures. Animals were placed in a recovery cage with soft food and water available *ad libitum*. Recovery was carefully monitored until the animals regained normal mobility and were eating, at which time they were returned to their home cage.

Lesion size varied across animals. In cases with smaller lesions, the tissue removed was limited to the dorsolateral surface of striate cortex and did not include significant portions of the less accessible upper visual field representation, located on the ventral surface of the hemisphere, the lower medial wall, and the lower bank of the calcarine fissure (Allman and Kaas, 1971b). In one owl monkey with a very small striate cortex lesion (98-78), the V1 ablation did not extend to the caudal pole but only included a strip of V1 near its border with V2 (see Fig. 3*A*). In monkeys with large V1 removals, the entire exposed dorsolateral surface of cortex was removed to the caudal pole, in addition to regions of ventrolateral V1, which includes the paracentral upper visual field representation. The largest lesions also encompassed parts of V1 devoted to more peripheral parts of the lower visual field, including segments of V1 folded into the calcarine fissure. Visuotopic positions of lesions were estimated from physiological maps. Other cortical areas were used as landmarks for alignment. For example, the narrowest part of V2 representing central vision is visible in flattened cortex sections stained for cytochrome oxidase (CO). The extent and visuotopic position of the V1 lesion were also confirmed in some cases by examining the distribution of cellular degeneration in the LGN (see Fig. 8*C*).

Recordings were made from MTs of six adult owl monkeys and one adult marmoset monkey. In five owl monkeys and one marmoset, the postlesion time between the V1 lesion surgery and the recording session ranged from 69 to 96 d. In addition, recordings were made in one owl monkey (00-54) 10 months after the striate cortex lesion.

Anesthesia. Four owl monkeys and one marmoset monkey were anesthetized for recording by injection of 30% urethane (125 mg/100 gm of body weight), supplemented as needed to maintain a surgical plane of anesthesia. This anesthesia was used because it has been used repeatedly in studies of the visual responsiveness of neurons in MT and other visual areas in owl monkeys (Allman and Kaas, 1971a; Kaas and Krubitzer, 1992). Recordings from one owl monkey (99-82) were obtained under gas anesthesia using halothane (0.8–2.0%) while ventilating the animal with a 2:1 mixture of nitrous oxide and oxygen, as in a study by Girard et al. (1992). Recordings were made from one additional owl monkey (00-54) under Sufenta anesthesia (12–15 $\mu g \cdot kg^{-1} \cdot hr^{-1}$; sufentanil citrate injection; Baxter Health Care Corporation, Deerfield, IL), as in studies by Rodman et al. (1989) and Rosa et al. (2000). The Sufenta was infused at a rate of 2.5–3.2 ml/hr in a mixture including vecuronium bromide (Norcuron; 0.1–0.2 $mg \cdot kg^{-1} \cdot hr^{-1}$), 50% dextrose, and lactated Ringer's solution. Anesthesia, paralysis, and hydration were maintained with this rate of infusion. The monkey was ventilated with a 3:1 mixture of nitrous oxide and oxygen. The end-tidal CO_2 was maintained between 3.5 and 4.0%. Anesthetic depth was monitored using measures of heart rate and blood pressure. Before recording, all animals were premedicated with dexamethasone (2 mg/kg) to prevent brain swelling and robinul (0.015 mg/kg) to aid respiration.

Electrophysiological recordings. Once fully anesthetized, the monkeys were positioned on a heating pad and fixed in a stereotaxic apparatus. An opening was made in the cranium over dorsal extrastriate cortex, and rongeurs were used to extend the opening laterally to expose the superior temporal sulcus (STS) and rostrally to expose the lateral sulcus. MT is located at the tip of the STS in owl monkeys, and MT in marmosets is just caudal to the lateral sulcus and dorsal to the STS. When the cranial opening was complete, it was enclosed by a dam of acrylic plastic. The dura mater was cut and retracted to reveal the cortex, and the surface of the cortex was coated with a layer of silicone fluid (dimethylpolysiloxane) to prevent drying. A high-resolution photograph of the exposed cortex, used for recording electrode penetration sites, was taken with a Cohu CCD camera (model 4910). The camera was connected to an Apple (Cupertino, CA) Macintosh G3 computer equipped with a frame-grabber card and running NIH Image software (version 1.62).

Cyclopentolate hydrochloride drops were used to dilate the pupil of the eye contralateral to the exposed cortex. The eye was then covered with a thin coating of silicone fluid to prevent drying. In owl monkey 00-54,

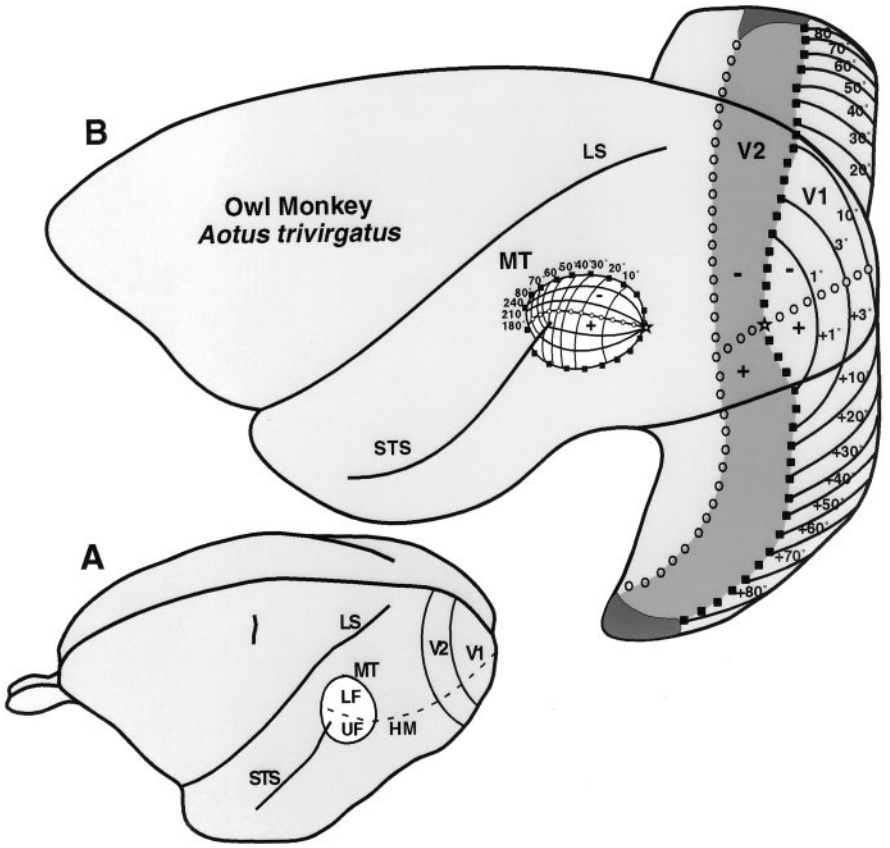

Figure 1. *A*, Schematic dorsolateral view of an owl monkey brain with selected cortical areas labeled. *LS*, Lateral sulcus; *UF*, upper field; *LF*, lower field; *HM*, horizontal meridian. B, Enlargement of an owl monkey brain with visuotopic maps of V1 and MT superimposed. V1 and V2 are unfolded to illustrate the layout of the visuotopic map. *Filled squares* indicate the representation of the vertical meridian on the border between V1 and V2. *Circles* indicate the representation of the horizontal meridian and stars indicate representations of central vision. Modified from Allman and Kaas (1971a,b).

the animal was paralyzed during recording, so it was not necessary to physically stabilize the eye contralateral to the exposed cortex. The animal remained in a modified stereotaxic apparatus, with the ear bars at the open end of the horizontal supports to allow unobstructed vision for the duration of the recording session. Owl monkeys and marmosets recorded without paralysis were in a stereotaxic apparatus for surgical preparation for recording but were removed for the duration of the recording session. In these animals, the eye contralateral to the exposed cortex was physically stabilized by suturing the sclera to a metal ring on a connecting rod, which was cemented to the acrylic dam on the skull. An additional metal rod cemented to the acrylic dam on the skull was fixed in an adjustable vise so the stereotaxic apparatus could be removed. With the stereotaxic apparatus removed, the animals had unobstructed views of the visual field. The stabilized eye was centered in a translucent plastic hemisphere, which served as a screen for presenting visual stimuli. The unfixed eye was covered, except when testing for binocular responses. Using a fiber-optic system, light was briefly shone into the fixed eye, so the position of the optic disk would be reflected on the hemisphere to use as a reference for the visuotopic map (Fernald and Chase, 1971).

Recordings were made with low-impedance (1.0–1.5MΩ) tungsten microelectrodes. Electrode penetrations were made in the presumptive location of MT, and within a single electrode penetration, responses were tested at 100–150 μm intervals throughout the depth of cortex. Most electrode penetrations represent three to six recording depths. Recordings were occasionally from single neurons but usually from clusters of neurons. Visual stimuli in the form of moving bars or spots of light were projected onto the translucent hemisphere with a handheld projector. MT neurons in normal owl monkeys respond best to thin bars, $<1°$ wide, when the bars are the approximate length of the receptive field and moving at a moderate speed in the preferred direction (Felleman and Kaas, 1984). Sites where neurons were found to be unresponsive to such stimuli were examined with a wider range of visual stimuli, including larger bars, slower or faster movement, and light flashes. Sites in unresponsive cortex were also examined at a larger number of recording depths. Neurons were judged to be responsive to visual stimuli when two or more of the investigators agreed that responses coincided with visual stimuli. Receptive fields were defined as the region where visual stimuli were effective in evoking responses, because these stimuli were repeatedly moved through and around the receptive field at various angles. The functional condition of the cortex and the status of the electrodes and recording equipment were evaluated throughout the experiment by alternating between responsive and unresponsive regions of MT. In cases in which cells across a large portion of MT were unresponsive, the electrode and general condition of the cortex were tested by alternately recording from MT and then auditory or somatosensory cortex.

Histology. After all recording was complete, electrolytic lesions were made to mark important electrode penetration sites. Animals were injected with an overdose of sodium pentobarbital and usually perfused with 0.9% PBS, followed by 2% paraformaldehyde in phosphate buffer and 2% paraformaldehyde in phosphate buffer with 10% sucrose. After fixation, brains were immediately removed from the skull into a 30% sucrose solution.

In three owl monkeys and one marmoset (99-9, 99-10, 99-82, and 99-24), both the recorded hemisphere and the opposite control hemisphere were flattened. The cortex was separated from the brainstem and bisected in preparation for flattening. The pia mater was removed from the cortical surface; sulci were gently opened; and underlying white matter was thinned. One cut was made to open the calcarine fissure, and one small cut was made to partially open the lateral sulcus. Cortices were then flattened between two glass plates and immersed in a 30% sucrose solution for ~24 hr. The cortex was then frozen and sectioned parallel to the cortical surface at a thickness of 40 μm. In the fourth owl monkey (98-103), only the recorded hemisphere was flattened and sectioned, and in the fifth owl monkey (98-78), the brain was left unperfused and intact, immersion-fixed in 4% paraformaldehyde, and sectioned in the coronal plane. In the final owl monkey (00-54), the brain was perfused and cut in the coronal plane. For all cases, the thalamus was cut into 40-μm-thick sections in the coronal plane. In all cases, one set of cortex sections was stained for myelin (Gallyas, 1979), and another set was stained for CO (Wong-Riley, 1979) to reveal areal boundaries and the extent of V1 lesions. Sections throughout the thalamus were divided into three series and stained for myelin, CO, or Nissl substance.

In owl monkey 00-54, sets of coronal sections were also processed for immunocytochemistry for *Wisteria floribunda* agglutinin (WFA; L-1766; Sigma, St. Louis, MO) according to the method of Preuss et al. (1998). Briefly, sections were rinsed in 0.05 M Tris-buffered saline (TBS), blocked for 2 hr in TBS with sheep serum and 0.1% Triton X-100, and incubated overnight in primary antibody solution (0.005 mg/ml). After incubation, sections were rinsed in 0.05 M TBS, incubated for 1 hr in avidin–biotin–peroxidase solution (PK-6100 kit; Vector Laboratories, Burlingame, CA), and visualized with a DAB reaction. Label was intensified by adding 0.02 M imidazole to the DAB solution, which resulted in a light brown reaction product. Sections were mounted out of dilute 0.005 M Tris

buffer. Some sets of sections were counterstained for Nissl substance (thionin purple) to better reveal laminar patterns of WFA distribution.

Results

Microelectrodes were used to record from neurons in MT after recoveries from partial lesions of V1 in six adult owl monkeys and one adult marmoset. Recovery times varied from 69 d to 10 months, and lesion sizes varied from ~10% of V1 to greater than half. The important, relevant variable was clearly the size of the lesion. Small lesions left most of V1 intact and capable of activating large portions or all of MT. Larger lesions deactivated portions of MT so that neurons were unresponsive to visual stimuli and altered other parts of MT so that neurons were more difficult to activate and may have had displaced receptive fields. In other parts of MT, neurons remained essentially normal, with normal response characteristics and receptive field locations. As expected, the long-standing lesions of V1 produced severe retrograde degeneration in the affected parts of the lateral geniculate nucleus of the thalamus. However, the parts of MT that were deprived of their normal inputs from V1 showed only slight changes, if any, in histological appearance. Thus, results are presented in two sections. First, the electrophysiological results are described, starting from monkeys with the smaller lesions and proceeding to those with the larger lesions and more alterations in MT. Second, the histology of MT after these long-standing lesions of V1 is described.

As an aid to interpreting the physiological results, retinotopic maps are presented for MT, V1, and V2 (Fig. 1). The V1 map is based on that of Allman and Kaas (1971b). The retinotopy of the V2 map is not shown in detail (but see Allman and Kaas, 1974), because lesions did not intrude on V2. The MT map is based on that of Allman and Kaas (1971a). However, our recordings suggest that proportionately more of MT (approximately half) is devoted to the first 10° of central vision. The maps can be used to estimate the portion of the representation of the visual hemifield in V1 that was removed by each lesion, thus the portion of MT that was deprived of normal activation from V1.

Lesions and recording sites were localized in brain sections cut parallel to the surface of flattened cortex. This preparation has become common for studies of connection patterns of cortex (Kaas and Morel, 1993). Sections of flattened cortex from the intact hemisphere of an experimental monkey are shown in Figure 2. In such sections processed for myelin (Fig. 2*A*) or cytochrome oxidase (Fig. 2*B*), areas MT, V1, and often V2 can easily be delimited by their architectonic features. In the experimental hemispheres, microlesions marked reference locations in MT (Fig. 2*C*), and often sites were located relative to these reference marks and brain surface features (the end of the superior temporal sulcus). The lesion in V1 was simply the missing portion. The portions of lesions on the dorsolateral surface of the hemisphere were nicely revealed by this procedure, and the relationship of the lesions to the dorsolateral margin of V1 and V2 was clear. Because the unfolding process repositioned portions of V1 in the calcarine fissure, the lesions no longer were surrounded by intact cortex.

Microelectrode recordings from MT after V1 lesions

Owl monkey 98-78 received a small lesion of dorsolateral V1, removing approximately one-sixth of V1, which represents much of the central 8° of vision in the lower visual quadrant (Fig. 3*A*). Because the lesion would deprive only part of the lower visual quadrant representation in MT, our recording sites were concentrated in medial MT (Fig. 3*A*). After 77 d of recovery, neurons at locations throughout the recorded portions of MT had normal

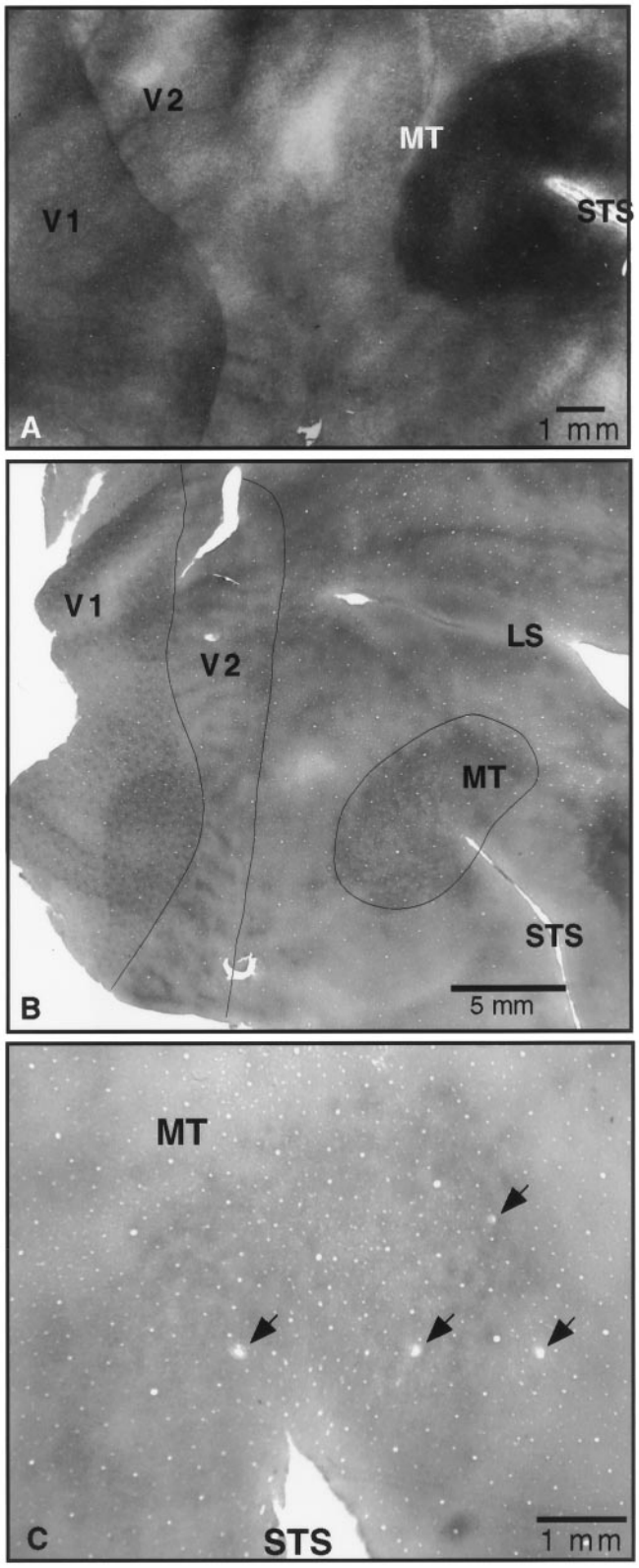

Figure 2. *A*, Section of flattened cortex stained for myelinated fibers. Areal borders of MT can be more readily defined in myelin-stained sections. *B*, Section of flattened cortex from owl monkey 99-9 stained for CO. *Blobs* in V1, *stripes* in V2, and *patches* in MT help define areal boundaries. *C*, Enlargement of flattened section of area MT, stained for CO. *Arrows* mark electrolytic lesions, placed at specific recording locations at the end of the recording session. Lesions are used for MT reconstruction and to confirm that recording sites are within MT. Abbreviations are as in Figure 1.

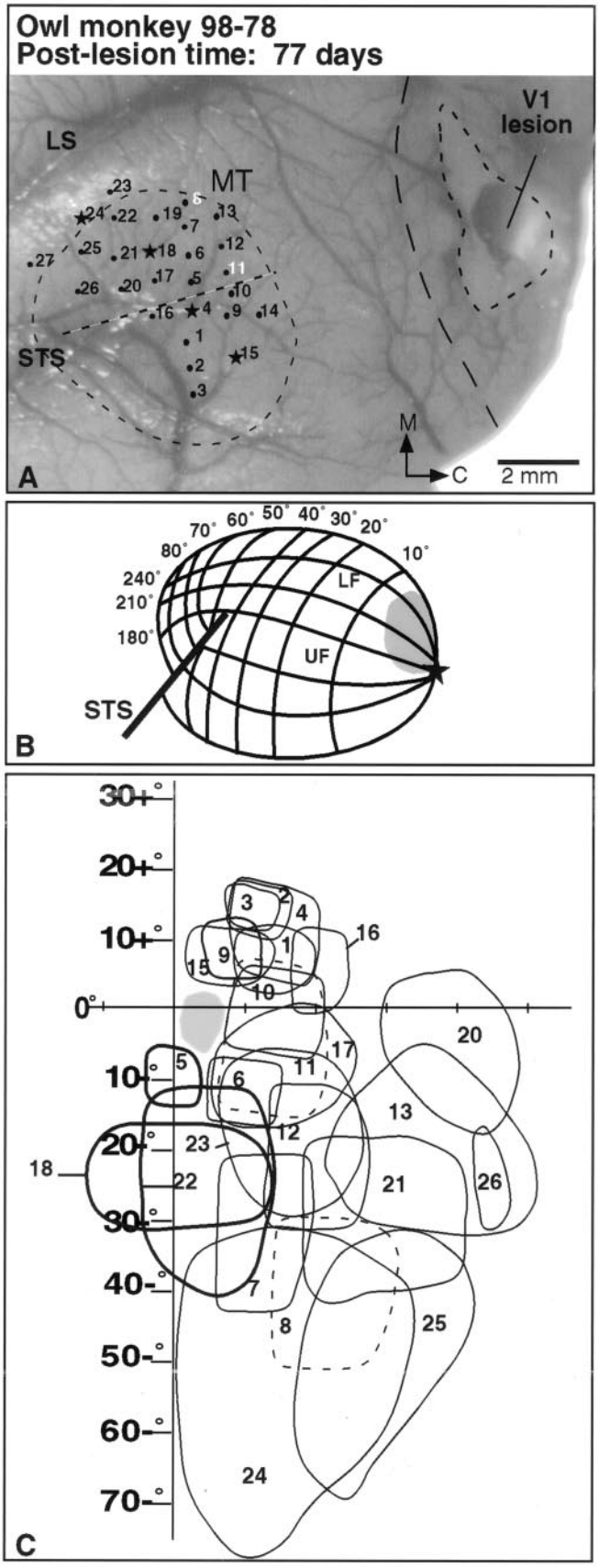

Figure 3. V1 lesion and physiological mapping data from owl monkey 98-78. *A*, Photograph of the unfixed brain showing the V1 lesion. *Black circles* mark electrode penetrations sites in MT. Those sites with *white numbers* indicate locations of transiently responsive neurons. *B*, *Gray shading* shows the small part of MT expected to be affected by the V1 lesion on a schematic representation of the visuotopic map in MT. *C*, Receptive fields of MT neurons 77 d after V1 lesion. *Gray shading* indicates the approximate area of visual loss expected from the cortical lesion. No unresponsive zones were found in MT. Two receptive fields bounded by *dashed lines* correspond to transiently responsive neurons at penetrations *8* and *11*. These RFs have less definite borders. Other abbreviations are as in Figure 1.

receptive fields and response characteristics. At 96 recording sites in 21 electrode penetrations in MT, consistent vigorous responses were obtained for moving visual stimuli. These neurons had the characteristically large receptive fields of MT neurons (Fig. 3*C*) (Allman and Kaas, 1971a; Kaas and Krubitzer, 1992). Neurons were selective for direction of movement, as reported for normal owl monkeys (Allman and Kaas, 1971a; Zeki, 1980; Felleman and Kaas, 1984; Allman et al., 1985). Neurons at only two locations (8 and 11) displayed weak and transient responses (Fig. 3*C*), although they had receptive fields in locations corresponding to intact portions of V1. Possibly, these neurons were partially deprived or simply were less responsive for unknown reasons. Neurons at recording site 11, but not 8, would likely be in altered cortex. Neurons at four other locations (12–14, and 19) responded less robustly than others, and neurons at sites 12 and 13, but not 14 and 19, were likely in altered cortex. No neurons had receptive fields that included the 5–8° of the lower visual quadrant that was missing from V1. Additional recordings were made in the location of the DM and in one location in intact V1 along the lower margin of the lesion. Normal responses to visual stimuli were recorded in both locations.

In summary, a lesion restricted to the part of V1 representing the central 8° of vision of the lower visual quadrant had no clear impact on the responsiveness of neurons in V1 to visual stimuli, although neurons at two or more sites may have had reduced responsiveness. No neurons had receptive fields that related to the missing portion of V1. Because recorded neurons at sites 11–13 were in locations that normally have more central receptive fields, possibly these neurons had been deprived of normal V1 inputs and had acquired new, displaced receptive fields.

Recordings after larger lesions

Owl monkey 98-103 received a larger lesion that included most of dorsolateral V1, thereby removing the central ≥10° of the representation of the lower visual quadrant (Fig. 4*A*). The lesion reached the V1–V2 border without invading V2, because the banding pattern of V2 remained intact along the rostral margin of the lesion. Because the unfolding of cortex involved a disjunction of less central portions of V1, the full extent of the lesion into the representation was not as obvious, and it likely was more extensive than shown.

The most obvious difference in results from this case and the one with the smaller lesion was that neurons at several recording sites were completely unresponsive to visual stimuli or nearly so. Neurons at several successive recording sites, through the depth of cortex, failed to respond in electrode penetrations 2, 3, 8, 9, and 46 (Fig. 4*B*). In addition, neurons throughout most of penetration 4 failed to respond, except at a depth of 650 μm, at which a weak response could be obtained to moving stimuli in the visual field, but no receptive field could be determined. Likewise, a weak response to visual stimuli was detected for neurons in penetration 45, but no receptive field could be accurately determined. Together, the unresponsive and nearly unresponsive recording sites defined a region of MT that approximately matched the zone expected to be deprived of V1 activation. At two other locations (penetrations 13 and 44) along the edge of this unresponsive zone, neurons responded to visual stimuli

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in an inconsistent manner. Typically, the first presentation of a moving stimulus would produce a response, whereas subsequent stimuli were ineffective. After a few minutes of rest, a clear response could be evoked again. The estimated receptive field locations for neurons in these penetrations (Fig. 4*D*) were either approximately normal (13) or extended abnormally into the upper visual quadrant (44). In other parts of MT, normal, robust responses to visual stimuli were obtained at ~150 recording sites in 34 electrode penetrations. Receptive field sizes and locations were in the normal range. No neurons had receptive fields that included the 8–10° of central vision missing from V1.

In summary, a larger lesion resulted in part of MT being unresponsive to visual stimuli, even after 77 d of recovery. Some deprived or partially deprived neurons may have acquired new receptive fields, but this is uncertain. No neurons had receptive fields in the portion of the visual hemifield missing from V1.

Similar results were obtained from an owl monkey with an even larger lesion of V1 (owl monkey 99-9) (Fig. 5). The portion of the lesion in dorsolateral cortex along the V1–V2 border is shown in Figure 5*A*. The lesion approached but spared V2. Other parts of the lesion were apparent in sections from separate regions of V1 that were on the medial wall and in the calcarine fissure. Cortex representing central and paracentral vision of the upper visual quadrant was spared, as was a portion of central vision of the lower quadrant, but a large extent of nearly 30° of the representation of paracentral vision of the lower quadrant was missing. After 69 d of recovery, neurons at 13 electrode penetration sites were completely unresponsive to visual stimuli through the depth of cortex (sites 1, 2, 15, 23–25, 36–38, 43, 45, 46, and 49). Neurons at penetration 22, at a depth of 500 μm, displayed a very weak response, but a receptive field could not be located. Together, electrode penetrations were grouped to define a large segment of MT where neurons would be expected by location to be activated by missing portions of V1, and no other source of visual activation was evident. Neurons at six other penetration sites (7, 11, 16, 21, 30, and 32) responded robustly to an initial visual stimulus but were fatigued and often failed to respond to rapidly repeated stimuli. Receptive fields for these neurons were in locations corresponding to intact portions of V1 (Fig. 5*C*), so the transient responsiveness of these neurons may not have been related to the V1 ablation but possibly to other factors such as the depth of anesthesia. Neurons at other sites responded vigorously to visual stimuli.

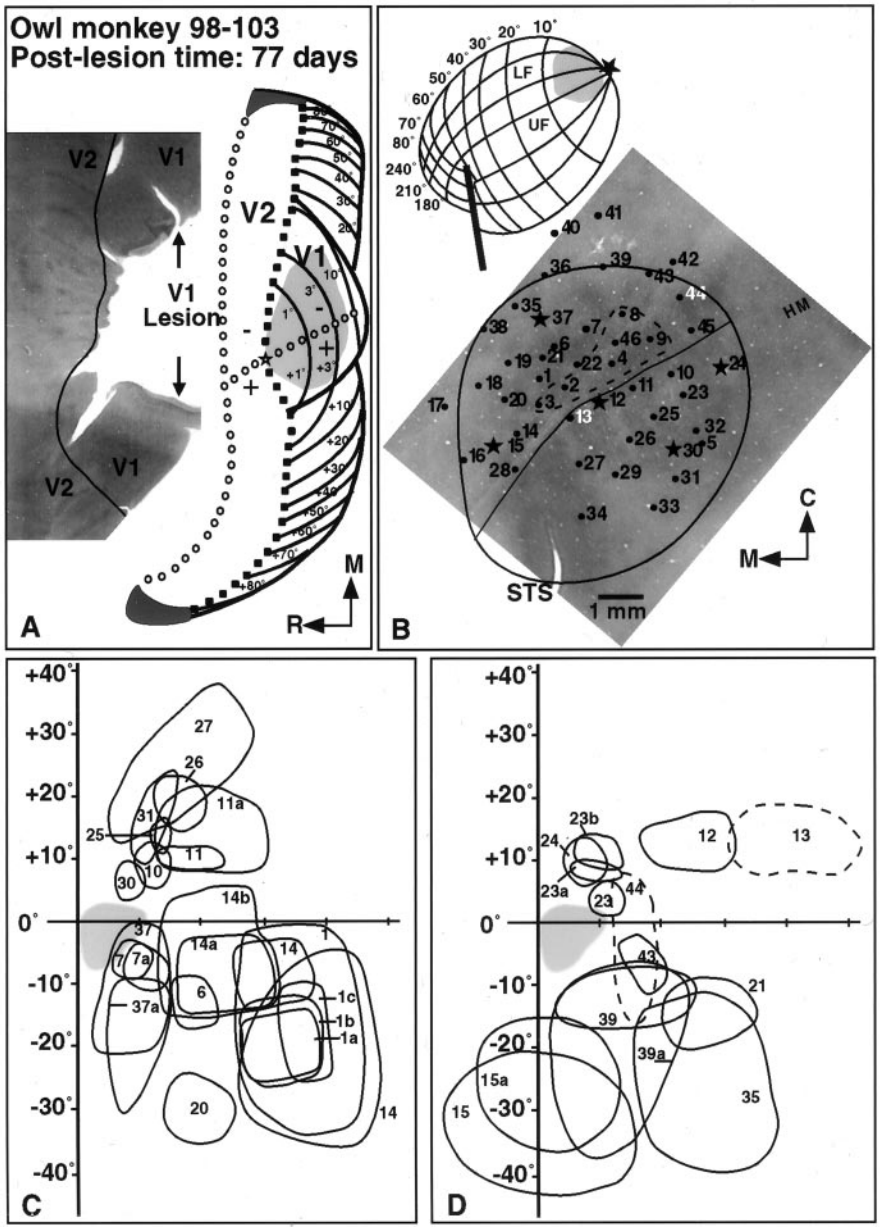

Figure 4. V1 lesion and physiological mapping data from owl monkey 98-103. *A*, Section of flattened cortex stained for CO shows the location of the V1 lesion. CO-dense *stripes* indicate an intact V2. The schematic of unfolded V1 and V2 illustrates the approximate visuotopic position of the lesion with *gray shading*. *B*, *Gray shading* on the schematic of the visuotopic map of MT illustrates the part of MT affected by the V1 ablation. An enlarged CO-stained section of MT shows electrode penetration sites with *numbered black dots* and recording sites with electrolytic lesions marked with *stars*. Six penetration sites where there were no responses to visual stimuli are encircled by a *dashed line*. *White numbers* indicate penetrations where neurons were only transiently responsive to visual stimuli. *C, D*, Receptive fields (RFs) corresponding to penetration sites in *B* illustrated on two diagrams for clarity. RF *numbers* correspond to penetration numbers in *B*. RFs drawn with *dotted lines* were only transiently responsive to visual stimuli. Borders of these RFs are less precise. Conventions are as in previous figures.

Comparable recordings were obtained from a third owl monkey with a large lesion of V1. In owl monkey 99-10 (Fig. 6), most of dorsolateral V1 and adjoining cortex of the medial wall and upper bank of the calcarine fissure was removed. The lesion extended to the border of V2, along the representation of the paracentral lower visual quadrant, but it did not include more than the margin of V2 (Fig. 6*B*). Although as much as 50° of the representation of the lower visual quadrant was missing, most of the cortex devoted to the upper visual quadrant was intact. After 77 d of recovery, neurons in 21 electrode penetrations within MT were totally unresponsive to visual stimuli. These electrode penetrations were within a single zone in MT (outlined in Fig. 6*C*)

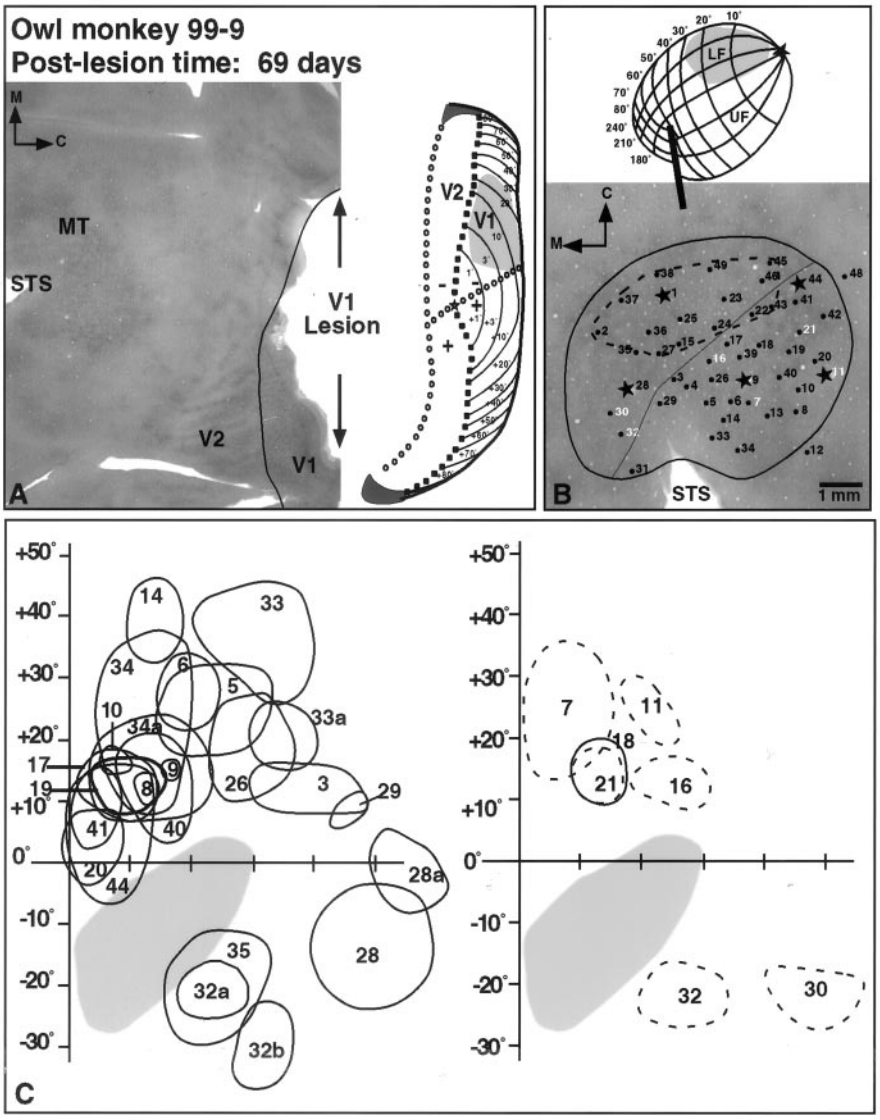

Figure 5. V1 lesion drawing and physiological mapping data from owl monkey 99-9. *A*, Section of flattened cortex stained for CO showing the location of the V1 lesion and spared portions of the upper field representation of V1. Additional spared V1 medial to the lesion is not shown. A schematic of unfolded V1 and V2 illustrates the approximate visuotopic position of the lesion with *gray shading*. *B*, The *gray* zone on the schematic of the visuotopic map of MT illustrates the part of MT affected by the V1 ablation. An enlarged CO-stained section of MT shows electrode penetration sites with *numbered black dots* and recording sites with electrolytic lesions marked with *stars*. Fifteen penetration sites where there were no responses to visual stimuli are encircled by a *dashed line*. *White numbers* indicate penetrations where neurons were only transiently responsive to visual stimuli. *C*, Receptive fields corresponding to penetration sites in *B*. Conventions are as in previous figures.

that corresponded to cortex that would be deprived of V1 input by the lesion. Neurons at another site (9) were weakly and inconsistently activated by visual stimuli, and a receptive field could not be localized. This site likely was at least partially deprived by the lesion. Neurons at another site (28) were robustly but transiently responsive to visual stimuli, but neurons at this site were in the normally innervated portion of MT, and the receptive field was normally located. Neurons in sites 4 and 5 had unusually small receptive fields that may have been reduced by a partial loss of V1 inputs. Neurons at other sites scattered across lateral MT were strongly activated by visual stimuli, were sensitive to the direction of moving stimuli, and had receptive fields of normal sizes in portions of the upper visual quadrant that were represented in intact portions of V1.

The results from these three monkeys, with moderate to moderately large lesions of V1 and recovery periods of just >2 months, clearly indicate that the neurons in MT that have been totally deprived of V1 inputs do not recover responsiveness to other inputs. Some partially deprived neurons may have unusual response characteristics, such as failing to respond to repeated stimuli, but neurons at sites in innervated portions of MT also expressed some of these properties. Neurons along the edges of deprived zones of MT may have had smaller-than-normal receptive fields because of a partial loss of V1 inputs, or acquired new, displaced receptive fields because of the potentiation of formerly subthreshold inputs, but this was far from evident. There was no clear evidence that any neurons could be activated by stimuli in parts of the visual field that were no longer represented in V1.

Recordings after a massive lesion of V1

In one additional owl monkey (99-82), a large lesion included dorsolateral, ventral, and most of calcarine V1 (data not shown). After a recovery of 78 d, recordings were attempted at 34 electrode penetration sites in MT. Only two sites had neurons that responded at all to visual stimuli, and those responses were weak to large moving stimuli. We were not able to precisely locate any receptive fields, and the responses could have related to the intact parts of V1, which represented peripheral vision. Thus, after a nearly complete lesion of V1 and a long recovery period, neurons in MT did not respond to some other source of visual activation.

Recordings after 10 months of recovery

To evaluate the possibility that deprived neurons would recover responsiveness to visual stimuli with ever longer recovery periods, recordings were obtained from one owl monkey 10 months after a large lesion of much of dorsomedial and upper calcarine V1, devoted to the central 30–40° of the lower visual quadrant. The brain was cut in a coronal plane so that the precise alignment of the lesion with the V2 border was less obvious than in other cases, but much of V2 appeared undamaged, and most of the ventral cortex, representing the upper visual quadrant in V1, was intact. Again, recordings revealed a large zone of MT where neurons were unresponsive to visual stimuli. Neurons at 15 penetration sites (Fig. 7*A*, *white dots*) were totally unresponsive to visual stimuli. Neurons at 13 of these sites collectively formed a continuous zone in portions of MT that would have been deprived of input from V1 by the lesion. Weak and transient responses were obtained from neurons at eight other sites (Fig. 7*A*, *white numbers 2, 5, 14, 16, 19, 20, 21, 24*) adjoining the unresponsive zone, whereas 3 additional sites (7, 22, and 28) were weakly and transiently activated by stimuli in the lower visual quadrant, but receptive field locations

could not be determined. Possibly these neurons were partially or mostly deprived of V1 inputs. Neurons at other sites were strongly responsive to visual stimuli and had receptive fields corresponding to intact portions of V1. However, the receptive fields for neurons at sites 23 and 8 were unusually large. In conclusion, there was no evidence that neurons totally deprived of V1 inputs recovered and responded to an alternative source of visual activation with recovery times as long as 10 months.

Effects of V1 lesion on MT neurons in a marmoset

To address the possibility that V1 lesions affect MT neurons differently in marmosets than in owl monkeys, a large lesion of V1 was placed in one marmoset. The lesion included dorsolateral V1 and V1 of the upper medial wall, portions related to central and paracentral vision of the lower visual quadrant (Fritsches and Rosa, 1996). Preserved portions of V1 related to peripheral vision of both the upper and lower visual quadrants and paracentral vision of the upper visual quadrant. In this case, the preserved portions of V1 were estimated directly from coronal brain sections through cortex and from retrograde degeneration in coronal sections through the LGN. The extensive zone of degeneration through the midportion of the LGN (Fig. 8*C*) corresponds to approximately the central 30° of vision (Kaas et al., 1972). The preserved medial portion of the LGN represents more peripheral vision of the lower visual quadrant, whereas the preserved lateral portion represents paracentral and peripheral vision of the upper quadrant.

Recordings from MT of the marmoset 96 d after the large V1 lesion revealed a large region of unresponsive cortex. Almost all of the 67 electrode penetrations over MT (Fig. 8*A*, *white dots*) failed to encounter neurons responsive to visual stimuli. However, neurons at four sites (32, 34, 44, and 45) were weakly and transiently activated, and receptive field locations could be estimated (Fig. 8*B*). More consistent but weak responses were obtained for neurons in penetrations 30, 33, and 35. All activated neurons at these sites had receptive fields in paracentral portions of the upper visual quadrant, corresponding to a portion of V1 that was mostly preserved. Neurons were strongly responsive at one site (61) in rostromedial MT, and the normal receptive field could be localized to the peripheral lower visual quadrant (Fig. 8*B*).

In summary, the recordings in MT after a V1 lesion in a marmoset were similar to those in owl monkeys. The results provide no evidence for a source of above-threshold activation of neurons in MT in addition to those from V1. Deprived neurons either failed to respond to visual stimuli or responded to locations represented in intact portions of V1.

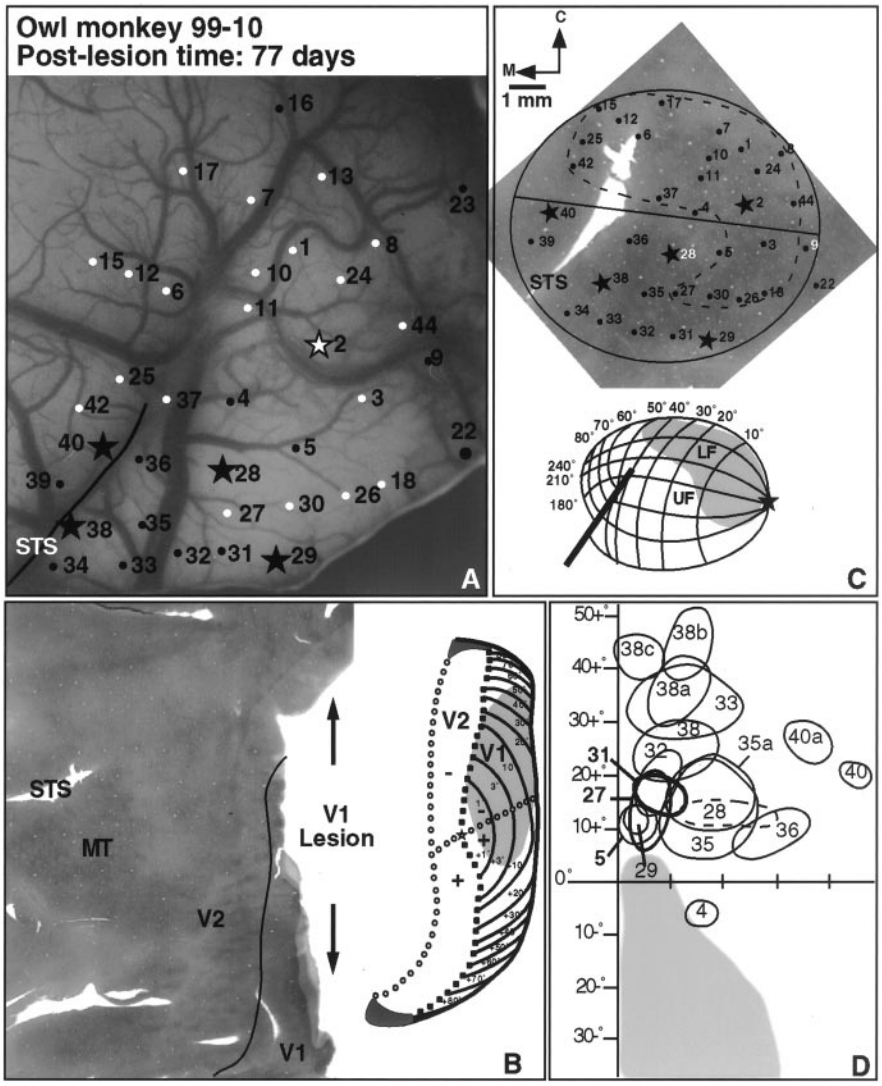

Figure 6. V1 lesion and physiological mapping data from owl monkey 99-10. *A*, Photograph of the cortical surface used for recording electrode penetration sites during mapping. Penetration sites are marked with *numbered dots*, and locations of lesions made at five recording sites are marked with *stars*. *White dots* and *stars* indicate sites where neurons were unresponsive to visual stimuli. *Black dots* and *stars* mark sites where neurons were responsive. *B*, Section of flattened cortex stained for CO illustrating the location of the V1 lesion. Spared parts of the upper field representation in V1 are evident. Additional spared V1 located medially is not pictured. The visuotopic location of the lesion is represented by *gray shading* on the schematic of unfolded V1 and V2. V2 is not damaged. *C*, The *gray* zone on the schematic of the visuotopic map of MT illustrates the part of MT affected by the V1 ablation. An enlarged CO-stained section of MT shows electrode penetration sites with *numbered black dots* and recording sites with electrolytic lesions marked with *stars*. Twenty electrode penetration sites where there were no responses to visual stimuli are encircled by a *dashed line*. *White numbers* indicate penetrations where neurons were only transiently responsive to visual stimuli. *D*, Receptive field locations corresponding to recording sites in MT. RF *28* is outlined with a *dashed line* to indicate that neurons at that location were only transiently responsive and the RF has less precise borders. Abbreviations are as in previous figures.

Architecture of MT after long-standing V1 lesions

The physiological results indicate that V1 provides the dominant, activating input to MT and that, even after long periods of recovery, MT neurons are not activated by other sources of input. This major loss of activating input, in addition to direct damage to the axons of feedback neurons in layer 6 of MT that project to V1 (Tigges et al., 1981), might alter the histological appearance of MT. However, no major changes at the histological level were noted. More specifically, MT remained CO-dense and primarily myelin-dense throughout. The CO-dense region that identifies

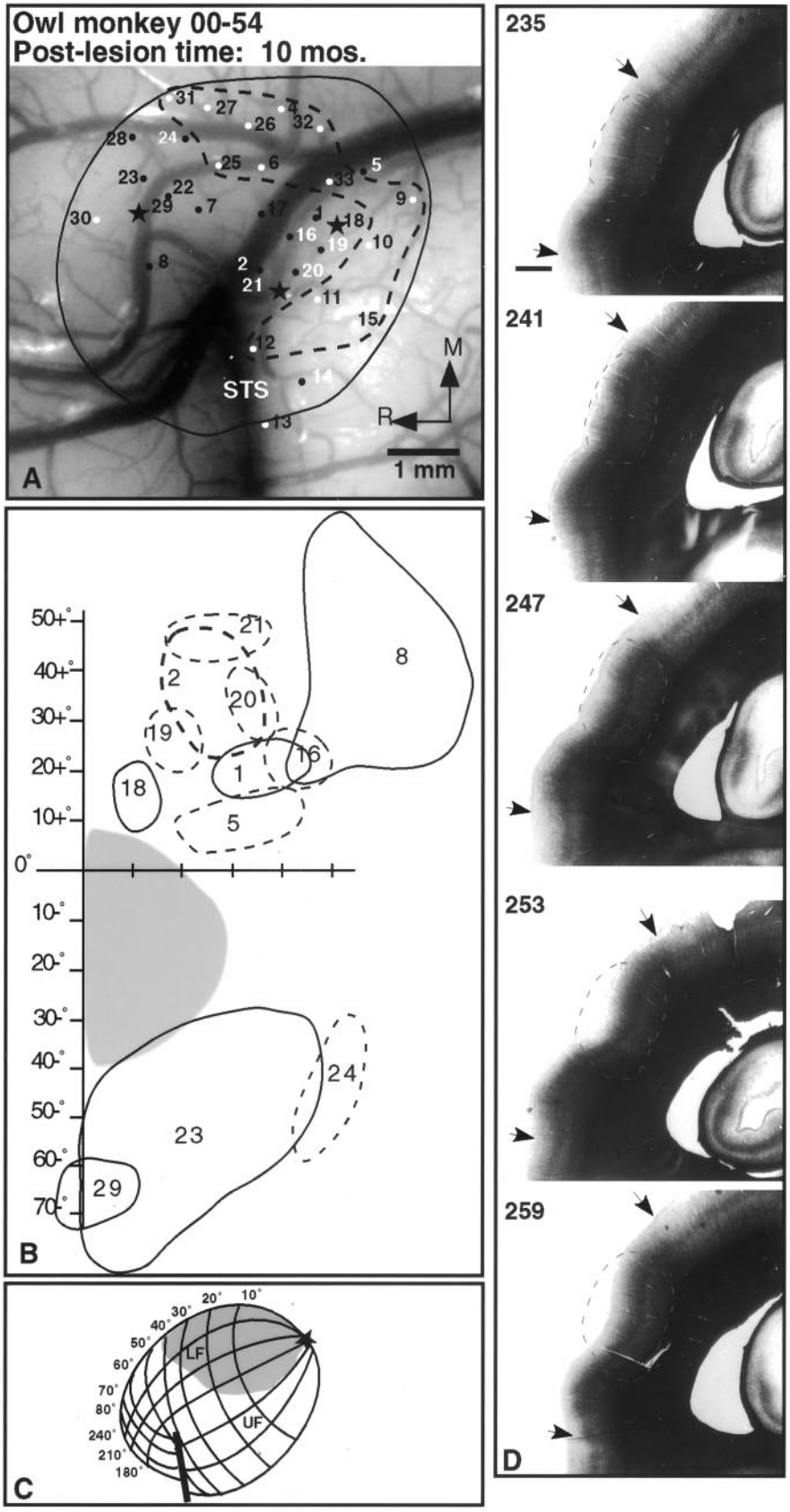

Figure 7. *A*, Photograph of the surface of cortex from owl monkey 00-54 for recording electrode penetration sites during mapping. Electrode penetration sites are shown as *dots*. *B*, *Numbered* receptive field locations correspond to penetration sites in MT shown in *A*. Precise RF locations were impossible to determine for penetrations 7, 17, 22, and 28 because of the transient nature of the responses. *C*, Series of coronal sections stained for myelinated fibers from owl monkey 00-54. *Black arrows* mark the approximate borders of MT, and *dashed lines* outline a myelin-light zone in MT. Conventions are as in previous figures.

MT is normally somewhat patchy as a result of a modular substructure (Tootell et al., 1985; Krubitzer and Kaas, 1990) (Fig. 2*A*), and the patchy, CO-dense look of MT appeared to be unaffected by V1 lesions. The myelination of MT also appeared to be mostly unaffected by V1 lesions, although some reduction in the density of the myelination was suggested. In a series of sections of flattened cortex stained for myelin in owl monkey 99-9 with a long-standing V1 lesion, MT was myelin-dense throughout, except for a small oval of MT, corresponding to the region deprived of V1 input, which appeared to be somewhat reduced in myelin density (Fig. 9*A*). Likewise, in owl monkey 00-54 in which cortex was cut coronally, the deprived portion of MT appeared to be slightly less myelinated than the rest of MT (Figs. 7*D*, 9*B*, section *253*). Because the myelination pattern of cortical areas is thought to be primarily attributable to the myelination of intrinsic connections (Hellwig, 2002), the present evidence of a slight reduction in myelination as a result of V1 lesions should be treated with caution. Nevertheless, adjacent sections in case 00-54, stained for WFA or Nissl substance (Fig. 9*B*), also show slight differences in the deprived zone of cortex, with a slight reduction in WFA reactivity and more dense thionin staining.

Discussion

The major result of the present study was that, even after 2.5–10 months of recovery, neurons were unresponsive to visual stimuli over most of the portion of MT that was deprived of direct V1 inputs. Although neurons along the margin of the deprived region in MT may have recovered some responsiveness to intact V1 inputs, no massive reactivation of deprived MT by remaining V1 inputs occurred. Likewise, there was no obvious activation of deprived MT by other potential sources, such as from the superior colliculus via the pulvinar. The V1 lesions did not result in marked histological alterations in MT, although some change was suggested.

V1 appears to be the dominant source of activation of MT neurons in at least New World owl monkeys and marmosets

Overall, the results obtained after V1 lesions in New World owl monkeys are highly consistent. Kaas and Krubitzer (1992) found that partial lesions of V1 in owl monkeys immediately deactivated retinotopically matched portions of MT. In the present experiments, recordings

from neurons at a substantial number of electrode penetrations clearly within MT failed to encounter visually responsive neurons even after recoveries of as long as 10 months. Similar results were obtained in one marmoset. All unresponsive zones in MT appeared to closely correspond to regions deprived of direct V1 inputs.

In contrast, Rosa et al. (2000) concluded from recordings weeks after V1 lesions in marmosets that many neurons (at least 31%) in the deprived portions of MT had receptive fields displaced from predicted locations so that they responded to stimuli activating intact portions of V1. Thus, there was evidence for considerable retinotopic reorganization of MT. In addition, 20% of the recording sites within the deprived MT responded to visual stimuli with receptive fields centered within the scotoma produced by the V1 lesion, providing evidence for a source of visually driven activation that is independent of V1. However, the remaining 18% of the sites within deprived MT encountered neurons that were spontaneously active but failed to respond to visual stimuli. In addition, given uncertainties about the sizes of the scotomas, Rosa et al. (2000) stated that only 13 neurons "unequivocally responded to stimulation restricted to the scotoma." As in the experiments on owl monkeys, many neurons in deprived MT of marmosets were rendered unresponsive to visual stimuli, and others responded to stimuli activating intact portions of V1, yet 13 neurons appeared to be activated by a source of input that was independent of V1.

The reasons for differences in results are not clear. Because Rosa et al. (2000) studied MT neurons weeks after V1 lesions, they suggested that total deactivation obtained immediately after the lesions by Kaas and Krubitzer (1992) could be attributable to a widespread depression that might follow a lesion (Seitz et al., 1999). This explanation seems unlikely, given that there were normal responses in portions of MT outside the deprived zone in the study of Kaas and Krubitzer (1992). Another possibility is that anesthetics depressed weak responses in deprived neurons more strongly in our experiments than those of Rosa et al. (2000). As in all the studies with V1 lesions or deactivations, our monkeys were anesthetized, but types of anesthetics differ in effects and depths of anesthesia may differ. We addressed this issue by using three different anesthetics in different experiments, and our results were the same across anesthetics. No neurons clearly demonstrated a nonstriate source of effective input. Another difference is that Rosa et al. (2000) studied marmosets, whereas the present results and those of Kaas and Krubitzer (1992) were from owl monkeys, yet we included one marmoset in the present study and got results comparable with those in owl monkeys.

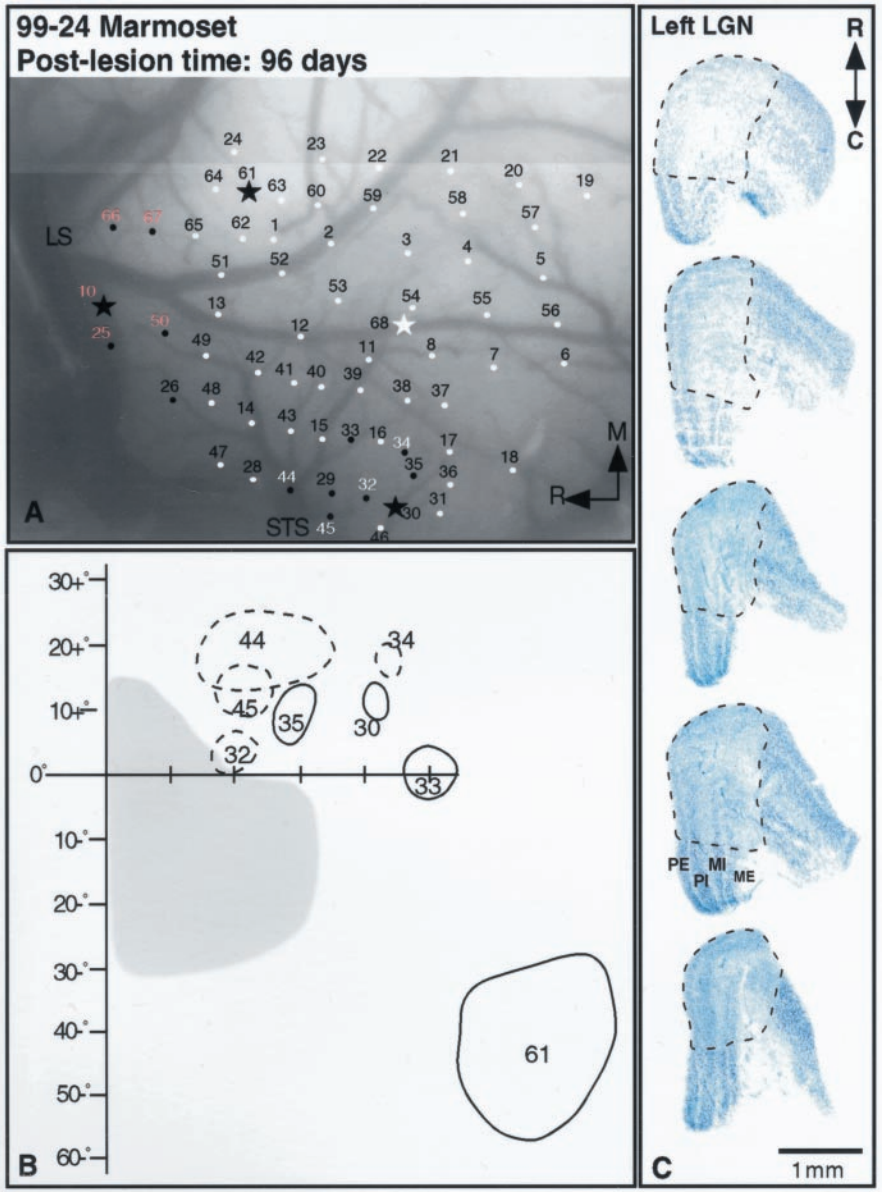

Figure 8. *A*, Photograph of the cortical surface over area MT in a marmoset monkey (case 99-24). Electrode penetration sites are marked with *dots*. Penetrations marked with *red numbers* near the lateral sulcus indicate sites responsive to auditory stimuli. *B*, Receptive field locations for eight recording sites where responses were obtained. Four RFs drawn with *dashed lines* indicate that neurons were only transiently responsive at those locations. *C*, Series of coronal sections from the LGN ipsilateral to the V1 lesion. *Dashed lines* outline the borders of the retrograde degeneration resulting from the large V1 ablation. Conventions are as in previous figures.

Does MT retinotopically reorganize to represent more of the preserved inputs from V1?

Neurons in primary visual cortex acquire new receptive fields after being deprived by retinal lesions (Kaas et al., 1990; Heinen and Skavenski, 1991; Gilbert and Wiesel, 1992) (for review, see Kaas et al., 2001). Similar reorganizations might be expected in MT, because individual axons of neurons in V1 branch and form multiple terminal arbors over considerable distances in MT (Rockland, 1989). In addition, the intrinsic (Weller et al., 1984; Krubitzer and Kaas, 1990) and callosal (Cusick et al., 1984; Maunsell and Van Essen, 1987; Krubitzer and Kaas, 1990) connections of MT are widespread. Thus, one might expect that when as much as half or more of V1 remains, neurons over most or all of MT would either remain somewhat responsive or regain

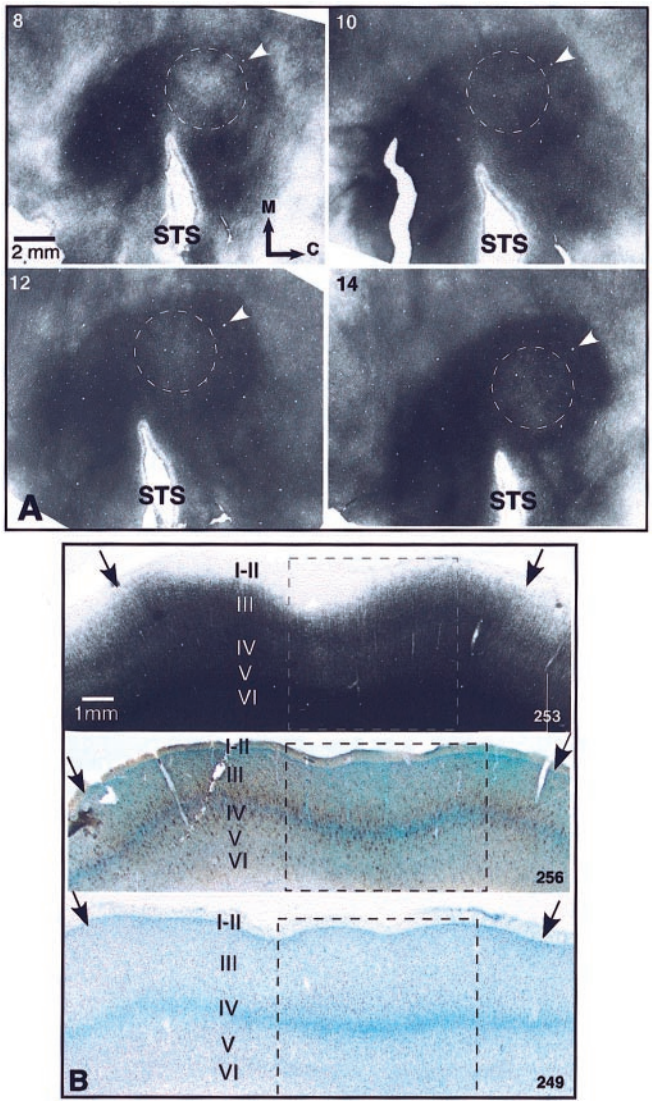

Figure 9. *A*, Serial sections of owl monkey flattened cortex illustrating the patchy myelin-staining pattern in MT ipsilateral to a V1 ablation. Myelin-light *ovals* corresponding to columns of neurons are visible in some sections. A myelin-light *patch* medial and caudal to the STS (inside *white dashed circle*) may be a result of V1 ablation. *White arrowheads* mark a common blood vessel. *B*, Three coronal sections from owl monkey 00-54. *a*, A myelin-light zone is shown in the *box* in section *253*, which may be related to the V1 ablation. *b*, Section *256* stained for WFA (*brown*) and counterstained with thionin (*purple*). The WFA staining is reduced in the same part of the section that is myelin-light in *a*. *c*, Section *249* stained for Nissl substance, which appears to have a higher cellular density in layers IV–VI in the V1-deprived part of MT that is myelin-light in *a*. Conventions are as in previous figures.

responsiveness with significant recovery times. Instead, the functional reorganization of MT after V1 lesions appears to be limited. Immediately after V1 lesions, neurons near the margins of the deprived zone may have acquired new receptive fields (Kaas and Krubitzer, 1992), but neurons in the core of the deprived zone remained unresponsive. Approximately similar results were obtained in the present experiments after months of recovery, although one monkey with a small lesion demonstrated no unresponsive zone (Fig. 10). Rosa et al. (2000) reported more extensive retinotopic reorganization of MT after weeks of recovery from V1 lesions. However, the evidence for reorganization depends on demonstrating that neurons have receptive fields in abnormal locations. This can be difficult to demonstrate when retinotopy varies across individuals. Van Essen et al. (1981) commented on how irregular and variable the retinotopy of MT is in macaque monkeys, and the modular organization of MT in owl monkeys and other primates (Born and Tootell, 1992) suggests that separate neurons in the two types of modules can have similar receptive fields. Thus, a completely smooth and predictable retinotopy is not expected. Another issue is the source of activation from outside of the scotoma. Neurons in MT could be excited by direct contacts with formerly subthreshold V1 inputs that remain or indirectly by V1 activation of intrinsic or callosal connections. The weak and strongly habituating responses could reflect excitatory inputs from such widespread connections or even the release of lateral inhibition on spontaneously active neurons after stimulation, as has been reported for neurons in deprived somatosensory cortex (Rasmusson and Turnbull, 1983). The evidence for some retinotopic reorganization of MT indicates the importance of being certain that receptive fields and visual stimuli are fully within the scotoma before visual responsiveness can be taken as evidence for a source of activation independent of V1.

Are New and Old World monkeys similar?

As New and Old World monkeys diverged into separate lines of evolution some 40 million years ago, the effects of V1 lesions on MT in New and Old World monkeys do not necessarily need to be the same. Nevertheless, the impact of V1 lesions on MT neurons appears to be similar in New and Old World monkeys.

The original observations in macaques on MT after V1 lesions were obtained from three macaque monkeys 5–6 weeks after unilateral or bilateral lesions of part or most of V1 (Rodman et al., 1989). In deprived parts of MT, neurons often responded to visual stimuli, but responses were weak, and receptive fields were difficult to localize precisely. Few neurons (5%) responded strongly to visual stimuli. There was no evidence for retinotopic reorganization in MT. In the two monkeys with bilateral V1 lesions, a subsequent lesion of the superior colliculus on the side of recording in MT abolished all remaining responsiveness of MT neurons to visual stimuli, providing evidence that any remaining responsiveness depended on the superior colliculus and not intact parts of V1 (Rodman et al., 1990).

In a related study in macaques, Maunsell et al. (1990) selectively blocked activity in part of the lateral geniculate nucleus with lidocaine and found that a block of both magnocellular and parvocellular layers completely abolished the responses of neurons in MT. A block of the magnocellular layers alone was highly effective in reducing the responsiveness of neurons in MT to an average of ~10% of the original response. Because the effects of these blocks are thought to be mediated by LGN projections to

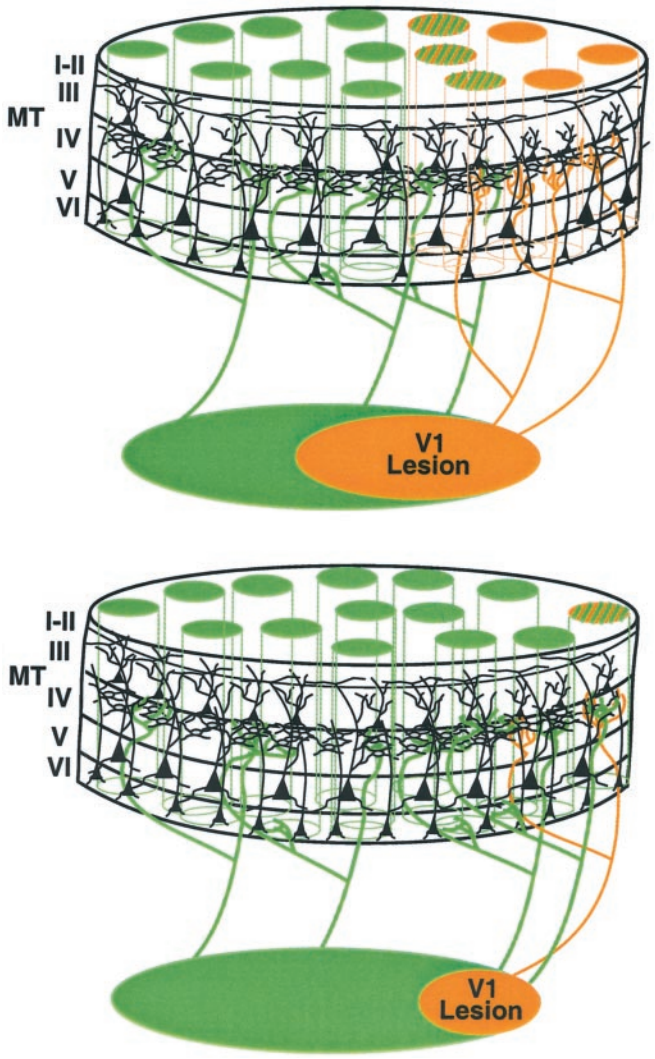

Figure 10. Schematic representations of the reorganization of MT after removal of a portion of V1. *A*, Removal of a large portion of V1 (*orange*) results in a part of MT that is completely deprived of V1 inputs and no longer responsive to visual stimuli (*solid orange columns*) and a part of MT bordering this deprived zone that still has limited inputs from V1 and transient or weak responsiveness to visual stimuli (*green* and *orange striped columns*). *B*, With a much smaller V1 ablation, there would be no area of MT completely deprived of V1 inputs, but some areas may be partially deprived and may respond less robustly to a visual stimulus.

V1, these results demonstrate at least a strong dependence of MT neurons on V1.

Using another approach, Girard et al. (1992) concluded that 80% of sites in MT responded to visual stimuli after a corresponding part of V1 was deactivated with a cooling plate. Because a cooling plate on the dorsolateral surface of striate cortex would relate to only the central 4° or so of lower quadrant vision (Adams and Horton, 2001) few neurons would be completely deprived of V1 activation, even if the cooling were fully effective. Of nine recording sites in MT where neurons were judged to have receptive fields completely within the estimated scotoma from cooling V1, visual responses were completely blocked only at one. In one monkey in which an additional anesthetic (halothane) was added to the ventilation mixture of nitrous oxide and oxygen, neurons at 19 of 22 sites in MT were inactivated. Although the authors attributed the unresponsiveness of MT neurons to the combination of the extra anesthesia and V1 cooling, the results could reflect more effective cooling alone. If the halothane did have an added effect, this suggests that the responses of neurons totally or partly deprived of V1 inputs are highly fragile and easily disruptable.

Together, the results from macaque monkeys are limited, open to interpretation, and somewhat contradictory. All studies reveal a considerable reduction in the responsiveness of MT neurons after a partial to complete loss of V1 inputs. Results in two of the studies suggest that at least some of the remaining responsiveness could come from the contralateral V1 via the corpus callosum. Other responses could involve expanded effectiveness of intact inputs from V1, especially over longer recovery times. Whether a cooling probe on dorsolateral V1 fully or consistently deprives MT neurons of V1 activation is questionable, yet Rodman et al. (1989) reported that many MT neurons in the scotoma remained at least weakly responsive to visual stimuli in two monkeys with long-standing, bilateral, extensive lesions of V1.

If the results from both New and Old World monkeys are considered together, it seems obvious that the major driving input to MT, directly and indirectly, is from V1. Another much less effective input that is not dependent on V1 appears likely, especially for macaques, but additional experiments on awake monkeys would be useful. A functional reorganization of MT after V1 lesions has been suggested, but the evidence for this is limited and even questionable.

The role of MT in blindsight

The present results relate to the issue of whether MT activation is important in blindsight in humans. According to some reports, detection of visual motion (Barbur et al., 1993), wavelength (Stoerig and Cowey, 1989), emotion (de Gelder et al., 1999), and other visual stimuli is possible in parts of scotomas related to V1 lesions in humans. Evidence for blindsight has also been reported after V1 lesions in macaque monkeys (Moore et al., 1995; Cowey and Stoerig, 1999). There are positron emission tomographic, electroencephalographic, and magnetoencephalography data that suggest that the MT+/V5 region of visual cortex can be activated by moving stimuli in a well studied patient (G.Y.) with a loss of much of V1 (Barbur et al., 1993; ffytche et al., 1996), yet a more recent functional magnetic resonance imaging (fMRI) study in the same patient (Baseler et al., 1999) showed no activation in MT+/V5, and the authors suggested that the visual behavior in blindsight may depend on callosal inputs from the intact hemisphere. In addition, there is evidence from other patients with V1 lesions that small islands of V1 are often preserved, and that the effectiveness of visual detection depends on these islands of V1 and possibly on their projections to MT (Fendrich et al., 2001). Evidence from fMRI indicates that small, preserved portions of V1 in humans are capable of activating larger-than-normal portions of extrastriate cortex, and that callosal inputs also expand their territories of activation (Baseler et al., 1999).

Conclusions

Overall, the evidence indicates that MT is highly dependent on V1 for visual activation in primates. Although the emphasis in some reports has been on the preserved responsiveness of MT neurons after V1 deactivation, this emphasis seems misplaced. In New World monkeys, totally deactivated zones in MT have been reported immediately after (Kaas and Krubitzer, 1992) and months after (this report) V1 lesions, whereas the evidence for any preserved responsiveness that is independent of V1 is limited to the primarily impaired responses of a few neurons (Rosa et al., 2000). In macaque monkeys, the overwhelming effect is a total

lack of responsiveness or greatly impaired responsiveness, with evidence for preserved responses primarily coming from recordings from two monkeys weeks after V1 lesions (Rodman et al., 1989). The effects of cooling parafoveal V1 appear to be variable (Rodman et al., 1989; Girard et al., 1992), whereas limited recordings from MT after a lidocaine block of the LGN suggest that MT neurons are completely dependent on an LGN-to-V1 relay (Maunsell et al., 1990). In one human (G.Y.) with an extensive V1 lesion, activity in the MT region was reported (Barbur et al., 1993; ffytche et al., 1996; Baseler et al., 1999), but the independence of blindsight from V1 function has been questioned (Schärli et al., 1999a,b; Fendrich et al., 2001).

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