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Area 17 lesions deactivate area MT in owl monkeys

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Abstract

The middle temporal visual area, MT, is one of three major targets of the primary visual cortex, area 17, in primates. We assessed the contribution of area 17 connections to the responsiveness of area MT neurons to visual stimuli by first mapping the representation of the visual hemifield in MT of anesthetized owl monkeys with microelectrodes, ablating an electrophysiologically mapped part of area 17, and then immediately remapping MT. Before the lesions, neurons at recording sites throughout MT responded vigorously to moving slits of light and other visual stimuli. In addition, the relationship of receptive fields to recording sites revealed a systematic representation of the contralateral visual hemifield in MT, as reported previously for owl monkeys and other primates. The immediate effect of removing part of the retinotopic map in area 17 by gentle aspiration was to selectively deactivate the corresponding part of the visuotopic map in MT. Lesions of dorsomedial area 17 representing central and paracentral vision of the lower visual quadrant deactivated neurons in caudomedial MT formerly having receptive fields in the central and paracentral lower visual quadrant. Most neurons at recording sites throughout other parts of MT had normal levels of responsiveness to visual stimuli, and receptive-field locations that closely matched those before the lesion. However, neurons at a few sites along the margin of the deactivated zone of cortex had receptive fields that were slightly displaced from the region of vision affected by the lesion into other parts of the visual field, suggesting some degree of plasticity in the visual hemifield representation in MT. Subsequent histological examination of cortex confirmed that the lesions were confined to area 17 and the recordings were in MT. The results indicate that the visually evoked activity of neurons in MT of owl monkeys is highly dependent on inputs relayed directly or indirectly from area 17.

Keywords: Visual cortex, Pulvinar, Area 18, Superior colliculus

Introduction

The middle temporal visual area, MT, in the upper temporal lobe of primates, is a major target of direct projections from primary visual cortex, VI or area 17 (e.g. Ungerleider & Mishkin, 1979; Montero, 1980; Weller & Kaas, 1983; see Krubitzer & Kaas, 1990*a*, for review). Additional inputs to MT are from the other two principal targets of area 17, the second visual area, VII (e.g. DeYoe & Van Essen, 1985; Shipp & Zeki, 1989; Krubitzer & Kaas, 1989*a*, 1990*a*) and the dorsomedial visual area, DM (Lin et al., 1982; Krubitzer & Kaas, 1990*b*). Because of these direct and indirect cortical inputs and evidence from earlier studies that lesions of area 17 deactivate large regions of visual cortex (Rocha-Miranda et al., 1975; also see Girard et al., 1991), it seemed reasonable to assume that the visually evoked activity in MT largely or completely depends on area 17, as does the visually evoked activity in the other major target of area 17, area 18 (Schiller & Malpeli, 1977; Bullier & Girard, 1988). However, the results of recent experiments suggest that considerable responsiveness to visual stimuli persists in MT after deactivations of area 17, at least in macaque monkeys. Rodman et al. (1989) recorded from neurons in MT after partial or total lesions or cooling of area 17, and found the majority of cells remained responsive to visual stimuli, although responses were typically weaker. In addition, normal response properties of direction selectivity, binocularity, and receptive-field size, as well as a systematic representation of the contralateral visual hemifield, were preserved. Similarly, Bullier and Girard (1988) reported that many neurons in MT remained responsive to visual stimuli after activity in area 17 was blocked by cooling. In contrast, Maunsell et al. (1990) found neurons in MT of macaque monkeys to be profoundly affected by blocking the relay of information from the magnocellular layers of the lateral geniculate nucleus (LGN) to area 17, slightly affected by blocking the parvocellular layers, and, in a small sample of cells, completely unresponsive after a complete block of LGN transmission. Thus, deactivating neurons projecting to and activating area 17 also deactivated neurons in MT.

Any responsiveness that remains in MT after area 17 deactivation would presumably depend on rather indirect relays of visual inputs from the superior colliculus to parts of the pulvinar complex, and then to visual cortex (see Lin & Kaas, 1980), since only a few neurons in the lateral geniculate nucleus project

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outside of area 17 (Benevento & Yoshida, 1981; Yukie & Iwai, 1981; Lysakowski et al., 1988; Cowey & Stoerig, 1989), and none project to MT (Benevento & Standage, 1982). Although major activation of MT *via* a relay from the superior colliculus seems improbable in view of the indirectness of the pathway, and the evidence that area 17 lesions severely depress visual responsiveness of pulvinar neurons (Burman et al., 1982; Bender, 1983), it appears that lesions of the superior colliculus, combined with removal of area 17, totally eliminate the responsiveness of MT neurons to visual stimuli (Rodman et al., 1990).

For the present study, we further examined the dependence of visual activity in MT on area 17, but considered another primate, the New World owl monkey, *Aotus trivirgatus*. One reason for studying owl monkeys is that primate species may differ in the degree of dependence of MT on inputs from area 17. In addition, it seemed useful to examine the impact of area 17 lesions on MT neurons in view of the more recent evidence of Maunsell et al. (1990) that the deactivation of area 17 may have a more profound effect than suggested by the experiments of Rodman et al. (1989) and Bullier and Girard (1988). Owl monkeys offer the technical advantage of having most of MT exposed on the dorsolateral surface of the brain. Thus, it was relatively straightforward to map MT both before and after area 17 lesions, and note changes in responsiveness. Furthermore, cortex in the region of MT can be easily separated from the rest of the brain, flattened, and cut parallel to the surface so that myelin stains can reveal the complete outline of MT (see Krubitzer & Kaas, 1990*a*). Thus, both mapping data and myeloarchitecture can be used to determine if recordings are from MT or one of the visual areas adjoining MT. In addition, as in macaque monkeys, the connections of MT with other parts of the visual system have been extensively studied in owl monkeys (see Discussion), allowing results to be interpreted in the context of an understanding of anatomical connections. Some of the results have been briefly reported elsewhere (Krubitzer & Kaas, 1989*b*).

Methods

The effects of removing part of area 17 on the responsiveness of neurons in MT were investigated in three adult owl monkeys, *Aotus trivirgatus*. The recording methods closely followed those used in the first mapping study of MT in primates (Allman & Kaas, 1971*a*), and the histological preparation of the tissue was similar to that recently described in a study of MT connections (Krubitzer & Kaas, 1990*a*).

In brief, each monkey was anesthetized for recording with an intraperitoneal injection of urethane (125 mg/100 g body weight) that was supplemented as needed to maintain a surgical level of anesthesia. The animal was placed in a stereotaxic apparatus so that the skull over the upper temporal and occipital lobe could be exposed, and the bone over the cortex just rostral to MT to the caudal pole of the hemisphere was removed. The opening in the skull was surrounded by a dam of acrylic plastic, filled with a viscous silicone fluid, and the dura was retracted. A high-resolution photograph of the vascular pattern of the cortex was obtained for citing electrode penetrations. The eyelids were retracted, the pupils dilated with cyclopentolate hydrochloride, and the surfaces protected from desiccation by a thin coating of silicone fluid (dimethylpolysiloxane). The eye contralateral to the exposed cortex was immobilized by suturing the sclera to a ring on a connecting rod which was cemented to the acrylic dam on the skull. A second rod cemented to the acrylic dam was fixed in an adjustable vice so that the stereotaxic apparatus could be removed, and the eyes would have unobstructed views of the visual field. The fixed eye was centered in a translucent plastic hemisphere, 60 cm diameter, which served as a screen for presenting visual stimuli. The other eye was covered, except for tests of binocular activity of neurons. A fiber optic system was used to briefly direct light into the eye so that retinal features including the optic disk and *area centralis* were projected onto the screen as references (see Fernald & Chase, 1971). Recordings were obtained from small clusters of neurons or occasionally from single neurons with tungsten microelectrodes of low impedances (1.0–1.5 MΩ). Multiunit recordings increased the probability of obtaining visually evoked activity at each recording site. Visual stimuli consisted of small to large (0.3–30 deg) spots and slits of light presented and moved on the surface of hemispheres with a handheld projector. Back-lit bars as dark shadows were also used. Neurons in MT of owl monkeys respond best to bars of narrow width (less than 1 deg), lengths equaling the size of the receptive field, and moderate rates of movement (about 10 deg/s) in the preferred direction (Felleman & Kaas, 1984). Before and after the area 17 lesions, neurons were tested for responsiveness with a range of optimal and suboptimal stimuli, including the onset and offset of full-field light. Most electrode penetrations were roughly perpendicular to the surface of cortex, and recordings were made at several depths through the thickness of cortex. However, receptive fields and detailed examinations of responsiveness were typically obtained for neurons in the middle layers of cortex, 400–800 μm in depth, where the most vigorous responses to visual stimuli were obtained. Electrode penetrations were placed in the expected locations of MT, area 17, and area 18. During recording, MT and the border between area 17 and area 18 were identified by patterns of retinotopic organization (Allman & Kaas, 1971*a*,*b*), and dorsomedial area 17 was removed by gentle aspiration. Cortex was removed down to the white matter. Immediately after aspiration and up to a period of 6 h thereafter, sites in MT were reexamined for responsiveness to visual stimuli, and receptive fields and response characteristics were obtained for sites where neurons remained responsive. Sites where neurons were unresponsive to visual stimuli were examined with an extended range of visual stimuli, and at a number of recording depths. In addition, electrodes were placed so that they usually alternated between responsive and unresponsive regions of MT. This procedure controlled for the possibility that temporal changes in the responsiveness influenced the results. Key recording sites were marked with small electrolytic lesions.

At the end of the recording session, each animal was given a lethal dose of sodium thiopental and perfused with 0.9% saline followed by 2% paraformaldehyde in saline with 10% sucrose. The brain was removed immediately after perfusion, and the cortex was separated from the brain stem. The caudal half of the experimental hemisphere was flattened by opening the sulci, separating the cortex from the underlying white matter, placing cuts at stress points, and placing the cortex under a lightly weighted glass plate (see Krubitzer & Kaas, 1990*a*). The flattened cortex was submerged in fixative in 30% sucrose and buffered saline. After 12–24 h, a flattened cortex containing MT and cortex caudal to MT was sectioned parallel to the surface at 40 μm. Sections were stained for myelin (Gallyas, 1979),

so that the extents of area 17, area 18, MT, and the lesion in area 17 could be determined.

Results

Partial lesions of area 17 in owl monkeys completely abolished the responsiveness of neurons to visual stimuli in retinotopically matched portions of MT, while normal responsiveness was preserved in other parts of MT. In addition, neurons at a few recording sites along the margin of the deactivated zone appeared to have slightly displaced receptive fields.

Recordings from area 17 and area 18

In all cases, electrophysiological recordings were made from parts of areas 17 and 18 on the dorsolateral surface of the occipital pole (Fig. 1). As reported previously (Allman & Kaas, 1971*b*, 1974), neurons in areas 17 and 18 responded vigorously to spots of light, moving bars, and shadows. Receptive fields for neurons in this location in area 17 were approximately 1–3 deg in diameter and were located in the central 10 deg of the lower visual quadrant. Receptive fields for neurons in area 18 were somewhat larger than those in area 17, ranging from 3–10 deg in diameter. As recording sites progressed from area 17 into area 18, receptive fields moved towards the vertical meridian. At the 17/18 border, the progression reversed and the receptive fields moved away from the vertical meridian. Thus, recording sites in area 17 could be distinguished from sites in area 18 by changes in receptive-field size as well as by reversals in progressions of receptive fields for rows of recording sites across the border.

The recordings in areas 17 and 18 were used to determine the border of the two fields so that lesions in area 17 could extend to the border without including area 18. The recordings also indicated the portion of the visual field represented in a region of cortex that was then ablated.

Recordings from MT before and after area 17 lesions

Before the lesions of area 17, neurons in MT had normal responsiveness to visual stimuli and MT had normal features of visuotopic organization. Receptive fields ranged from about 5 deg in diameter in central vision to over 20 deg in peripheral vision (Figs. 2–4). These values compare closely with those obtained in a previous mapping study of MT in owl monkeys

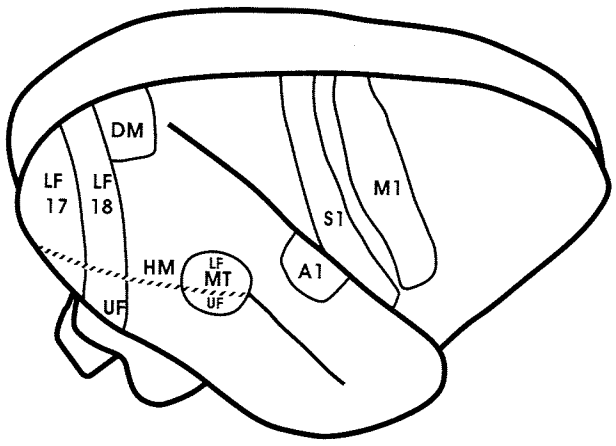

Fig. 1. A dorsolateral view of an owl monkey brain showing the locations of areas 17, 18, MT (the middle temporal area), and DM (the dorsomedial area). The broken line marks the extension of the representation of the horizontal meridian (HM) across the fields (the HM also forms the rostral border of area 18 or V-II). Medial portions of areas 17, 18, and MT are devoted to the lower visual quadrant, while lateral portions represent the upper visual quadrant. Primary auditory, somatosensory, and motor areas are shown for reference (A1, S1, M1). LF: lower field; and UF: upper field.

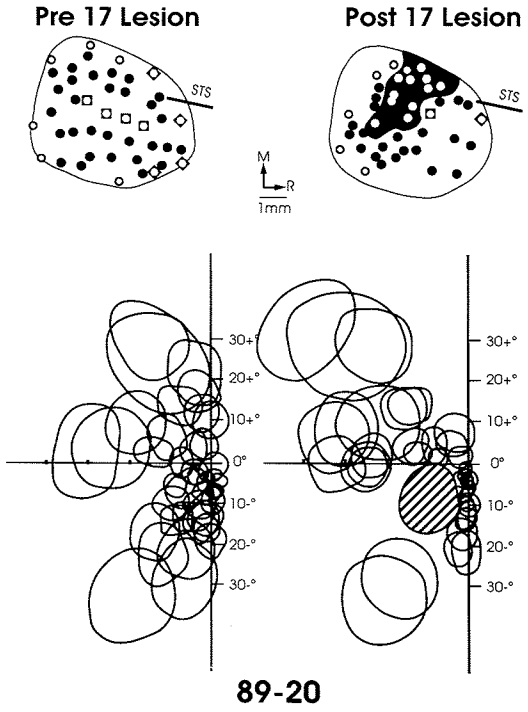

Fig. 2. The effects of area 17 lesions on the responsiveness of neurons in MT. Above: Duplicated outlines of MT determined from the myeloarchitecture in case 89-20. Electrode penetrations made before (left) and after (right) the area 17 lesion are marked by black or white dots. Circles mark sites where neurons had receptive fields along the zero vertical meridian; circles embedded in squares indicate sites with receptive fields along the zero horizontal meridian; circles embedded in diamonds mark sites with the most peripheral receptive fields. The black area on the right indicates that zone of MT presumed to be altered by the area 17 lesion according to retinotopic maps. The white dots in the black region (upper right) represent electrode penetrations where no responsive neurons were encountered. STS: superior temporal sulcus. Below: Receptive fields for neurons in MT before (left) and after (right) the area 17 lesions. The zero vertical and zero horizontal meridians through gaze were estimated from retinal landmarks (see Methods). The number of receptive fields exceeds the number of electrode penetrations because more than one recording site was sampled in some penetrations that angled slightly from medial to lateral with increasing depth. Cortex in the superior temporal nucleus was difficult to sample, and a gap in the receptive-field arrays resulted. This unexplored cortex is likely to also represent the peripheral visual field (Allman & Kaas, 1971*a*). The lesion removed a portion of area 17 representing the striped portion of the lower visual field on the right. Note that no receptive fields for MT neurons occupied this zone.

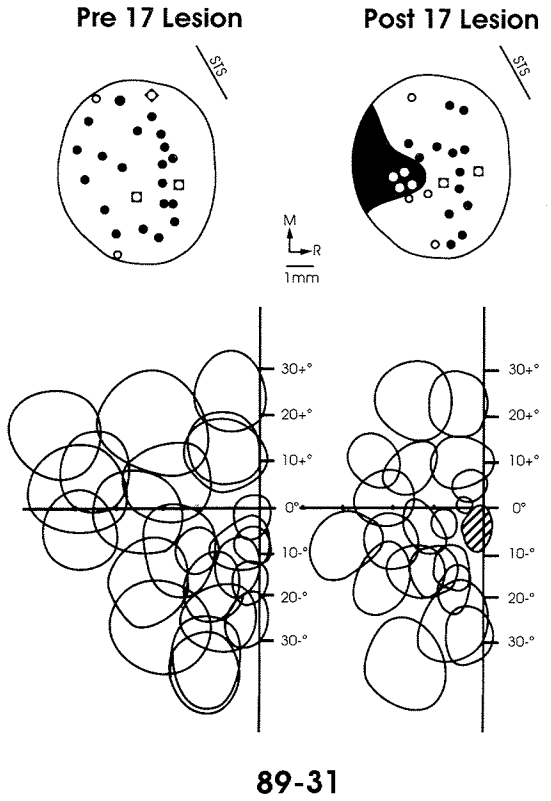

Fig. 3. Receptive fields for neurons in MT before and after a partial lesion of area 17 in case 89-31. Conventions are as in Fig. 2.

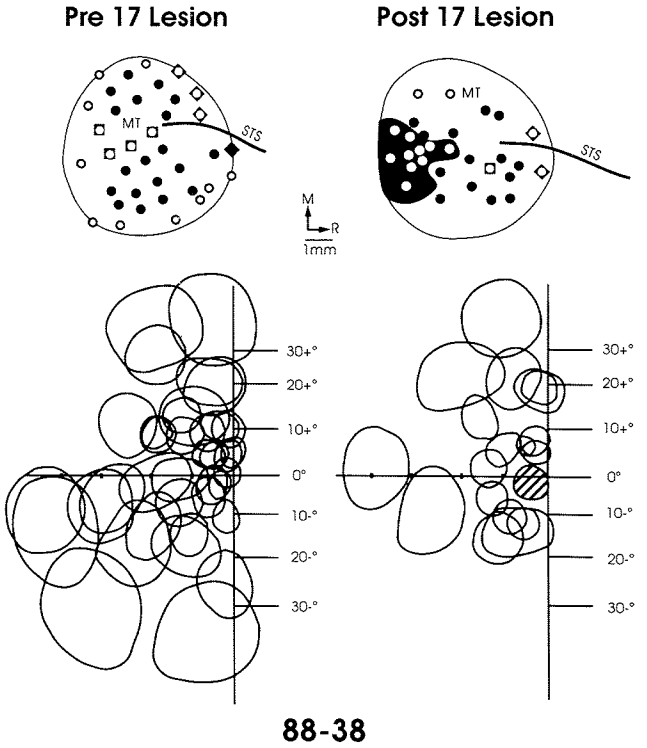

Fig. 4. Receptive fields for neurons in MT before and after a partial lesion of area 17 in case 89-38. Conventions are as in Fig. 2.

(Allman & Kaas, 1971*a*). In addition, as previously reported, neurons were activated by both eyes (Allman & Kaas, 1971*a*), and had strong preferences for specific directions of stimulus movement; narrow bars of less than 1 deg in width and of lengths approximating the receptive-field sizes were most effective (see Allman & Kaas, 1971*a*; Felleman & Kaas, 1984). The relationship of recording site locations to receptive-field locations in MT revealed a systematic representation of the contralateral hemifield. In close correspondence with previously published maps (Allman & Kaas, 1971*a*), central vision was represented caudally and peripheral vision rostrally in MT. The present maps of MT are incomplete because most of MT is devoted to central and paracentral vision and we did not explore the small portions of rostral MT devoted to peripheral vision.

Immediately after recording from area 17, area 18, and MT, dorsomedial striate cortex was removed by aspiration. In each case, the lesion extended to or near the border of area 17, but only marginally involved area 18 (Figs. 5 and 6). Each lesion extended to, but did not include, the white matter, and there was no evidence from recordings in MT that fibers from other parts of area 17 were disrupted. In case 89-20, the lesion (Fig. 5) included tissue that represented central and paracentral vision of the lower visual quadrant as far as 15 deg below the horizontal meridian and 15 deg from the zero vertical meridian (Fig. 2).

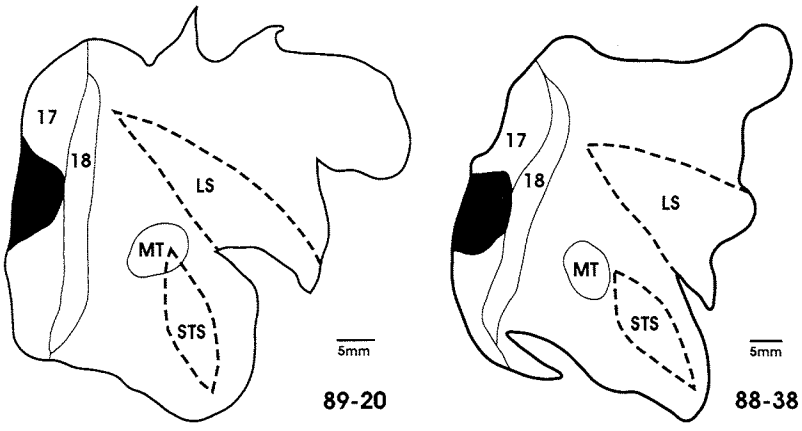

Fig. 5. The locations of lesions (black) of area 17 in flattened cortex of owl monkeys 89-20 and 88-38. Architectonic borders of areas 17, 18, and MT are indicated. The opened superior temporal sulcus (STS) and lateral sulcus (LS) are also marked.

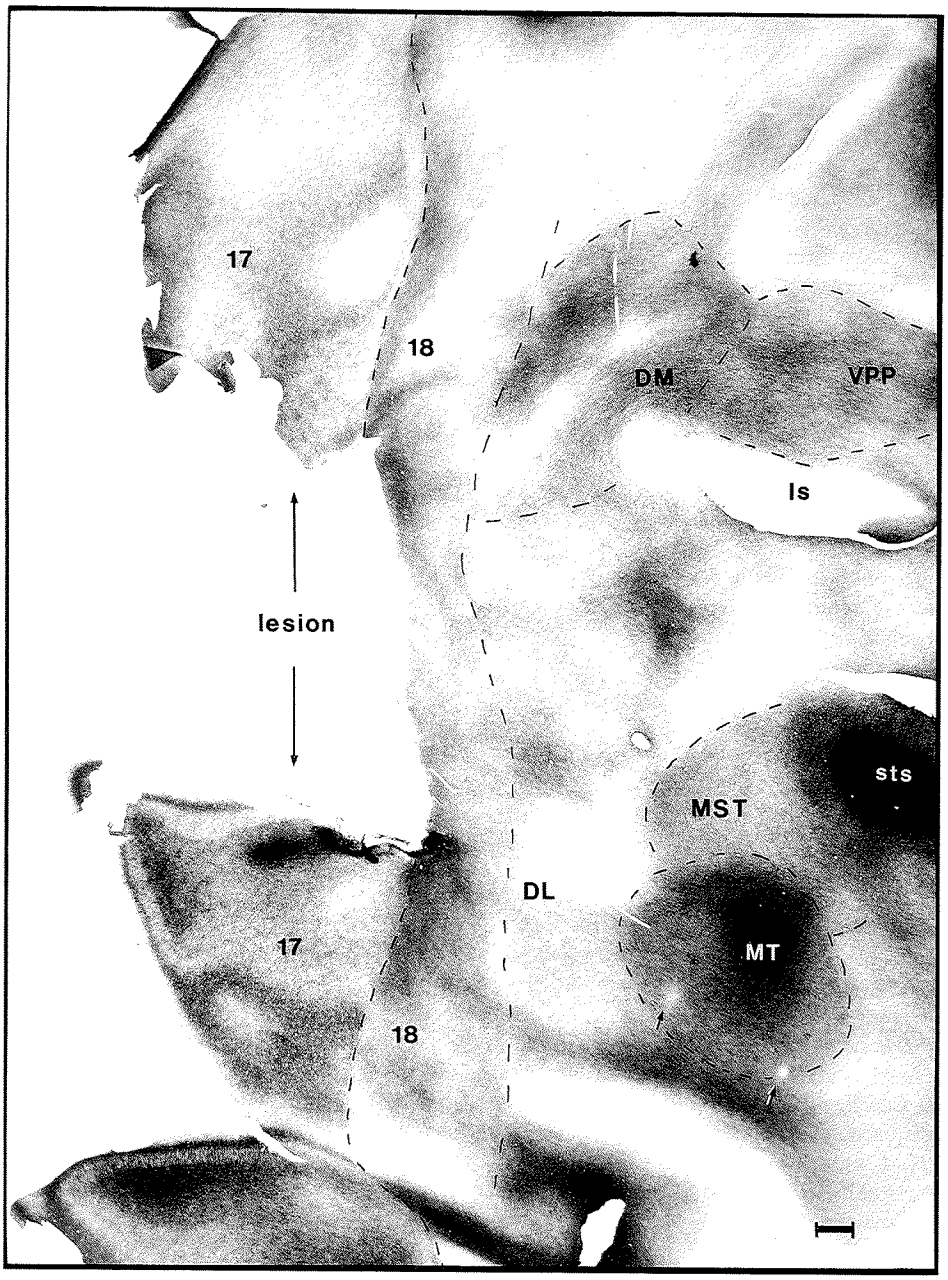

Fig. 6. The location of the lesion in area 17 of owl monkey 89-31. The brain section from flattened cortex is somewhat unevenly stained for myelin as different cortical depths are reflected. Nevertheless, the border between area 17 and 18 is obvious, and it can be seen that the lesion reaches the border of area 17 without including area 18. The results of microelectrode recordings designed to locate the border were used to guide placement of the lesion. MT is apparent as a densely myelinated oval on the right. Arrows point to two microlesions placed at recording sites judged to be near the border of MT. Such lesions allowed the electrophysiologic results to be related to cortical architecture. The borders of area 18, the dorsolateral visual complex (DL), the dorsomedial area (DM), the ventral posterior parietal area (VPP), and the medial superior temporal area (MST) were estimated from a series of sections. LS: lateral sulcus; STS: superior temporal sulcus; medial is up; rostral is right. Scale bar = 1 mm.

The lesion largely avoided the 17/18 border, and the representation along the zero vertical meridian was preserved. The lesion in case 88-38 included central vision and 2–3 deg of the upper visual quadrant, as well as 5 deg or so of the lower visual quadrant (Fig. 5). In case 89-31, the lesion involved the lower visual quadrant as far as 10 deg below the zero vertical meridian and about 5 deg from the zero vertical meridian. The rostral extent of the lesion closely approximated the 17/18 border (Fig. 6).

Immediately after the lesions in area 17, recordings were made throughout the MT region. In each case, a portion of MT was completely unresponsive to visual stimuli, while other portions were normally responsive. Neurons at the tested unresponsive sites did not respond to a variety of visual stimuli including moving bars of light shorter or longer than the receptive field, wide or narrow bars, or even the onset and offset of full-field light. The unresponsive sites did not reflect a period of general depression of responsiveness, since we typically alternated electrode penetrations from responsive to unresponsive portions of MT. Neurons in the responsive portions of MT could be activated by either eye, had normal receptive-field sizes, and demonstrated strong preferences for direction of stimulus movement. The neurons were highly responsive to moving bars of light. No differences from prelesion levels of responsiveness were noted in the responsive portions of MT.

The location of the unresponsive portion of MT retinotopically matched the location of the lesion in area 17 for each case. In case 89-20, the unresponsive zone was in a middle to dorsal (medial) sector of MT that represents paracentral vision of the lower quadrant (Fig. 2). A total of ten recording elec-

trode penetrations were placed in this unresponsive cortex, and no responses to visual stimuli were observed, even though previously effective stimuli were presented within expected receptive-field locations as well as other parts of the visual field. At each of these unresponsive sites, neurons were tested at several recording depths from 200–300 μm to 2 mm or more below the surface. In contrast, neurons in penetrations outside the unresponsive zone responded strongly to visual stimuli. In case 89-31, the unresponsive zone was not fully explored, but four electrode penetrations were placed in the caudal extent of this zone (Fig. 3), and no visually activated neurons were encountered at any recording depth through cortex. Again, neurons in nearby electrode penetrations were normally responsive to visual stimuli. Although the full extent of the unresponsive zone was not determined, the four unresponsive sites were in a midcaudal portion of MT known to represent paracentral vision. In the third case, 88-38, ten electrode penetrations were placed in a caudal portion of MT that was completely unresponsive to visual stimuli (Fig. 4). This cortex formerly represented central and paracentral vision near the zero horizontal meridian, including a small portion of the upper visual quadrant. Five electrode penetrations along the margin of the unresponsive zone encountered neurons that were highly responsive to visual stimuli.

As a final point, some neurons in recording sites along the edge of deactivated cortex appeared to have acquired receptive fields that were slightly displaced from their prelesion locations. In case 89-20, for example, five prelesion and five postlesion electrode penetrations were matched in location, and yet receptive fields were changed somewhat. Most notably, receptive fields *a–d* (Fig. 7) did not extend as far into the contralateral visual hemifield, a result simply explained by assuming that the lesion only partially deactivated the recorded neurons by removing parts of area 17 responsible for the more temporal parts of the receptive fields. However, the new receptive fields also extended further across the zero vertical meridian, suggesting perhaps that callosal inputs to MT had more of an activating role after ipsilateral inputs from area 17 were partially removed. The receptive field for recording site *e* was displaced upward in the visual field. This shift in the receptive field, as well as the extensions of receptive fields across the vertical meridian, could reflect plasticity in the map as a result of partially deactivating MT. These limited observations suggest that alterations in the effective receptive fields of some neurons in MT may follow partial lesions of area 17, but we did not attempt a systematic study of receptive-field changes.

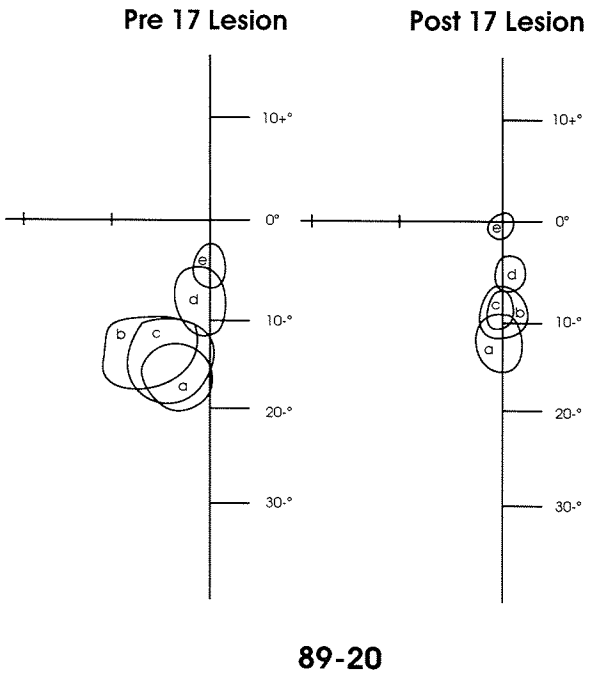

Fig. 7. Receptive fields for similar responsive recording sites in cortex caudal to the deactivated zone in MT of case 89-20 before (left) and after (right) deactivation. After the area 17 lesion, note the restriction of receptive fields to the region of the zero vertical meridian, the displacement of receptive fields into the ipsilateral hemifield, and new location of one receptive field at the center of gaze.

Myeloarchitecture of visual cortex and verification of ablations in area 17 and recording sites in MT

In all cases, cortex was flattened, cut parallel to the cortical surface, and stained for myelin. We chose this preparation because it allowed us to verify the total extent of the ablation in area 17 in a single section, without the inaccuracies introduced by serial reconstruction of coronal or parasagittally sectioned tissue. In addition, the architectures of area 17, area 18, and MT were readily apparent. Fig. 6 illustrates the myeloarchitecture of area 17 and shows the relation of the ablation of the dorsolateral surface of area 17 to the entire field. This figure also demonstrates that the lesion extended to the border of area 17 and that it did not spread significantly into area 18. In two additional owl monkeys, ablations were restricted to area 17 and did not include parts of area 18 (Fig. 5).

It was also important that the total extent of area MT could be identified in all cases so that recording sites with changes in neural responses following ablations of area 17 could be unambiguously assigned to MT and not adjacent cortical fields. MT is a densely myelinated circle of cortex surrounded by lightly or moderately myelinated cortex (e.g. Allman & Kaas, 1971*a*; Krubitzer & Kaas, 1990*a*). In the present investigation, our physiologically defined MT was coextensive with a darkly myelinated circle of cortex that could be easily defined, even in a single section (Fig. 6). Thus, neurons with changes in responses after ablations of area 17 were clearly in MT.

Discussion

Our main result is that partial lesions of area 17 in anesthetized owl monkeys deactivated retinotopically corresponding parts of MT. The recordings also confirm a finding, now over 20 years old, that the contralateral visual hemifield is systematically represented in MT (Allman & Kaas, 1971*a*). Since the initial study in owl monkeys, microelectrode mapping methods have been used to reveal similar retinotopic representations in MT of galagos (Allman et al., 1973); cebus monkeys (Fiorani et al., 1989); and macaque monkeys (Gattass & Gross, 1981; Albright & Desimone, 1987; Maunsell & Van Essen, 1987). The present results also suggested that some deactivated or partially deactivated neurons acquire new receptive fields, but the evidence is too fragmentary to be compelling and further study is needed.

Yet, such a demonstration of the potential of deactivated cortical neurons to acquire new receptive fields would not be surprising in that such changes have been demonstrated previously in visual, somatosensory, and auditory cortex (see Kaas, 1991, for review). The following discussion centers on the clear result, the partial deactivation of MT in owl monkeys, and why this result may differ from those reported for macaque monkeys (Rodman et al., 1989; Bullier & Girard, 1988).

The deactivation in owl monkeys

After partial lesions of area 17, neurons in retinotopically matched parts of MT failed to respond to visual stimuli, although they were spontaneously active. Neurons in other parts of MT responded normally to visual stimuli. This result is one that might be expected from what we know about the organization and connections of the visual system in owl monkeys. First, visual information that could activate cortex, as in other primates, is relayed from the retina to the lateral geniculate nucleus (LGN) (Kaas et al., 1978) and the superior colliculus (Kaas & Huerta, 1988). The relay from the LGN to area 17, as reported for macaque monkeys (Benevento & Yoshida, 1981; Bullier & Kennedy, 1983), is almost exclusively to area 17 (Kaas et al., 1976). The major cortical projections of area 17 are to area 18 (V-II) and MT (Lin et al., 1982), although area 17 also projects sparsely to the dorsomedial visual area, DM (Lin et al., 1982; Krubitzer & Kaas, 1990*b*). In addition, the dorsolateral portion of area 17 that is devoted to central vision projects to the dorsolateral visual complex, DL (see Weller & Kaas, 1985). MT receives additional inputs from the targets of area 17, that is area 18, DM, and to some extent DL, as well as other inputs, largely of the feedback type, from such fields as FST, MST, DI, posterior parietal cortex, inferotemporal cortex, and visuomotor fields of the frontal lobe (Wagor et al., 1975; Weller et al., 1984; Krubitzer & Kaas, 1990*a*; for macaques see Van Essen et al., 1981; Maunsell & Van Essen, 1983; Ungerleider & Desimone, 1986). However, lesions of area 17 remove almost all of the direct influence of LGN neurons on cortex, and other fields with feedforward projections to MT are likely to be highly dependent on area 17.

The superior colliculus is another potential source of activation of MT. Thalamic projections of the superior colliculus in owl monkeys and other primates are almost exclusively to the LGN and the inferior pulvinar complex (see Kaas & Huerta, 1988). The projections to the LGN would almost solely relay to area 17, and would be removed by an area 17 lesion. Extremely sparse projections of the superior colliculus to the lateral pulvinar have been noted in some studies in macaque monkeys (Harting et al., 1980), but not yet in New World monkeys. The lack of clear evidence for this projection in New World monkeys, and the apparent sparseness in Old World monkeys, makes superior colliculus inputs to the lateral pulvinar an extremely unlikely source of activation of visual cortex. Thus, if the superior colliculus is capable of activating MT, it is most likely to do so *via* the inferior pulvinar complex.

We have previously presented evidence that the traditional inferior pulvinar "nucleus" of owl monkeys and other primates consists of three separate nuclei, the "medial," "central," and "posterior" nuclei of the inferior pulvinar complex; IPm, IPc, and IPp (Lin & Kaas, 1979, 1980; see also Kaas & Huerta, 1988). The superior colliculus projects densely to IPp and IPc, but sparsely if at all to IPm. IPm projects densely to MT (Lin & Kaas, 1980; also see Ungerleider et al., 1984), but without a notable superior colliculus input, this projection would not seem to be a possible route for the superior colliculus to activate MT. IPp projects to cortex rostral and rostralateral to MT, possibly to areas such as FST and MST, but if this source of visual input to cortex activates MT, it would be multisynaptic and over feedback connections (see Maunsell & Van Essen, 1983; Krubitzer & Kaas, 1990*a*). IPc projects to area 18 and several fields bordering area 18. These fields have feedforward projections to MT, as well as projections to IPm, which relays to MT. Thus, if the superior colliculus activates MT, then it would most likely do so over projections to IPc, then to caudal visual cortex, and then to MT directly or indirectly *via* cortical projections to IPm. This route would seem to provide only a weak source of activation, partly because of the number of synaptic steps, and partly because of the apparent sparseness of the local terminations of the widespread IPc projections (Lin & Kaas, 1980). Also, in macaque monkeys, visually evoked activity in the inferior pulvinar is nearly completely eliminated by area 17 lesions (Bender, 1983), and at least part of the target of IPc, area 18 or V-II, is unresponsive to visual stimuli after area 17 deactivation (Schiller & Malpeli, 1977; Girard & Bullier, 1989).

Given the anatomy of the visual system, the total elimination of responsiveness to visual stimuli was what we expected after area 17 lesions in owl monkeys. However, we would also expect this effect in other primates, but MT appears to preserve some responsiveness to visual stimulation after area 17 deactivation in macaque monkeys (Rodman et al., 1989; Bullier & Girard, 1989).

Why do results in owl and macaque monkeys differ?

While it is important to consider explanations for the difference in results for owl monkeys and macaque monkeys, it is also important to stress that aspects of the results are highly similar. Lesions or other deactivations of area 17 abolish (owl monkeys) or severely depress (macaque monkeys) the responsiveness of neurons in MT to visual stimuli. Rodman et al. (1989) reported that about one-third of the sampled neurons in MT were completely unresponsive to visual stimuli, while the responses of other neurons were weaker than normal. In a brief report, Bullier and Girard (1988) stated that a number of neurons in MT remained active after cooling area 17, implying that most neurons were depressed or became unresponsive. Maunsell et al. (1990) reported that evoked neural activity in MT was highly dependent on the magnocellular layers of the LGN, and somewhat dependent on the parvocellular layers, while all of a small sample of MT neurons were completely unresponsive after a block of all LGN layers. Thus, there is agreement across reports that area 17 inputs are profoundly important for maintaining responsiveness to visual stimuli in MT. On the other hand, both Rodman et al. (1989) and Bullier and Girard (1988) report that many neurons in MT remain responsive after deactivations of area 17, and that features of retinotopic organization, receptive-field size, direction selectivity, and binocularity are preserved.

Given that the inputs to MT from area 17 are important, why did lesions of area 17 abolish visual responsiveness in owl monkeys but not in macaque monkeys? There are several obvious possibilities. One possibility for remaining responsiveness in macaques is that the lesions or deactivations of area 17 were

incomplete and that the preserved responsiveness of neurons in MT was a result of intact area 17 inputs. Even rather extensive lesions of area 17 in macaque monkeys may deactivate only a small portion of MT, given that receptive fields are considerably larger in MT than area 17, and area 17 emphasizes central vision much more than MT (e.g. see Gattass & Gross, 1981). It seems possible, for example, that the half of area 17 on the dorsolateral surface, which represents only the central 5 deg of vision (see Weller & Kaas, 1983), could be removed without deactivating any or much of MT. If some plasticity occurs so that previously ineffective inputs become effective (Fig. 7; also see Kaas et al., 1990), then even larger lesions could leave most or all of MT responsive to area 17 inputs. However, inputs from remaining portions of area 17 cannot account for all of the responsiveness of neurons in the MT region, since responsive neurons remained after extensive and complete removals of area 17 (Rodman et al., 1990).

Another possibility is that recordings from macaque monkeys were not from MT. Prelesion maps of MT were not attempted, and the architectonics of MT were not illustrated. Thus, it is difficult to determine for certain if recording sites in the deep superior temporal fissure were from MT. Although such a possibility could explain the disparity in results between owl and macaque monkeys, we would still have to account for the preserved responsiveness of cortex near MT and the finding that the neurons had response characteristic features that are like those of MT neurons. Neurons in the superior temporal polysensory area of macaque monkeys remain responsive to visual stimuli after area 17 lesions, and this responsiveness depends on the superior colliculus (Bruce et al., 1986), but this cortex is not close to MT, and the response properties of neurons in this cortex are quite different from those in MT.

A third possibility is that after area 17 lesions, inputs relayed from the superior colliculus to the inferior pulvinar complex and ultimately to MT become more potent over time. Bender (1983) found that the immediate effect of area 17 lesions was to totally abolish visual responses in the inferior pulvinar. Clearly, such deactivated neurons in the inferior pulvinar cannot account for the responsiveness of those neurons in MT recorded immediately after deactivating area 17 (Bullier & Girard, 1988; Rodman et al., 1989). However, with survival times longer than 3 weeks, some 15% of neurons recorded in the inferior pulvinar were activated by visual stimuli, and thus they could contribute to responses in MT in those cases with recordings longer than 3 weeks after area 17 removal (Rodman et al., 1989). Nevertheless, any input from the recovered inferior pulvinar would differ greatly from normal. Neurons with recovered responsiveness in the inferior pulvinar lacked selectivity to stimulus orientation or direction of movement (Bender, 1983) and they thereby fail to resemble neurons in MT (e.g. Felleman & Kaas, 1984) or neurons in area 17 that project to MT (Movshon & Newsome, 1984). Thus, cortical circuits would have to recreate normal response properties for neurons in MT from quite different types of inputs and from a very sparse population of active neurons. Nevertheless, Rodman et al. (1990) present evidence from one monkey that neurons responsive to visual stimuli persist in MT even after complete bilateral removal of area 17, and they report that lesions of the superior colliculus eliminate the responsiveness of MT neurons that remains after area 17 lesions. Thus, the preserved responsiveness of neurons in MT appears to depend on the superior colliculus rather than intact portions of area 17.

Other explanations for the differences in results include procedural and species differences in the experiments. All recorded animals were anesthetized, but the owl monkeys were anesthetized with urethane while the macaque monkeys were anesthetized with nitrous oxide or nitrous oxide plus halothane. There is some evidence that even moderate levels of halothane anesthesia abolishes the remaining responsiveness of MT neurons after area 17 deactivation (Bullier, personal communication), and thus levels as well as kinds of anesthesia may be critical. In addition, most, but not all, of the recordings in macaque monkeys were done after periods of recovery, and it is now known that previously ineffective inputs to sensory cortex can gain effectiveness over periods of hours to months (see Kaas, 1991, for review). It is possible that under different recording conditions, MT neurons would be responsive to some extent after area 17 lesions in owl monkeys, or that with urethane anesthesia and immediate recording, MT neurons would be unresponsive in macaques. It is also possible that owl monkeys and macaque monkeys differ somewhat in the effectiveness of extrastriate sources of activating MT. Further experiments can resolve some of these questions. Yet, results across studies support the clear and reasonable conclusion that neurons in MT of both New World and Old World monkeys are highly dependent on area 17 for visual activation.

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