Analysis of Visual Behavior

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18 Two Cortical Visual Systems

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Damage to the primary visual cortex (striate cortex or area 17), either by disease, by acute assault, or through surgical intervention for therapeutic purposes, results in a scotoma or blind region in the visual field. Moreover, because of the precise topographic mapping of the visual field onto striate cortex, the region of the scotoma corresponds to the part of striate cortex damaged. In contrast to this primary sensory impairment, "higher-order" visual dysfunctions ensue when damage occurs more rostrally in the brain, in either the temporal or parietal lobes; however, the effects that follow lesions of these two cortical areas are markedly different (Newcombe and Russell 1969). Whereas damage to temporal cortex produces an impairment in visual recognition (see, for example, Milner 1958, 1968; Kimura 1963; Lansdell 1968; Benson et al. 1974; Meadows 1974), damage to parietal cortex produces a constellation of visual spatial impairments (see, for example, McFie et al. 1950; Semmes et al. 1963; De Renzi and Faglioni 1967; Butters et al. 1972; Ratcliff and Davies-Jones 1972; Ratcliff and Newcombe 1973).

Although it is clear that visual information must reach the temporal and parietal association areas to enable their participation in visual recognition and visual spatial perception, respectively, the complex circuitry through which this information is transmitted has yet to be unraveled. It is known, however, that two major fiber bundles emerge from occipital cortex and project rostrally in the brain (Flechsig 1896, 1920). One, the superior longitudinal fasciculus, follows a dorsal path, traversing the posterior parietal region in its course to the frontal lobe; the other, the inferior longitudinal fasciculus, follows a ventral route into the temporal lobe. It has been our working hypothesis (Mishkin 1972; Pohl 1973) that the ventral or occipitotemporal pathway is specialized for object perception (identifying *what* an object is) whereas the dorsal or occipitoparietal pathway is specialized for spatial perception (locating *where* an object is). This distinction between the two types of visual perception is not new (see, for example, Ingle 1967; Held 1968). In the past, however, the neural mechanisms underlying object and spatial vision were seen as localized in geniculostriate and tectofugal systems, respectively (Schneider 1967; Trevarthen 1968).

By contrast, in the present formulation, corticocortical connections originating in the striate area are viewed as mediating both types of vision, with two diverging cortical systems replacing the geniculostriate-tectofugal dichotomy. The reasons for stressing cortico-cortical mechanisms for both types of visual perception in primates are developed below, but this emphasis is not meant to deny that the tectofugal system (including the tectofugal pathway to cortex) contributes to spatial vision, particularly to its visuomotor aspects; it is simply that, with regard to the perceptual aspects of spatial vision, the tectofugal system in the primate appears to play a subsidiary role. In our investigations of the two cortical visual systems, we have used the rhesus monkey (*Macaca mulatta*) as our subject and have employed a combination of behavioral, electrophysiological, and anatomical techniques.

Occipitotemporal Mechanisms in Object Perception

The results of numerous behavioral studies have demonstrated that bilateral removal of inferior temporal cortex in monkeys produces a severe impairment in visual-discrimination performance (for reviews see Gross 1973; Dean 1976; Wilson 1978). In brief, inferior temporal lesions produce a deficit that is exclusively visual, affecting both the retention of discriminations acquired prior to surgery and the postoperative acquisition of new discriminations. Among the deficits that have been reported are those involving hue, brightness, two-dimensional patterns, and three-dimensional shapes. More recent work has shown that damage to the posterior part of the inferior temporal cortex (area TEO) interferes mainly with discriminative ability, whereas damage to the anterior part (area TE) affects primarily visual memory (Iwai and Mishkin 1968; Cowey and Gross 1970).

The pathway through which inferior temporal cortex receives visual information was first suggested on the basis of early neuronographic data (von Bonin et al. 1942). It had been shown in both the monkey and chimpanzee that if strychnine is applied to the striate cortex spike discharges can be recorded from a prestriate cortical belt, whereas if strychnine is applied to any part of this prestriate region spikes can be recorded in the inferior part of the temporal lobe. These neuronographic findings were later confirmed in a neuroanatomical study (Kuypers et al. 1965) that employed the Nauta-Gygax (1954) technique for tracing projections by the silver-staining of degenerating axons after removal of their cell bodies. As anticipated, it was found that striate cortex projects to a prestriate cortical belt, which, in turn, projects to the inferior temporal area. It was also confirmed that each prestriate area projects across the splenium of the corpus callosum to reach the prestriate area of the opposite hemisphere.

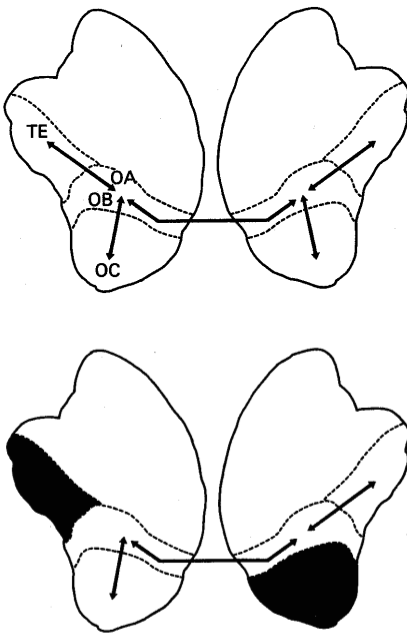These neuronographic and neuroanatomical findings are sche-

Figure 18.1 Striate-prestriate-temporal pathways. Abbreviations refer to cytoarchitectonic divisions of von Bonin and Bailey (1947). Lower diagram indicates pathways remaining after crossed striate and inferior temporal lesions (in black). Pathways are shown as two-way, in accord with the neuroanatomical evidence that the connections are reciprocal (Kuypers et al. 1965; Rockland and Pandya 1979). Adapted from Mishkin 1966.

matized in figure 18.1. As indicated in the upper diagram, each striate area transmits visual information, relayed through the prestriate cortex, to the ipsilateral inferior temporal area; but, in addition, because of the reciprocal prestriate connections across the corpus callosum, each striate area also transmits visual information to the contralateral inferior temporal area. The behavioral significance of these pathways was first demonstrated in a series of crossed-lesion disconnection experiments (Mishkin 1966). Since such disconnection studies are not commonly employed to investigate brain-behavior relationships, a brief explanation of the underlying logic may be in order.

The usual method of identifying the function of a cortical area is to remove that same area from both hemispheres and then determine what function has been lost. This method does not reveal whether the function was lost because it depended on the area (or station) that was removed or because it depended on a later station along the same pathway that could no longer operate after the pathway leading to it was destroyed. Crossed-lesion disconnection studies make it possible to demonstrate that a pathway with many cortical stations exists. The method is to remove one of the stations in the postulated pathway on one side of the brain, and to remove a different one (later in the pathway) on the other side (see lower diagram of figure 18.1). This leaves

one of each pair in the series intact, and it can often be shown that up to this point little or no loss in function occurs. However, if the connections between the two hemispheres are then cut, the functions served by the stations beyond the cut may be lost. This demonstrates that the function in question depends not only on the stations themselves, but also on the connections between them. The same demonstration theoretically could have been achieved by cutting the fiber connections between the stations within each hemisphere, but the complexity of these connections is so great, and they form such a compact network, that the method is technically difficult if not impossible.

In the disconnection study referred to above it was found that if an inferotemporal lesion in one hemisphere was combined serially with total striate removal in the other, animals continued to perform a pattern-discrimination task; however, when these asymmetrical or crossed lesions were followed by transection of the corpus callosum, the performance of the animals fell to chance and they failed to relearn the task. Presumably, the single crossed pathway from the intact striate cortex on one side across the corpus callosum to the intact inferior temporal cortex on the other side is sufficient to mediate pattern-discrimination habits, but if this pathway is cut a severe deficit results. This study thus provided the first behavioral indication that inferior temporal cortex is a late station along a cortical visual pathway running from striate through prestriate cortex.

Electrophysiological experiments (Gross et al. 1972) subsequently revealed that single neurons in inferior temporal cortex, like those in striate (Hubel and Wiesel 1968) and prestriate (Hubel and Wiesel 1970) cortex, have visual receptive fields. The optimal trigger features for inferior temporal neurons, however, are considerably more complex. That visual input to these neurons originates in striate cortex was shown in a combined recording and ablation study by Rocha-Miranda et al. (1975). The method entailed measuring the defect in the visual receptive fields of inferior temporal neurons after selective cerebral lesions or commissural transections. As predicted from earlier ablation studies (Mishkin 1972), the tectofugal pathway from the superior colliculus through the pulvinar to cortex turned out to be unimportant for the receptive field properties of inferior temporal neurons. By contrast, the cortical pathway from striate through prestriate to inferior temporal cortex proved to be essential, since interruption of this pathway by a striate removal or by transection of the forebrain commissures eliminated the corresponding visual input to inferior temporal cells. The results, shown in figure 18.2, indicate that normally over 60% of inferior temporal neurons have bilateral receptive fields. However, after bilateral striate-cortex removal these neurons are totally unresponsive to visual stimulation, after unilateral striate-cortex removal they respond to visual stimulation only in the hemifield opposite the intact striate cortex, and after commissurotomy they respond to stimulation only in the con-
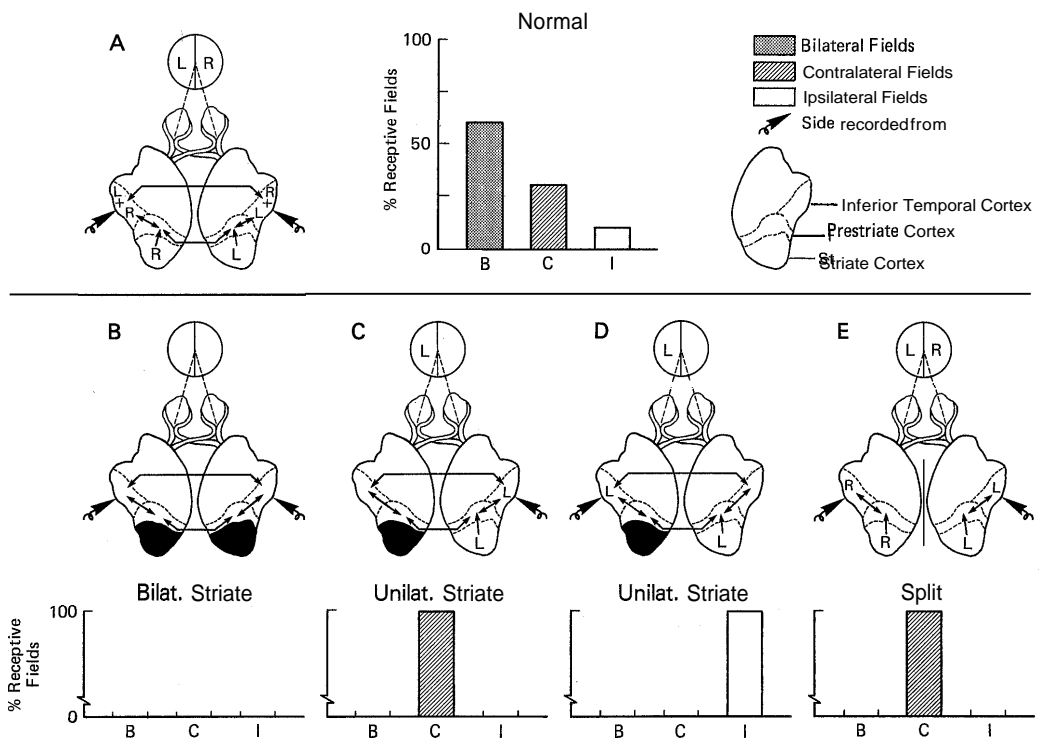
Figure 18.2 Proportion of inferior temporal neurons that had bilateral, contralateral, or ipsilateral receptive fields in (A) normal monkeys, (B) monkeys with bilateral removal of striate cortex, (C, D) monkeys with unilateral removal of striate cortex, and (E) monkeys with transection of the forebrain commissures. The brain diagrams show how information from the right (R) and left (L) visual hemifields could reach inferior temporal cortex along a corticocortical route and how each lesion (in black) interferes with this pathway. Adapted from Rocha-Miranda et al. 1975.

tralateral hemifield. Thus, the dependence of inferior temporal cortex on corticocortical connections arising in striate cortex has now been made evident at the single-cell level.

Most recently, this entire visual pathway was functionally mapped using ^{14}C-labeled 2-deoxyglucose (2-DG) via the method developed by Sokoloff and his colleagues (Kennedy et al. 1975). In this technique 2-DG is used as a marker of local cerebral glucose utilization and hence indicates regions that are metabolically active during the experimental procedure. The visual-mapping studies (Jarvis et al. 1978; Kennedy et al. 1978) were carried out in awake monkeys previously prepared with a unilateral optic-tract section combined with transection of the forebrain commissures (a procedure that visually deafferented one hemisphere while leaving the other intact). On the day of the experiment, the monkeys were presented with visual patterns in a rotating drum or in a discrimination apparatus. In both situations, reduced glucose utilization in the blind as compared with the seeing hemisphere was seen

cortically, not only in the geniculostriate projection but throughout the entire expanse of prestriate and inferior temporal cortex as far forward as the temporal pole. With one exception, no other cortical area in the deafferented hemisphere showed reduced activity.

The evidence that has been cited favors a sequential-activation model for object vision in which information that reaches the striate cortex is transmitted for further processing to the prestriate cortex, and from there to the inferior temporal area. This system appears to be important for the analysis and coding of the physical dimensions of visual stimuli needed for their identification and recognition. It is unlikely, however, that any part of this system up to and including the inferior temporal area is involved in the still higher-order process of associating visual stimuli with other events, such as motivational and emotional ones. Recordings from single cells in monkeys performing visual discrimination and reversal tasks have shown that although inferior temporal neurons are sensitive to the physical properties of stimuli, they are relatively insensitive to changes in the reward value of the stimuli (Jarvis and Mishkin 1977). Similar results have been reported by others (Rolls et al. 1977; but see Ridley and Ettlinger 1973). Presumably, the process of attaching reward value to a stimulus depends on stations beyond the occipitotemporal pathway (Jones and Mishkin 1972; Sunshine and Mishkin 1975; Spiegler and Mishkin 1978; Rolls et al. 1979b; Sanghera et al. 1979). However, for object vision, the inferior temporal cortex may well be the final station. It is significant in this regard that, by virtue of the extremely large receptive fields of inferior temporal neurons (Gross et al. 1972), this area provides the neural mechanism of stimulus equivalence across retinal translation (Gross and Mishkin 1977; Seacord et al. 1979)—that is, the ability to recognize a stimulus as the same regardless of its position in the visual field and, by extrapolation, regardless of its spatial location. Indeed, a necessary consequence of this equivalence mechanism is that within the occipitotemporal pathway there is a loss of information about the spatial locations of objects.

Occipitoparietal Mechanisms in Spatial Perception

The neural mechanism for the analysis of the spatial locations of objects also entails the transmission of visual information from the striate through the prestriate area; however, the rest of the pathway for spatial vision appears to be quite separate from the ventral pathway into the temporal cortex. Evidence in support of this dichotomy of cortical visual systems comes from recent studies in our laboratory on the parietal lobe.

In the initial study of this series, Pohl (1973) demonstrated a dissociation of visual deficits after inferior temporal and posterior parietal lesions. Whereas the temporal but not the parietal lesion produced a severe impairment on an object discrimination task, just the reverse was

found on tests in which the animal was required to choose a response location on the basis of its proximity to a visual "landmark" (see part A of figure 18.5). The results suggested that ". . . the inferior temporal cortex participates mainly in the acts of noticing and remembering an object's qualities, not its position in space," and that "conversely, the posterior parietal cortex seems to be concerned with the perception of spatial relations among objects, and not their intrinsic qualities" (Mishkin 1972).

Accumulating evidence from other laboratories supports this view of posterior parietal function. Not only has the impairment of landmark tasks after posterior parietal lesions been corroborated (Milner et al. 1977; Ungerleider and Brody 1977; Brody and Pribram 1978; however, see Ridley and Ettlinger 1975), but impairments after such lesions have also been found on other visual spatial tasks, including a stylus maze (Milner et al. 1977), patterned-string tests (Ungerleider and Brody 1977), cage finding (Sugishita et al. 1978), and route following (Petrides and Iversen 1979). Visual spatial disorientation, however, is not the only deficit produced by lesions of this region. Indeed, the classical symptoms of posterior parietal dysfunction are misreaching in the dark as well as in the light (Ettlinger and Wegener 1958; Bates and Ettlinger 1960; Ettlinger and Kalsbeck 1962; Hartje and Ettlinger 1973; LaMotte and Acuña 1978; Faugier-Grimaud et al. 1978; Stein 1978), contralateral neglect of auditory and tactile as well as visual stimuli (Denny-Brown and Chambers 1958; Heilman et al. 1970), and impairments of tactile discrimination (Blum 1951; Pribram and Barry 1956; Wilson 1957, 1975; Ettlinger and Wegener 1958; Pasik et al. 1958; Bates and Ettlinger 1960; Wilson et al. 1960; Ettlinger and Kalsbeck 1962; Ettlinger et al. 1966; Moffett et al. 1967; Moffett and Ettlinger 1970; Ridley and Ettlinger 1975).

The posterior parietal lesions in these studies have nearly always included two or even more cytoarchitectonic areas, and so it is natural to assume that the heterogeneity of effects is a consequence of the heterogeneity of the tissue included in the removals. However, the few studies that have directly examined this question (Moffett et al. 1967; Ridley and Ettlinger 1975) failed to uncover any clear-cut instance of functional dissociation. From the evidence at hand, the most parsimonious interpretation is one that has been offered by Semmes (1967), namely, that the multiple deficits are but different reflections of a single, supramodal spatial disorder (see also Ratcliff et al. 1977). According to this view, and in the light of more recent anatomical information (Pandya and Kuypers 1969; Jones and Powell 1970), a supramodal spatial framework could be constructed out of converging' inputs to posterior parietal cortex from all exteroceptive sensory modalities, with a significant contribution from vision. In the studies in our laboratory that followed the "landmark" experiment by Pohl (1973), we addressed the questions raised above regarding localization of spatial

function within the posterior parietal region and the dependence of this function on visual inputs.

As in most other posterior parietal ablation studies, the lesions in Pohl's study were composites that included not only inferior parietal but also dorsal prestriate tissue. To test for localization of function within this region, we partitioned it into three approximately equal sectors—inferior parietal, lateral preoccipital, and medial parietal-preoccipital—as shown in figure 18.3. Monkeys were then prepared with lesions of either one, two, or all three of these sectors and tested on the landmark, or distance discrimination, task (Mishkin, Lewis, and Ungerleider, in prep.). Like the earlier attempts, this one failed to reveal any evidence of functional specialization within the larger area. Instead, there was an especially clear relationship between severity of effect and extent of lesion, completely independent of locus. This finding is illustrated in figure 18.4.

The fact that performance on the landmark task depends as much on dorsal prestriate tissue as on inferior parietal tissue pointed to the possibility that the visual information on which posterior parietal cortex operates arises in striate cortex. The alternative possibility, that the critical visual input arises in the superior colliculus, found no clear support in a study of the effects of tectal lesions on landmark discrimination (Snyder and Mishkin, unpublished data); even complete destruction of the superior colliculus failed to produce a reliable loss in retention. Therefore, to test for the functional dependence of posterior parietal cortex on striate output, a crossed-lesion disconnection experiment analogous to the striate-temporal disconnection experiment described earlier (Mishkin 1966) was undertaken (Ungerleider and Mishkin 1978a). Monkeys were preoperatively trained on the landmark task, and then received a three-stage operation intended to serially disconnect posterior parietal from striate cortex. At each stage, the monkeys were given a preoperative retention test followed by surgery, allowed 2 weeks to recover, and then retested.

As shown in part B of figure 18.5, the first removal was a unilateral posterior parietal ablation, which included all three sectors investigated in the partial-lesion study to ensure a complete effect. On the hypothesis that each striate area has corticocortical connections with both posterior parietal areas, this first lesion may be viewed as a test of the effects of destroying two of these connections (the remaining two are indicated by the black arrows in the figure). A comparison of preoperative with postoperative retention scores on the task shows that this amount of damage to the system had only minimal effect.

The second-stage removal was a contralateral striate-cortex ablation, illustrated in part C of figure 18.5. It included all of the striate cortex, both laterally and medially. The results of the retest given after this operation revealed a severe impairment; the monkeys required an average of 880 trials to relearn, despite the remaining interhemispheric cor-
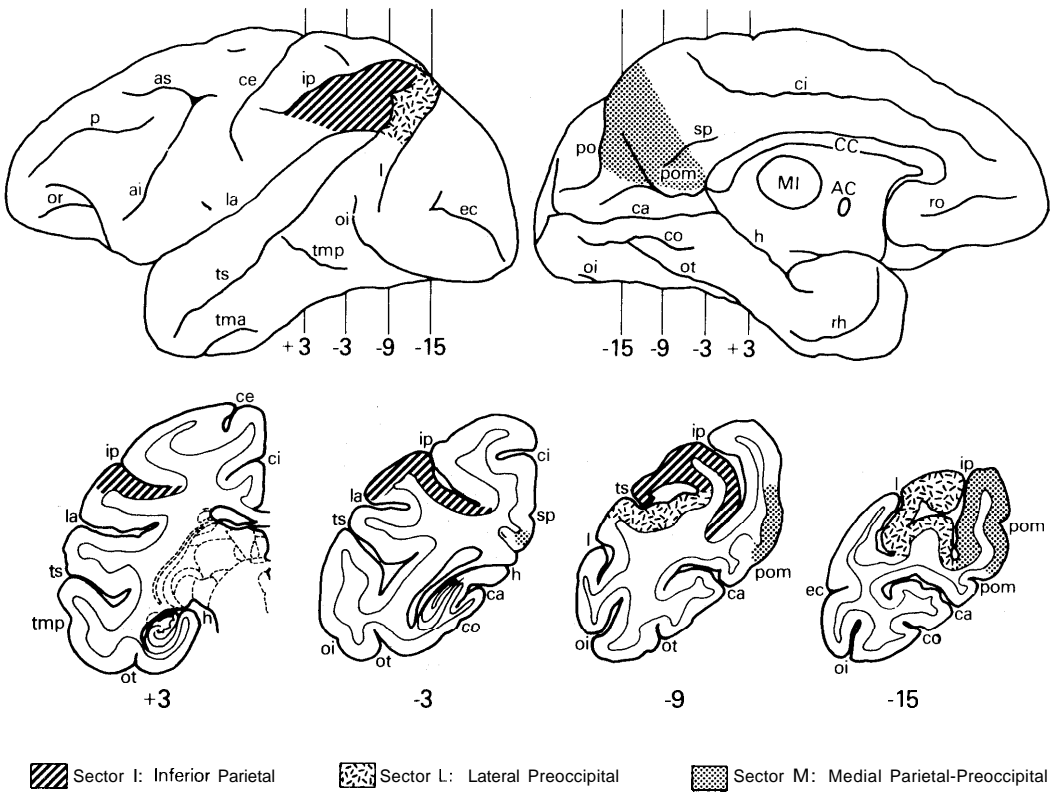
Figure 18.3 Partition of posterior parietal cortex into three sectors: inferior parietal (I), lateral preoccipital (L), and medial parietal-preoccipital (M). Monkeys received bilaterally symmetrical lesions intended to remove one (groups I, L and M), two (groups IL, LM, and IM), or all three (group PP) of these sectors. The sectors are shown on lateral and medial surface views and on representative cross-sections of a standard rhesus monkey brain. Numerals indicate approximate A-P stereotaxic levels. AC: anterior commissure. ai: inferior arcuate sulcus. as: superior arcuate sulcus. ca: calcarine fissure. CC: corpus callosum. ce: central sulcus. ci: cingulate sulcus. co: collateral sulcus. ec: ectocalcarine sulcus. h: hippocampal fissure. ip: intraparietal sulcus. l: lunate sulcus. la: lateral fissure. MI: massa intermedia. oi: inferior occipital sulcus. or: orbital sulcus. ot: occipitotemporal sulcus. p: principal sulcus. po: parieto-occipital incisure. pom: medial parieto-occipital fissure. rh: rhinal fissure. ro: rostral sulcus. sp: subparietal sulcus. tma: anterior middle temporal sulcus. tmp: posterior middle temporal sulcus. ts: superior temporal sulcus.
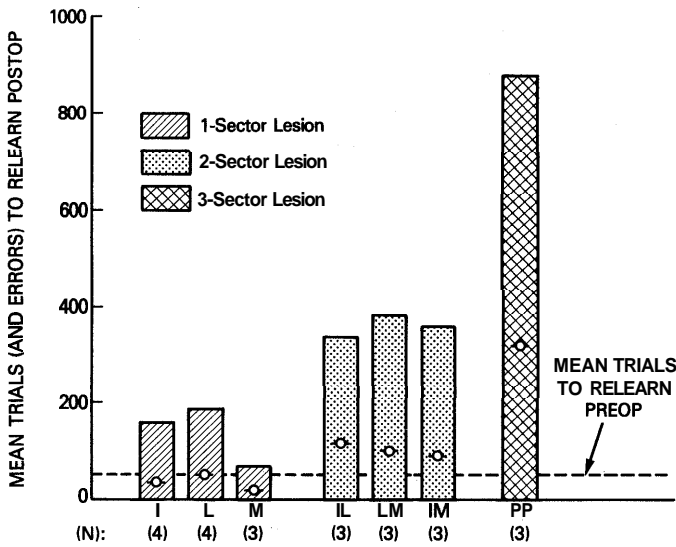
Figure 18.4 Postoperative retention on the landmark task after bilateral, lesions of one, two, or three posterior parietal sectors, illustrated in Figure 18.3. Bars indicate mean trials to criterion; circles within bars indicate mean errors. N in this and following figures equals number of monkey in group. Mean number of trials to relearn task before operation (dashed line) is based on scores on a two-week retention test given just prior to surgery and represents the data from all 23 monkeys. The results showed no significant differences among the groups given a one-sector lesion, or among the groups given a two-sector lesion. However, as the number of sectors included in the posterior parietal lesion increased, so did the severity of the impairment. (Source: Mishkin et al., in prep.).

tical connections between the intact striate and parietal areas. This suggested that the interaction between the striate and parietal cortex via uncrossed connections had been of considerable importance for performance. However, whether such interaction was mediated by a corticocortical pathway could only be determined by examining the effects of the third stage of operation, transection of the corpus callosum.

In this operation, the posterior half of the callosum was transected, thereby cortically disconnecting the intact striate and posterior parietal areas. As shown by the postoperative retention score in part D of figure 18.5, the effect of the transection was to produce a partial reinstatement of the impairment. We can infer from these results that performance on the landmark task had indeed been mediated via corticocortical pathways between the striate and parietal areas.

However, the disruption of landmark discrimination that followed this lesion was not complete; all monkeys successfully relearned the task. At this point, the question was whether recovery of the discrimination was mediated by the interaction of the intact posterior parietal cortex with subcortical visual structures or by the interaction of the intact striate cortex with other cortical areas. In subsequent work on this preparation we found that removal of the remaining posterior parietal cortex was completely without effect, which indicated that tectofugal
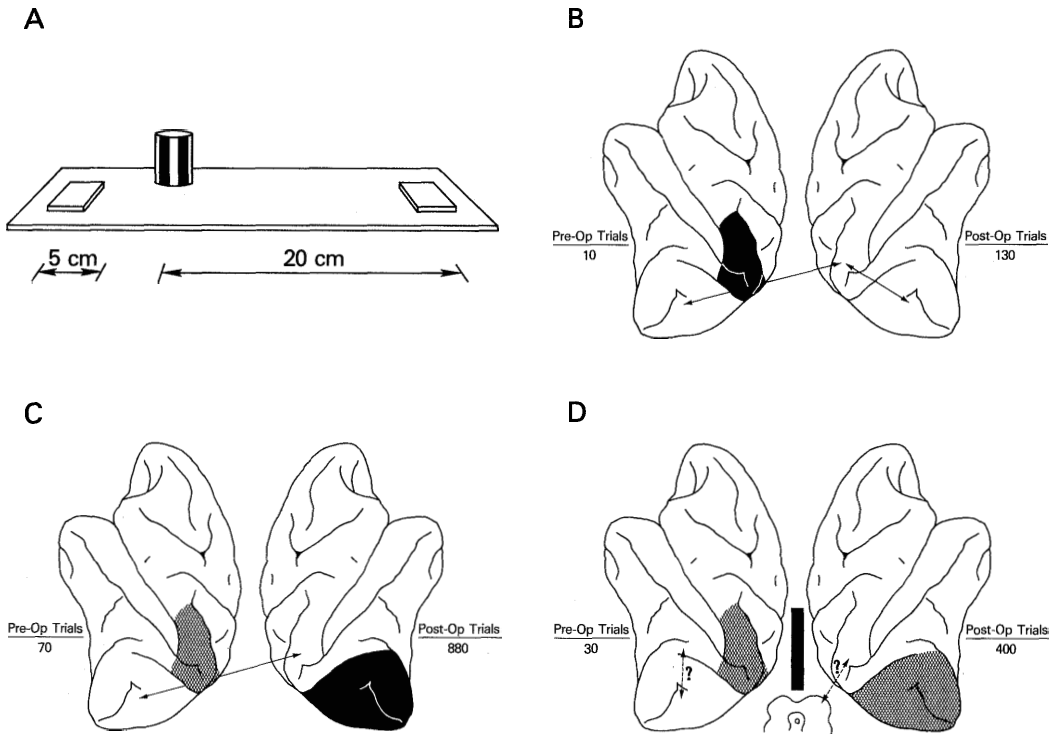
Figure 18.5 Design and results of the striate-parietal crossed-lesion disconnection study. (A) Landmark task. Monkeys were rewarded for choosing the covered foodwell located closer to a striped cylinder (the "landmark"), which was positioned on the left or the right randomly from trial to trial, but always 5 cm from one foodwell and 20 cm from the other. Training was given for 30 trials per day to a criterion of 90 correct responses in 100 consecutive trials. (B) Discrimination retention before and after first-stage lesion (unilateral posterior parietal; N = 3); 10 preoperative trials and 130 postoperative trials. (C) Discrimination retention before and after second-stage lesion (contralateral striate; N = 3); 70 preoperative and 880 postoperative trials. (D) Discrimination retention before and after third-stage lesion (corpus callosum; N = 3); 30 preoperative and 400 postoperative trials. At each stage the lesion is shown in black and the lesions of prior stages are shaded. Arrows denote hypothetical connections left intact by lesions. Adapted from Ungerleider and Miskin 1978a.

inputs to this cortex, which appear to be unnecessary when corticocortical connections are intact, also play no significant role in the recovery after corticocortical disconnection. By contrast, removal of prestriate cortex ipsilateral to the intact striate cortex produced a marked impairment on the task. Taken together, the results of this serial-lesion experiment demonstrate that the visual spatial functions of posterior parietal cortex depend on inputs from striate cortex and that such inputs are transmitted across corticocortical pathways. In this regard, it is significant that the only cortical area apart from those along the occipitotemporal pathway to show differential glucose utilization in the 2-DG experiments described earlier (Jarvis et al. 1978; Kennedy et al. 1978) was located within the posterior parietal region. The affected area extended forward from the dorsal prestriate cortex to include the posterior half of the inferior parietal gyrus, both on its crown and on the ventral bank of the intraparietal sulcus.

The Two Cortical Visual Systems Compared

These results on striate-parietal interaction parallel the earlier finding that corticocortical inputs from striate cortex are crucial for the visual recognition functions of inferior temporal cortex. But, though the results of these two studies are analogous, one important difference emerges (see figure 18.6). On the pattern-discrimination task of the earlier study, only a moderate effect followed the striate lesion, and a severe deficit did not result until the callosal transection. By contrast, on the landmark task, the relative effects of the striate and callosal lesions were reversed; that is, the striate-cortex lesion produced the more severe effect. These data suggest that the posterior parietal cortex, like the inferior temporal, depends heavily on corticocortical inputs from striate cortex for its visual function, but that, unlike the inferior temporal, the posterior parietal cortex in a given hemisphere does not seem to receive a heavy input via the corpus callosum from striate-cortex neurons representing the ipsilateral visual field. Thus, each posterior parietal area may be organized largely as a substrate for contralateral spatial function, and this could account in part for the symptom of contralateral neglect that has so often been reported after unilateral injury to the parietal lobe in man (see, for example, Denny-Brown and Chambers 1958).

A second important difference in the organization of visual inputs to the two systems was uncovered in an experiment comparing the behavioral effects of selective striate-cortex removals. In this experiment, monkeys received bilateral lesions of the striate area representing either central vision (lateral striate) or peripheral vision (medial striate). The lateral lesion included the entire lateral surface of striate cortex, and the medial lesion included the calcarine fissure as well as both banks of its ascending and descending limbs (figure 18.7). The monkeys were tested
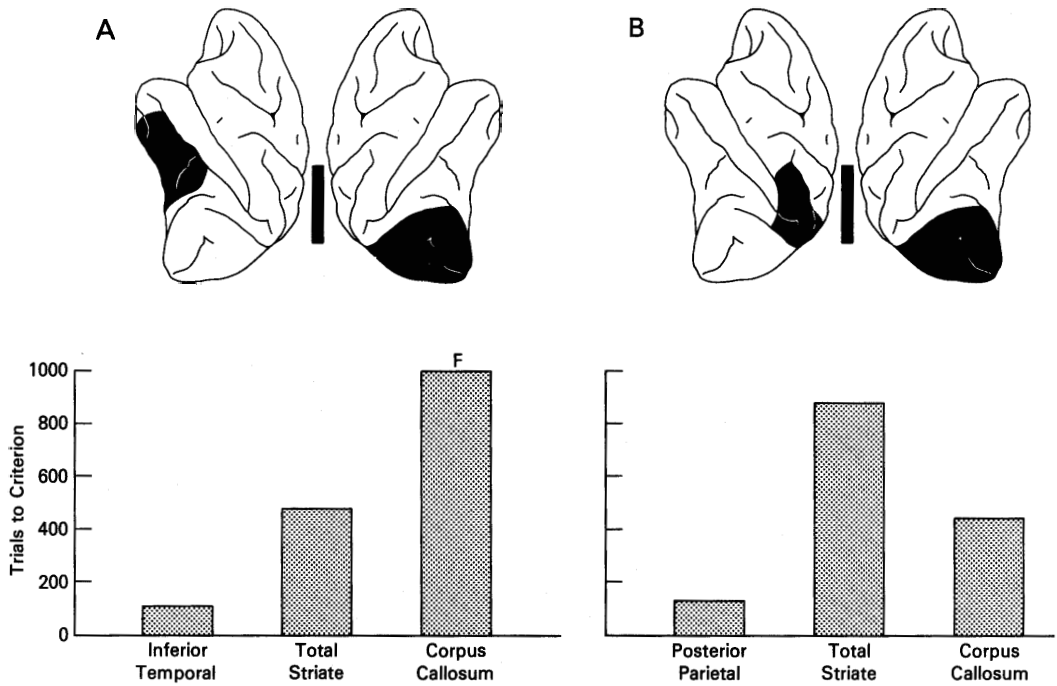
Figure 18.6 Comparison of two crossed-lesion disconnection experiments: (A) pattern discrimination after striate-temporal disconnection (Mishkin 1966) and (B) landmark discrimination after striate-parietal disconnection (Ungerleider and Mishkin 1978a). In each case, brain diagrams illustrate the three lesions (in black) involved in the disconnection and bar graphs show the mean trials to relearn after each lesion. F indicates failure to relearn within the limits of training.
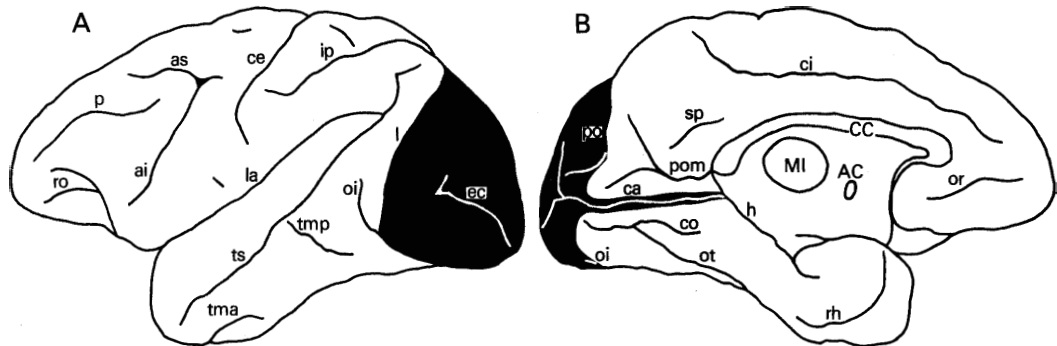

Figure 18.7 Locus of two different bilateral striate-cortex removals (in black): (A) lateral striate, the area representing central vision (N = 5); (B) medial striate, the area representing peripheral vision (N = 5). For details of visuotopic organization see figure 18.13; for abbreviations see figure 18.3.
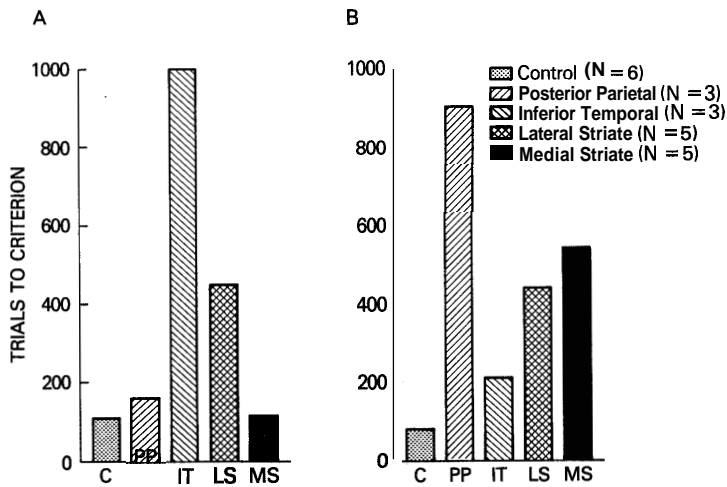
Figure 18.8 Comparison of the effects of lateral striate lesions with those of medial striate lesions on (A) postoperative acquisition of a pattern discrimination and (B) postoperative retention of the landmark task. Adapted from Ungerleider and Mishkin 1978a.

before operation both on a pattern-discrimination task, to assess residual inferior temporal function, and on the landmark task, to assess residual posterior parietal function. For purposes of comparison, their scores are plotted in figure 18.8 together with those of monkeys with either bilateral inferior temporal or bilateral posterior parietal lesions

The data indicate that on the pattern-discrimination task severe impairment was produced by inferior temporal but not by posterior parietal lesions, whereas on the landmark task severe impairment followed posterior parietal but not inferior temporal lesions. This, then, is another instance of the dissociation of visual recognition and visual spatial functions of temporal and parietal cortex, respectively. Of more direct concern for the question of differences in anatomical organization, however, were the effects of the striate lesions. Here the data indicate that on the pattern-discrimination task impairment was produced only by lateral striate-cortex lesions, whereas on the landmark task equally severe impairment followed lateral and medial striate-cortex lesions. Apparently, inputs from central vision are especially important for the visual recognition functions of the inferior temporal cortex, but inputs from central and peripheral vision are equally important for the visual spatial functions of posterior parietal cortex.

Thus, although interactions with striate cortex are critical for the parietal just as for the temporal area, the inputs of the striate cortex to these two regions appear to be organized differently; relative to inferior temporal cortex, posterior parietal cortex receives a smaller contribution from contralateral striate-cortex inputs (representing the ipsilateral visual field) but a greater contribution from medial inputs (representing
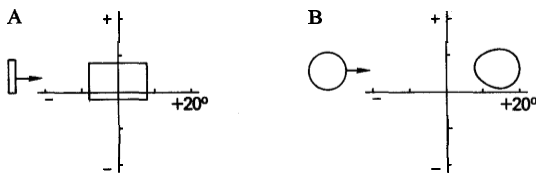
Figure 18.9 Receptive-field properties of neurons in (A) inferior temporal and (B) posterior parietal cortex. Axes represent horizontal and vertical meridians. Plus sign indicates upper or right visual field; minus sign indicates lower or left. Scale is in degrees of visual angle. A slow-moving stimulus typically effective in activating the given population of neurons is shown at the left of each diagram. The enclosed geometric shape within each set of axes indicates the border of the receptive field for a typical neuron in that population. Note that of the inferior temporal neurons over 60% are bilateral, 100% include the fovea, and there is selectivity for stimulus features; of the posterior parietal neurons over 60% are contralateral only, over 60% do not include the fovea, and there is no selectivity for stimulus features. (Part A adapted from Gross et al. 1972; part B adapted from Robinson et al. 1978.)

the peripheral visual field). Interestingly, these differences in the organization of striate-cortex inputs inferred from the ablation studies are clearly reflected in the receptive-field properties of neurons within the inferior temporal (Gross et al. 1972; Jarvis and Mishkin 1975; Rolls et al. 1977) and posterior parietal areas (Hyvärinen and Poranen 1974; Mountcastle et al. 1975; Robinson et al. 1978; Rolls et al. 1979a). The relevant properties are shown diagrammatically in figure 18.9.

First, there is a smaller representation of the ipsilateral visual field in the receptive fields of posterior parietal neurons than in those of inferior temporal neurons: Over 60% of inferior temporal neurons have bilateral receptive fields, while over 60% of posterior parietal neurons have contralateral fields only. Second, there is a relatively greater representation of the peripheral visual field in the receptive fields of posterior parietal neurons than in those of inferior temporal neurons: 100% of inferior temporal neurons have receptive fields that include the fovea, while over 60% of posterior parietal neurons have receptive fields outside the fovea. Finally, and perhaps most important, not only are the striate-cortex inputs to the temporal and parietal areas organized differently, but these inputs must also be carrying different information to the two areas, as is indicated by the behavioral evidence for functional dissociation. This too is clearly seen at the level of single neurons, for, in contrast to the specific and often complex trigger features needed for maximal activation of inferior temporal neurons (see, for example, Gross et al. 1972), most posterior parietal neurons can be maximally driven by simple spots of light (see Robinson et al. 1978). These several differences in the receptive-field properties of inferior temporal and posterior parietal neurons, each of which has a behavioral correlate that was revealed in the ablation studies, presumably reflect differences in the cortical processing required for object versus spatial vision.

Corticocortical Pathways Through Prestriate Cortex

The evidence presented thus far strongly supports the view that neural mechanisms underlying object and spatial perception depend on the relay of different kinds of visual information from striate cortex through prestriate cortex to targets in inferior temporal and posterior parietal areas, respectively. Despite the abundance of positive evidence, however, the support has not been unanimous. The results of prestriate-cortex ablation studies have repeatedly raised serious problems for this conception of corticocortical pathways. If prestriate cortex were an essential relay in either a striate-temporal or a striate-parietal pathway, then damage to this relay should yield effects at least as severe as damage to its target areas. Yet, monkeys sustaining extensive bilateral prestriate lesions commonly exhibit only mild visual effects. In a few instances in which severe visual deficits did follow prestriate removals (Keating and Horel 1972; Keating 1975), the removals included the posterior part of inferior temporal cortex (area TEO), damage to which, by itself, produces severe visual-discrimination impairment (Iwai and Mishkin 1968). Extensive prestriate-cortex removals that spare this posterior temporal region, however, have repeatedly failed to yield appreciable effects in either visual discrimination or in spatial orientation (Lashley 1948; Meyer et al. 1951; Riopelle et al. 1951; Chow 1952; Evarts 1952; Pribram et al. 1969; Ungerleider and Pribram 1977).

Is there a resolution of this paradox? It was suggested by Mishkin (1966) that failures to obtain severe impairments after extensive prestriate-cortex removals simply indicated that spared prestriate remnants continued to serve as effective relays between the striate area and its rostral targets—that is, that the prestriate cortex was invested with a high degree of equipotentiality. According to this proposal, only a complete prestriate lesion would be expected to yield a corticocortical disconnection. The results of a study by Iwai and Mishkin (Mishkin 1972) provided support for this proposal with regard to occipitotemporal transmission. When putatively complete prestriate-cortex lesions were made in a large group of monkeys, none was able to relearn a visual pattern discrimination. By contrast, partial prestriate ablations, irrespective of their location, were nearly without effect. These findings therefore pointed to the existence of multiple pathways through prestriate cortex, any of which can be utilized to convey visual information out of striate cortex. It seemed that only if all these equivalent pathways were destroyed would the transmission be completely disrupted and the anticipated deficit ensue.

Although it has since been found that even the massive removals of prestriate cortex referred to above did not totally disconnect the striate-temporal pathway, the conclusion reached remains valid nonetheless. Indeed, it has now been demonstrated that any sparing, no matter how minimal, of the visual functions of inferior temporal cor-

tex after a prestriate ablation is directly attributable to the continued relay of information across a viable prestriate pathway. This demonstration emerged from a series of interrelated behavioral, electrophysiological, and anatomical experiments (Ungerleider, Iwai, Gross, 'Bender, Snyder, and Mishkin, in prep.) that began in the following way: In an attempt to verify that the inferior temporal cortex had been functionally disconnected in the Iwai-Mishkin study, two of the monkeys that had failed to show relearning after the prestriate lesions were given prolonged pattern-discrimination training by a method of approximation, in preparation for a second-stage lesion. The method entailed presentation of a series of stimulus pairs that differed first in brightness, then in size, then in contour, and finally in pattern; the successive pattern pairs ,approximated the originals more and more closely until the original pattern discrimination had been relearned. Over several months both monkeys were successfully retrained by this method, and both were then given bilateral inferior temporal ablations in a second-stage operation. The supposition was that if the initial prestriate removal had produced a total disconnection of inferior temporal from striate cortex, then the slow pattern-discrimination relearning by approximation should have been achieved without the participation of inferior temporal cortex, and, consequently, removal of this cortex should not disrupt the relearned habit. As it turned out, however, the inferior temporal ablation produced a complete reinstatement of the deficit in both monkeys. Thus, unless inferior temporal cortex can participate in visual discrimination learning in the absence of all corticocortical visual input, the only conclusion to be drawn from this unexpected result is that the initial prestriate-cortex removal had not produced a total striate-temporal disconnection after all.

In order to examine directly this question of preserved neural transmission despite massive prestriate removals, an electrophysiological study was undertaken in animals prepared with removals identical to those in the Iwai-Mishkin study except that they were limited to one hemisphere. The distribution of the receptive fields of inferior temporal neurons in the operated animals is summarized in figure 18.10. According to the proposed route of visual information flow, indicated by the arrows in the brain diagrams, these neurons should have responded to visual stimulation only in the hemifield opposite the intact prestriate cortex. That is, inferior temporal neurons in the intact hemisphere should have had strictly contralateral visual fields, while inferior temporal neurons in the hemisphere with the prestriate ablation should have had strictly ipsilateral fields. In short, the results should have duplicated those obtained after unilateral striate removals, illustrated in parts C and D of figure 18.2. In fact, however, the receptive fields of approximately 25% of the neurons sampled did not fit this prediction.

In the normal monkey, stimulation in a given hemifield will activate about 80% of inferior temporal neurons. For example, as indicated in
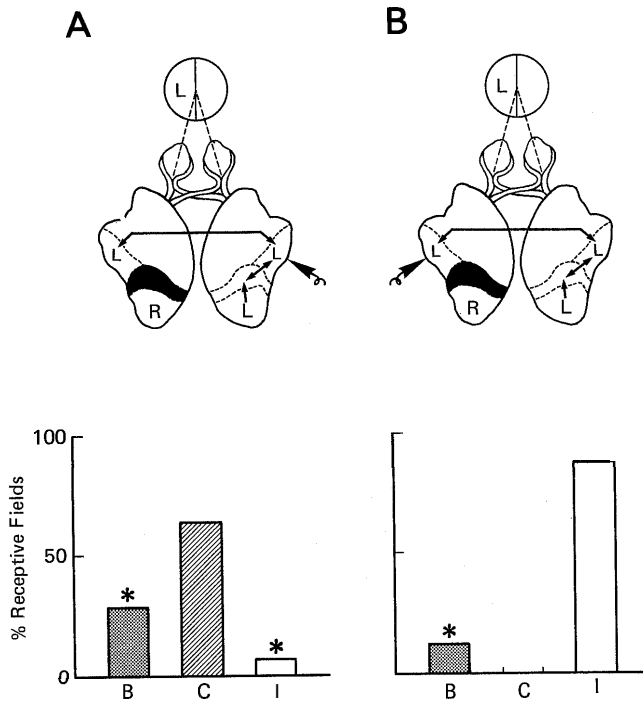
Figure 18.10 Proportion of inferior temporal neurons that had bilateral (B), contralateral (C), or ipsilateral (I) receptive fields after unilateral ablation of prestriate cortex (in black). Data for part A were obtained from inferior temporal neurons contralateral to the lesion, whereas data for part B were obtained from those ipsilateral to the lesion. Asterisks denote the receptive fields deviating from the prediction; that is, deviating from those obtained after unilateral striate-cortex removals, shown in figure 18.2. Adapted from Ungerleider, Snyder, and Mishkin, in prep.

part A of figure 18.2, stimulation in the right hemifield will activate about 90% of the inferior temporal neurons in the left hemisphere (all those with either bilateral or contralateral visual fields) and about 70% of the inferior temporal neurons in the right hemisphere (all those with either bilateral or ipsilateral visual fields). By contrast, in the animals with left prestriate removals, stimulation in the right hemifield activated about 25% of inferior temporal neurons. That is, as indicated in figure 18.10, right-hemifield stimulation activated about 15% of the inferior temporal neurons in the left hemisphere and about 35% in the right. Thus, the massive prestriate removals did greatly reduce the visual input to inferior temporal neurons — in fact, by more than two-thirds (80% to 25%) — thereby accounting for the severe deficit that such lesions produced in pattern discrimination relearning. On the other hand, the removals did not completely eliminate striate input to inferior temporal neurons (25%, as compared with 0% after striate removals), and this fact accounted for the ultimately successful retraining by approximation that was achieved in the animals with such lesions.

Even with this electrophysiological evidence, the conclusion that be-

havioral sparing resulted from sparing of remnants of the prestriate relay was only inferential. In an attempt to demonstrate the preserved pathway directly, a neuroanatomical experiment was undertaken on two additional monkeys with massive prestriate removals that had been successfully retrained by approximation on the pattern discrimination. More than a year after the prestriate-cortex removals, one of these animals was given a second-stage operation in which the entire occipital lobe posterior to the initial lesion was removed from one hemisphere, and the brain was examined for anterograde degeneration by the Fink-Heimer (1967) technique. The critical question was whether degeneration would appear in the inferior temporal cortex ipsilateral to the occipital lobectomy. Since striate cortex does not project directly to the inferior temporal area, the presence of degeneration here would indicate that the lobectomy had destroyed prestriate-relay tissue spared by the initial lesion—a finding that would account for the functional sparing that had been demonstrated both behaviorally and electrophysiologically. In fact, terminal degeneration did result in the ipsilateral inferior temporal cortex, specifically in the ventral part of area TEO. In addition, degenerating material was found in an unexpected area, the floor of the caudal portion of the superior temporal sulcus, which turned out to be a second spared route through which visual input could reach inferior temporal cortex. This was demonstrated in the other monkey of the anatomical experiment, which was given a second-stage lesion in just this portion of the superior temporal sulcus of one hemisphere, again more than a year after the initial prestriate removal. Terminal degeneration was found in the ipsilateral inferior temporal cortex of this case also, specifically in the dorsal part of area TEO.

The foregoing series of experiments made it clear that, although the massive prestriate-cortex removals had severely disrupted the prestriate relay, they had not totally abolished it. As illustrated in figure 18.11, spared pathways that presumably continued to serve as effective relays were directly demonstrated anatomically. At the same time, however, the anatomical results raised some important new questions. For example, what were the loci of the spared prestriate remnants whose removal gave rise to the degeneration in the ventral part of area TEO? Also, what was the source of the unexpected degeneration in the depth of the caudal portion of the superior temporal sulcus, an area from which a projection had been traced to dorsal TEO? These questions highlighted our lack of knowledge about the precise links through which visual information originating in the striate cortex is transmitted to inferior temporal cortex. Even greater ignorance surrounded the location and arrangement of the links in the striate-parietal pathway. It became apparent that, in order to obtain this information, a comprehensive anatomical investigation was needed, beginning with an analysis of the projections of the striate cortex itself.
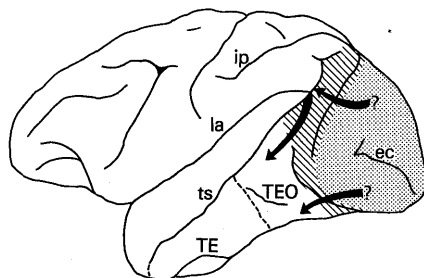
Figure 18.11 Schematic representation of results from two anatomical experiments, demonstrating that striate-prestriate-temporal pathways were preserved despite massive prestriate removals. In the first experiment, an occipital lobectomy (dots) was performed more than a year after the initial prestriate removal (stripes). Degeneration from the lobectomy was found in ventral area TEO and in the floor of the caudal portion of the superior temporal sulcus. The exact source of these two projections, however, was indeterminate (indicated by question mark). In the second experiment, the floor of the superior sulcus was removed, again more than a year after the initial prestriate lesion. Degeneration in this case was found in dorsal area TEO. For abbreviations see figure 18.3. Adapted from Ungerleider et al., in prep.

Clarification of Striate-Prestriate Connections

Early attempts to examine striate-prestriate connections (von Bonin et al. 1942; Bailey et al. 1944) employed the method of strychnine neuronography, described above. Although the map of striate projections obtained with this method has been confirmed by more recent work using degeneration (Kuypers et al. 1965; Cragg and Ainsworth 1969; Zeki 1969, 1971a; Jones and Powell 1970) and autoradiographic (Zeki 1976) and horseradish-peroxidase (Lund et al. 1975) tracing techniques, all these methods, both old and new, have been used to define projections primarily from lateral striate cortex, the part of striate cortex representing central vision. Surprisingly, there has been almost no information regarding the projections from posterior and medial striate cortex, the parts representing peripheral and far-peripheral vision. Indeed, it was undoubtedly the near absence of information about the location of the prestriate tissue serving noncentral vision that accounted for the repeated failure, recounted above, to completely disconnect the higher-order visual areas from their striate input. We therefore undertook a study of the cortical efferents from all parts of striate cortex, with the aim of defining the locus, extent, and topographic organization of the entire striate-prestriate projection system.

To delineate the entire map of striate projections to prestriate cortex, one series of monkeys was prepared with partial striate lesions such that, collectively, they included all of area 17 with little or no invasion of area 18. The brains were then processed by the Fink-Heimer (1967) procedure for silver staining of degenerating axon terminals. Reconstructions of the lesions in three of the five cases from this series are shown

in figure 18.12. The lateral lesion involved the lateral surface of area 17 only, with no invasion of area 18; the posterior lesion included the lateral and medial banks of the vertical limbs of the calcarine fissure, again with no invasion of area 18; and the medial striate lesion involved the tissue within the stem of the calcarine fissure, in this case with slight damage to area 18 around the lips of the fissure. According to the electrophysiological map of Daniel and Whitteridge (1961), these three sectors of striate cortex correspond to the central 7" of the contralateral visual field, to the field between 7° and 22" from fixation, and to eccentricities greater than 22" from fixation, respectively (see figure 18.13). Thus, it was anticipated that not only would the entire striate-prestriate projection system emerge from the data on this series of monkeys, but the general visuotopic organization of the system would be apparent as well.

To verify the degeneration results, and also to investigate the details of the visuotopic organization, a second series of monkeys were prepared with injections of radioactively labeled amino acids into selected sites in striate cortex, and the brains processed for autoradiography. A summary of the injection sites is shown in figure 18.14. These loci correspond to positions in the visual field ranging from less than ½° from fixation (site number 1 on the lateral surface) to greater than 45" from fixation (site number 10 in the stem of the calcarine fissure). The loci injected included representations of both the upper and the lower visual fields.

The results from both sets of experiments, degeneration and autoradiographic, indicated that all parts of striate cortex project to three separate visual areas within prestriate cortex (Ungerleider and Mishkin 1979a): The first is a circumstriate cortical belt surrounding area 17 at the 17–18 border, the second is located along the caudal portion of the superior temporal sulcus, and the third is buried deep within the caudal part of the intraparietal sulcus. Each of these three projection areas will be described in turn.

Within the first projection area—the circumstriate cortical belt—the representations of the upper and lower visual fields are entirely separate (figure 18.15). The topographic organization of these separate representations is illustrated in figure 18.16. Progression from central to peripheral to far peripheral vision is represented in the lower field by a progression into the posterior bank and depth of the lunate sulcus, medially along the surface of the buried annectent gyrus into the parieto-occipital incisure, and then rostrally along the upper lip of the calcarine fissure; and in the upper field by a progression into the inferior occipital sulcus, ventromedially into the occipitotemporal and collateral sulci, and then rostrally along the lower lip of the calcarine fissure. The total extent of this projection field corresponds remarkably closely to area OB of von Bonin and Bailey (1947); indeed, the two may be equivalent. Moreover, the autoradiographic evidence indicates that whereas the
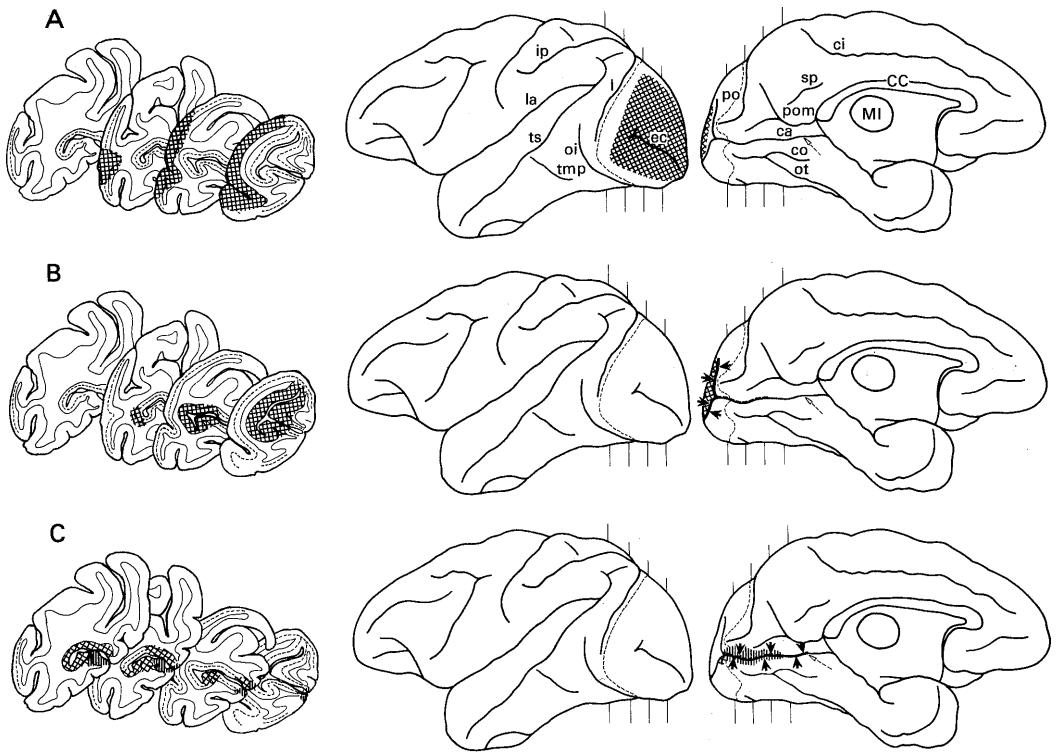
Figure 18.12 Location of cortical damage in brains with removals of (A) lateral, (B) posterior, or (C) medial striate cortex. The lesions are mapped on lateral and medial surface views and on representative cross-sections through a standard rhesus monkey brain. Cross-sections are at levels indicated by vertical lines on surface views. Dashed lines indicate extent of striate cortex on cross-sections but borders of striate cortex on surface views; anterior limit of striate cortex within the calcarine fissure is indicated on medial surface views by dashed arrow. Removal of striate cortex is shown in crosshatch; black arrows on the medial surface views indicate removal of striate tissue from within the banks and depths of the calcarine fissure. Damage to prestriate tissue is shown in stripes. For abbreviations see figure 18.3.
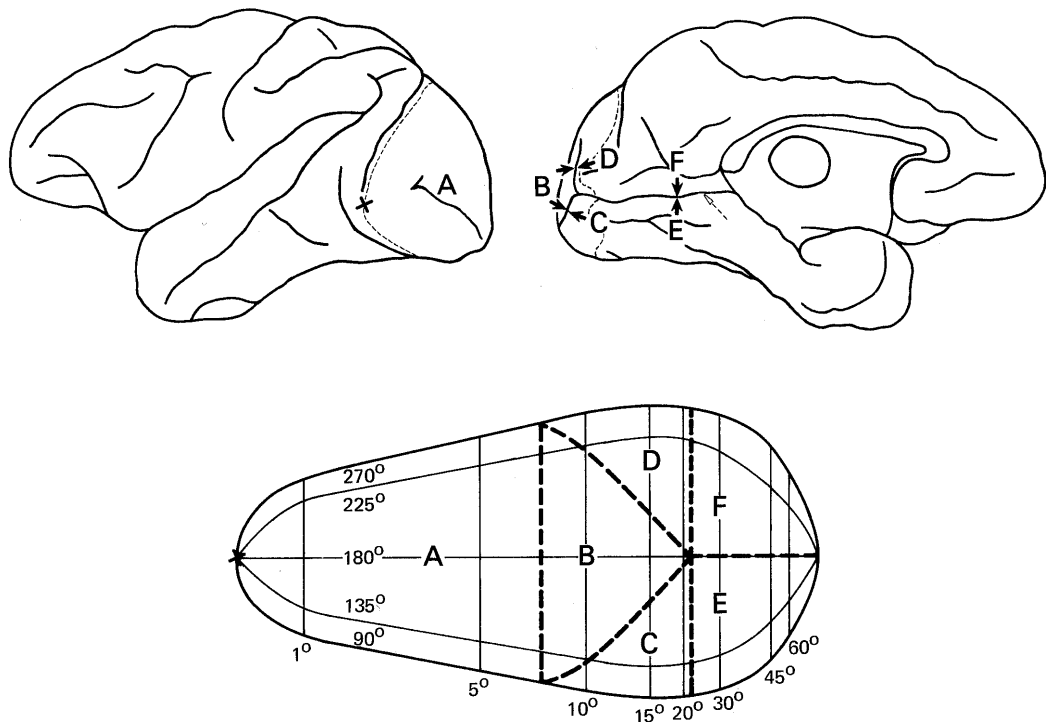
Figure **18.13** Relationship between topography of striate cortex and representation of the contralateral visual field. According to the electrophysiological map of Daniel and Whitteridge **(1961),** shown at bottom, the center of gaze (fixation) is represented in the far anterolateral part of striate cortex, just below the ventral tip of the lunate sulcus. The representation of the vertical meridian passes through fixation (X) and continues all along the **17–18** border (dashed line). The representation of the horizontal meridian is a horizontal line (not illustrated) passing from fixation across the lateral surface, entering the depth of the calcarine fissure, and continuing to the rostral limit of the medial striate cortex (dashed arrow). The upper visual field is represented below the horizontal meridian and the lower visual field above the horizontal meridian. The lateral surface of striate cortex (A), the lateral bank of the calcarine limbs (B), the medial bank of the calcarine limbs (C, D), and the stem of the calcarine fissure (E, F) represent approximately the center **7°, 7°–13°, 13°–22°,** and eccentricities greater than **22"** from fixation, respectively. Thus, a progression from lateral to posterior to medial striate cortex corresponds to a progression from central to peripheral to far-peripheral vision.

vertical meridian is represented at the inner boundary of this projection field (that is, at the striate-OB border), the horizontal meridian is represented at its outer boundary (at the OB-OA border).

The second projection field of striate cortex is located within cytoarchitectonic area OA along the caudal portion of the superior temporal sulcus (Ungerleider and Mishkin 1978b, 1979b). As indicated in the lateral view of the brain shown in figure 18.17, the ventral limit of this region can be demarcated by an imaginary line connecting the ventral tips of the lunate and intraparietal sulci; from this limit the region extends dorsocaudally for about 1 cm to the point at which the superior temporal sulcus frequently bifurcates, sending one spur forward into the inferior parietal lobule. Within this portion of the superior temporal sulcus there is again an orderly mapping of the contralateral visual field,
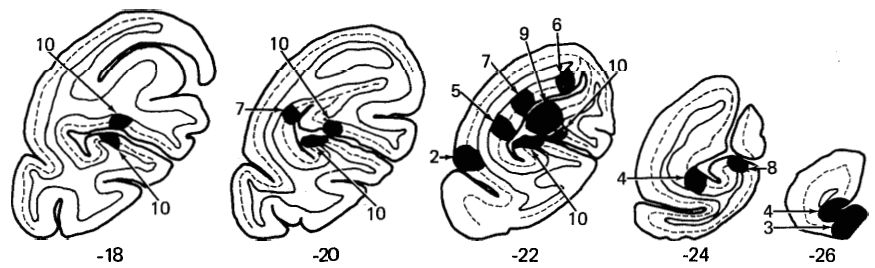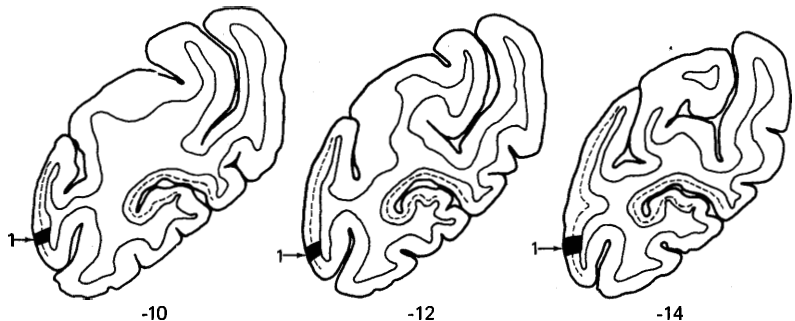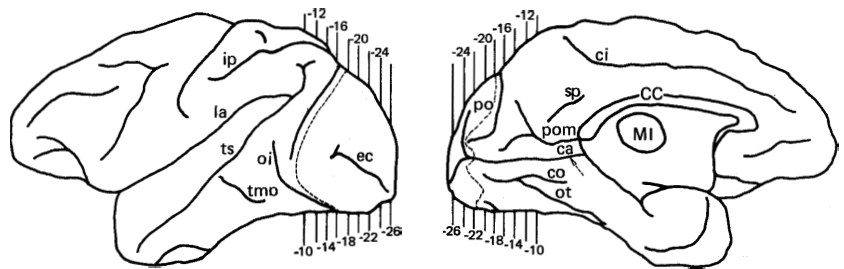
Figure 18.14 Sites in striate cortex injected with tritiated amino acids. Injection sites (shown in black) are numbered according to the eccentricity of their representation in the visual field. These representations ranged from less than one-half degree from fixation (site 1) to greater than 45° from fixation (site 10). Each injection was made in a separate hemisphere. Dashed lines indicate extent of striate cortex on cross-sections,'but borders of striate cortex on surface views; anterior limit of striate cortex within the calcarine fissure is indicated on medial surface view by a dashed arrow. Negative numerals indicate approximate A-P stereotaxic levels. For abbreviations see figure 18.3.
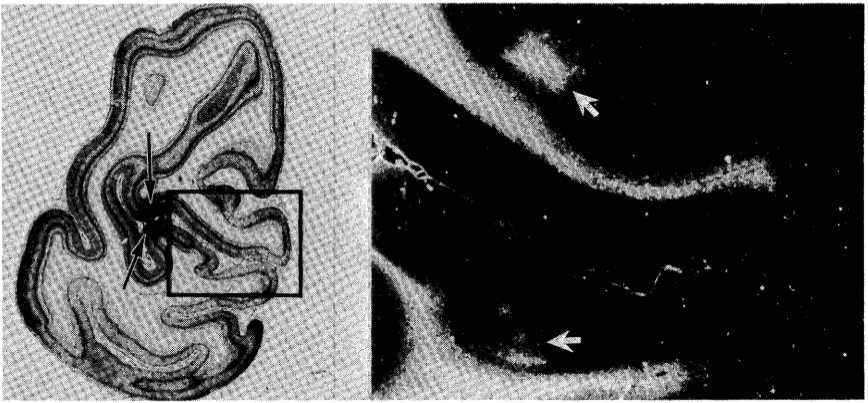
Figure 18.15 Photomicrographs exemplifying autoradiographic results after injections of striate cortex. The bright-field photomicrograph on the left shows an injection (marked by arrows) involving both the dorsal and ventral banks of the calcarine fissure (see figure 18.14, injection site 10). In the dark-field photomicrograph on the right, labeling is seen in two prestriate loci, marked by arrows: dorsally in the medial parieto-occipital fissure and ventrally in the ventral bank of the calcarine fissure. The patchy pattern of label, especially apparent in the ventral locus, is typical of striate projections to the circumstriate cortical belt. Adapted from Ungerleider and Mishkin 1979a.

as shown on the three selected cross sections. Progression from central to peripheral to far-peripheral vision is represented by a progression down the posterior bank of the superior temporal sulcus and up along the lower and then the upper part of the sulcal floor. Figure 18.18 summarizes this topographic arrangement. The finding that the floor of the superior temporal sulcus receives a direct projection from posterior and medial striate cortex provides an answer to one of the anatomical questions posed in the preceding section (see figure 18.11). As yet, however, we do not have an answer to the second question raised there, except the confirmation that the immediate source of the occipital projections to ventral area TEO is not the striate cortex.

The third area that receives direct striate-cortex projections is also located within cytoarchitectonic area OA, in the depth of the most caudal portion of the intraparietal sulcus. A cross-section through the projection shows that it is buried in the foot-shaped part of the sulcus beneath its lateral bank (figure 18.19). This projection field is by far the smallest of the three; its total rostral-caudal length extends only about 2 mm. There is no discernible topographic representation of the visual field within this area, for after a lesion or an injection involving any part of striate cortex a projection is always seen in the same part of the sulcus. Thus, although the dark-field photomicrograph in figure 18.19 shows degeneration in the foot of the intraparietal sulcus after a medial striate lesion, the very same picture could as well represent the projections of either posterior or lateral striate cortex. Indeed, the first evidence of direct striate projections to this area came from studies that
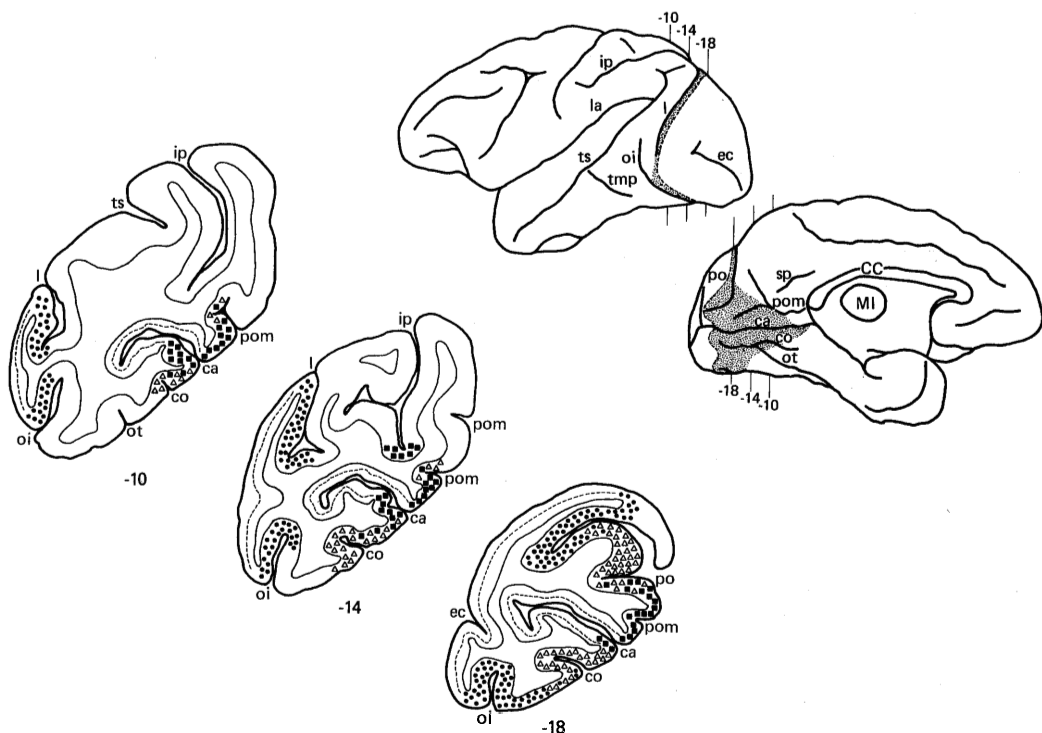
Figure 18.16 Location, extent, and topographic organization of striate projections (●: lateral; △: posterior; ■: medial) to the circumstriate cortical belt. The drawings are composites based on both degeneration and autoradiographic material. Shaded area represent total projection field on surface views. As shown on the surface views of the hemisphere, this projection zone surrounds the striate cortex at the 17–18 border and corresponds remarkably closely to cytoarchitectonic area OB of von Bonin and Bailey (1947). Within the projection zone, there is a precise topographic map of the contralateral visual field (see cross-sections); note, however, that the representations of the upper and lower fields are entirely separate. Dashed lines indicate extent of striate cortex on cross sections, but borders of striate cortex on surface views. Numerals indicate approximate A-P stereotaxic levels. For abbreviations see figure 18.3. Adapted from Ungerleider and Mishkin 1979a.

had examined only lateral striate connections (Kuypers et al. 1965; Jones and Powell 1970), though the area had not been recognized as a separate projection field at that time.

These three projection fields thus comprise all the possible first-stage prestriate relays through which striate output can reach the inferior temporal and posterior parietal cortex. Undoubtedly, second- and third-stage prestriate relays also participate in both corticocortical pathways (Zeki 1971b; Mesulam et al. 1977; Stanton et al. 1977; Desimone et al. 1980), although the exact location and topographic arrangement of these further relays remain to be delineated. But even from the evidence pertaining to the first stage alone, we can begin to understand why attempts to produce striate-temporal disconnection by removal of prestriate cortex have repeatedly failed. In all prestriate-
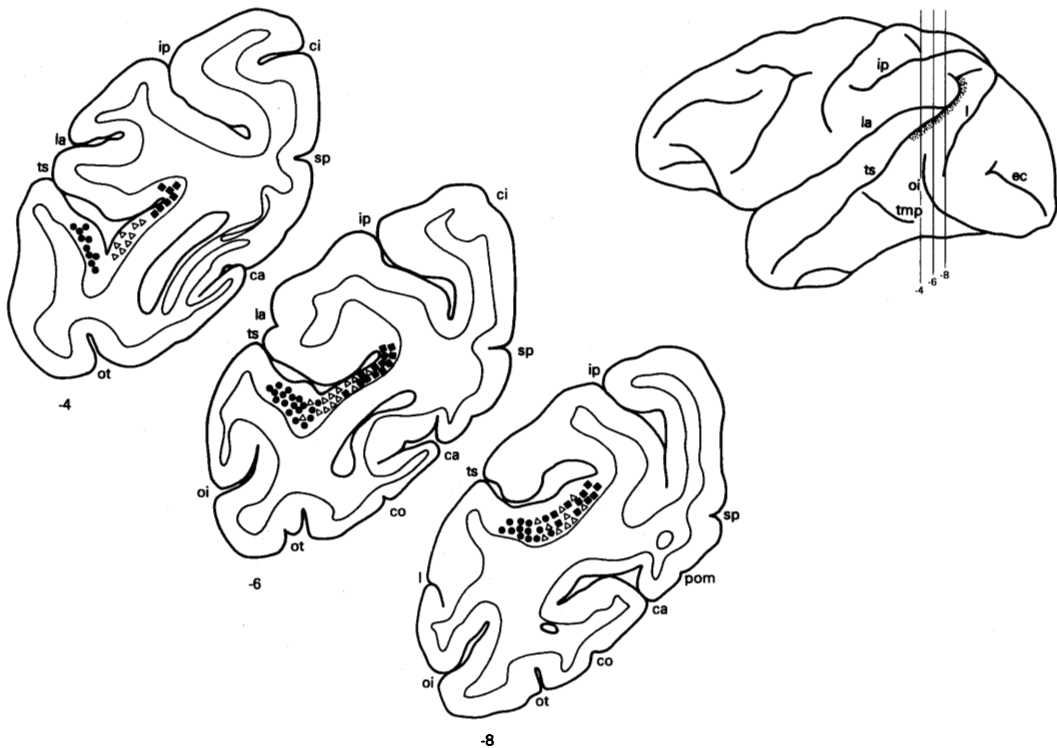
Figure 18.17 Location, extent, and topographic organization of striate projections (●: lateral; △: posterior; ■: medial) to cortex in the superior temporal sulcus. The drawings are composites based on both degeneration and autoradiographic material. Shaded area represents locus of projection on lateral view. All areas of striate cortex project to a restricted region inside the caudal portion of the superior temporal sulcus, its A-P extent indicated on the lateral view of the hemisphere. As indicated on the cross-sections, the topographic arrangement of the contralateral visual field is such that a progression from central to peripheral to far peripheral vision is represented by a progression down the posterior bank of the sulcus and up along the lower and then the lower part of the sulcal floor. Although this topography is clearest in the rostral portion of the projection zone, it is also discernible caudally. Numerals refer to approximate A-P stereotaxic levels. For abbreviations see figure 18.3. Adapted from Ungerleider and Mishkin 1979b.

ablation studies, certain areas have consistently been spared that can now be identified as receiving direct projections from posterior and medial striate cortex. These prestriate areas are the medial portion of area OB (figure 18.16), the floor of the caudal portion of the superior temporal sulcus (figure 18.17), and the depth of the most caudal portion of the intraparietal sulcus,(figure 18.19). Each of these three areas has escaped damage for a different reason: the first because of its relative inaccessibility, the second because it was not even recognized to be a part of prestriate cortex, and the third because its precise location had not been described. In light of the nearly complete sparing of these areas in most ablation studies, the finding of only mild visual impairment is no longer surprising. Only when these areas were invaded substantially, as in the massive prestriate removals of the Iwai-Mishkin study, did severe visual impairment result. However, even in those le-

Figure 18.18 Drawings of the lateral and medial views of the left hemisphere and of a cross-section through the superior temporal sulcus to illustrate the topographic arrangement of striate projections to this secondary visual area. The orderly mapping within the sulcus of the entire representation of the contralateral visual field is shown. For abbreviations see figure 18.3. Source: Ungerleider and Mishkin 1979b.

sions, where there was almost total destruction of the prestriate areas that receive direct projections from lateral striate cortex (the part representing central vision), there was no more than 50% destruction (Ungerleider et al., in prep.) of the prestriate areas that receive direct projections from posterior and medial striate cortex (the parts representing peripheral and far peripheral vision). Clearly, it was the sparing of this tissue that accounted for the continued (though reduced) transmission of visual information to inferior temporal cortex that had been demonstrated not only behaviorally but also electrophysiologically. Thus, the present anatomical findings, many of which have now been confirmed by others (Weller and Kaas 1978; Maunsell et al. 1979; Rockland 1979; Van Essen et al. 1979; Weller et al. 1979), provide a coherent explanation for a large body of seemingly paradoxical results regarding the effects of prestriate lesions. That is, just as "peripheral" striate cortex can mediate pattern discrimination in the absence of "central" striate cortex (Blake et
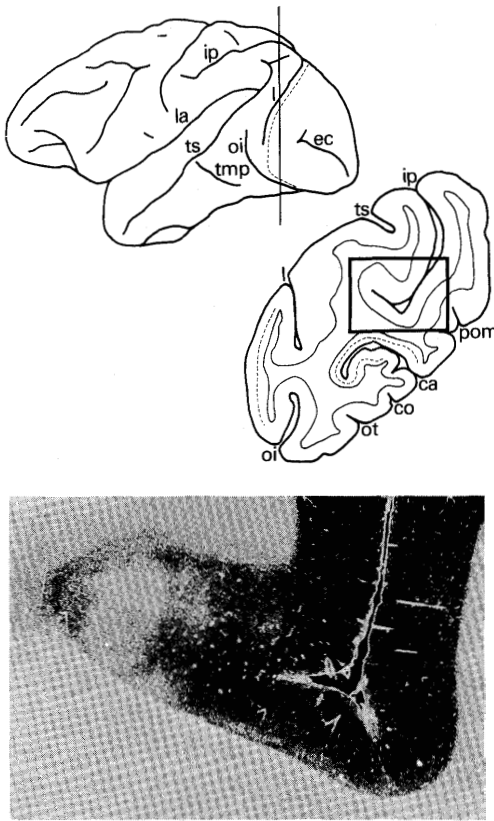
Figure 18.19 The striate projection zone within the intraparietal sulcus. This zone is located in the depth of the most caudal portion of the sulcus. Its approximate A-P level is indicated by the vertical line on the lateral view of the hemisphere, and its depth by the black square on the cross-section. The dark-field photomicrograph shows the locus of degeneration after a medial striate lesion. Projections are seen in this very same region from all parts of striate cortex, which suggests the absence of a visuotopic organization within this projection zone. Source: Ungerleider and Mishkin 1979a.

al. 1977), so can "peripheral" prestriate cortex substitute for "central" prestriate cortex in this function. And if this is the case for the visual recognition functions of inferior temporal cortex, where inputs from peripheral vision are less important than those from central vision, then the same must surely be true for the visual spatial functions of posterior parietal cortex, where inputs from peripheral vision are equal in importance to those from central vision (see figure 18.8).

Now that the total system of striate efferents has been delineated, the effects of completely disconnecting temporal or parietal from striate cortex can finally be investigated. Such disconnection should be achievable by removal of just the three striate projection fields that have been described, without inclusion of any other prestriate tissue. In addition, with special testing methods it may now be possible to study the behavioral effects of prestriate-cortex damage without producing total

disconnection. In view of the visuotopic organization of the striate projection zones in both area OB and the caudal part of the superior temporal sulcus, the details of which have been corroborated electrophysiologically (Gattass and Gross 1979; Gattass et al. 1979), even limited lesions within these prestriate zones should yield severe visual deficits if the animals are forced to use the part of the visual field corresponding to the area damaged. Testing of visual functions within specified parts of the visual field can be accomplished in monkeys who have been trained to maintain fixation (Wurtz 1969). Thus, the combination of this method and the use of lesions based on our new understanding of prestriate anatomy offers a promising approach for the study of the processing characteristics and functional organization of prestriate cortex.

Summary and Conclusions

The hypothesis that has been guiding our research is that appreciation of an object's qualities and of its spatial location depends on the processing of different kinds of visual information in the inferior temporal and posterior parietal cortex, respectively. From an initial concern with these higher-order visual areas, our research has proceeded backward. Having first obtained strong support from ablation studies for the postulated dichotomy of temporal and parietal function, we next examined the pathways through which visual information reaches these two cortical areas. Converging evidence from a variety of sources — behavioral and electrophysiological disconnection experiments as well as anatomical and metabolic mapping studies — indicated that both the temporal and parietal visual areas depend on corticocortical inputs relayed from striate through prestriate cortex. The results of prestriate ablation studies, however, conflicted with this conclusion, since they commonly indicated only modest deficits in both visual recognition and spatial orientation. The solution to this puzzle emerged from a series of interrelated behavioral, electrophysiological, and anatomical experiments that demonstrated that every case of functional sparing after prestriate damage can be directly attributed to the continued relay of information through viable prestriate remnants. To determine the locus of these prestriate remnants, we turned to a study of the cortical efferents from all parts of striate cortex. The results revealed that all prestriate lesions to date, no matter how massive, have failed to include varying extents of tissue that receive direct projections from posterior and medial striate cortex, the parts representing peripheral vision. Having delineated the projections of striate cortex, which were found to consist of three separate re-representations of the visual field, we are now in a position to proceed in a forward direction and follow these projections, with the inferior temporal and posterior parietal cortex as our targets. In the course of this endeavor, a major goal will be to determine where within

the complex of prestriate cortex the two cortical visual systems begin to diverge. On the assumption that both systems can indeed be followed stepwise to our target areas, not only in the temporal but also in the parietal lobe, a major question for the future will be how the object and spatial information carried in these two separated systems are subsequently integrated into a unified visual percept.

Acknowledgments

References

Bailey, P., G. von Bonin, E. W. Davis, H. W. Garol, and W. S. McCulloch. 1944. "Further observations on association pathways in the brain of *Macaca mulatta.*" *J. Neuropathol. Exp. Neurol.* 3: 413–415.

Bates, J. A. V., and G. Ettlinger. 1960. "Posterior biparietal ablations in the monkey." *Arch. Neurol.* 3: 177–192.

Benson, D. F., J. Segarra, and M. L. Albert. 1974. "Visual agnosia-prosopagnosia." *Arch. Neurol.* 30: 307–310.

Blake, L., C. D. Jarvis, and M. Mishkin. 1977. "Pattern discrimination thresholds after partial inferior temporal or lateral striate lesions in monkeys." *Brain Res.* 120: 209–220.

Blum, J. S. 1951. "Cortical organization in somesthesis: Effects of lesions in posterior associative cortex in somatosensory function in *Macaca mulatta.*" *Comp. Psychol. Monogr.* 20: 219–249.

Brody, B. A., and K. H. Pribram. 1978. "The role of frontal and parietal cortex in cognitive processing: Tests of spatial and sequence functions." *Brain* 101: 607–633.

Butters, N., C. Soeldner, and P. Fedio. 1972. "Comparison of parietal and frontal lobe spatial deficits in man: Extrapersonal vs. personal (egocentric) space." *Percept. Motor Skills* 34: 27–34.

Chow, K. L. 1952. "Further studies on selective ablation of associative cortex in relation to visually mediated behavior." *J. Comp. Physiol. Psychol.* 45: 109–118.

Cowey, A., and C. G. Gross. **1970.** "Effects of foveal prestriate and inferotemporal lesions on visual discrimination by rhesus monkeys." *Exp. Brain Xes.* **11: 128–144.**

Cragg, B. G., and A. Ainsworth. **1969.** "The topography of the afferent projections in the circumstriate visual cortex of the monkey studied by the Nauta method." *Vision Res.* **9: 733–747.**

Daniel, P. M., and D. Whitteridge. **1961.** "The representation of the visual field on the cerebral cortex in monkeys." J. *Physiol.* **159: 203–221.**

Dean, **P. 1976.** "Effects of inferotemporal lesions on the behavior of monkeys." *Psychol. Bull.* **83: 41–71.**

Denny-Brown, D.; and R. A. Chambers. **1958.** "The parietal lobe and behavior." *Res. Publ. Assoc. Res. Nerv. Ment. Dis.* **36: 35–117.**

De Renzi, E., and P. Faglioni. **1967.** "The relationship between visuo-spatial impairment and constructional apraxia." *Cortex* **3: 327–342.**

Desimone, R., J. Fleming, and C. G. Gross. **1980.** "Prestriate afferents to inferior temporal cortex: An HRP study." *Brain Res.* **184: 41–55.**

Ettlinger, G., and **J.** E. Kalsbeck. **1962.** "Changes in tactile discrimination and in visual reaching after successive and simultaneous bilateral posterior parietal ablations in the monkey." *J. Neurol. Neurosurg. Psychiatr.* **25: 256–268.**

Ettlinger, G., and J. Wegener. **1958.** "Somaesthetic alternation, discrimination, and orientation after frontal and parietal lesions in monkeys." *Q. J. Exp. Psychol.* 10: **177–186.**

Ettlinger, G., H. B. Morton, and A. Moffett. **1966.** "Tactile discrimination performance in the monkey: The effect of bilateral posterior parietal and lateral frontal ablations, and of callosal section." *Cortex* **2: 5–29.**

Evarts, E. V. **1952.** "Effects of ablation of prestriate cortex in auditory-visual association in monkey." *J. Neurophysiol.* **15: 191–200.**

Faugier-Grimaud, S., C. Frenois, and D. G. Stein. **1978.** "Effects of posterior parietal lesions on visually guided behavior in monkeys." *Neuropsychologia* **16: 151–168.**

Fink, R. P., and L. Heimer. **1967.** "Two methods for selective silver impregnation of degenerating axons and their synaptic endings in the central nervous system." *Brain Res.* **4: 369–374.**

Flechsig, **P. 1896.** *Gehirn und Seele.* Leipzig: von Veit.

———. **1920.** *Anatomie des menschlichen Gehirns und Rückenmarks auf myelogenetischer Grundlage.* Leipzig: Thieme.

Gattass, R., and C. G. Gross. **1979.** "A visuotopically organized area in the posterior superior temporal sulcus of the macaque." *Invest. Ophthalmol.* (ARVO suppl.) 18: 184.

Gattass, R., J. H. Sandell, and C. G. Gross. **1979.** "V2 in the macaque: Visuotopic organization and extent." *Soc. Neurosci. Abstr.* **5: 786.**

Gross, C. G. 1973. "Visual functions of inferotemporal cortex." In *Handbook of Sensory Physiology,* VII/3, R. Jung, ed. Berlin: Springer.

Gross, C. G., and M. Mishkin. 1977. "The neural basis of stimulus equivalence across retinal translation." In *Lateralization in the Nervous System,* S. Harnad et al., eds. New York: Academic.

Gross, C. G., C. E. Rocha-Miranda, and D. B. Bender. 1972. "Visual properties of neurons in inferotemporal cortex of the macaque." *J. Neurophysiol.* **35**: 96–111.

Hartje, W., and G. Ettlinger. 1973. "Reaching in light and dark after unilateral posterior parietal ablations in the monkey." *Cortex* **9**: 344–352.

Heilman, K. M., D. N. Pandya, and N. Geschwind. 1970. "Trimodal inattention following parietal lobe ablations." *Trans. Amer. Neurol. Assoc.* **95**: 259–261.

Held, R. 1968. "Dissociation of visual functions by deprivation and rearrangement." *Psychol. Forsch.* **31**: 338–348.

Hubel, D. H., and T. N. Wiesel. 1968. "Receptive fields and functional architecture of monkey striate cortex." *J. Physiol.* **195**: 215–243.

———. 1970. "Stereoscopic vision in macaque monkey." *Nature* **225**: 41–42.

Hyvärinen, J., and A. Poranen. 1974. "Function of the parietal associative area 7 as revealed from cellular discharges in alert monkeys." *Brain* **97**: 673–692.

Ingle, D. 1967. "Two visual mechanisms underlying the behavior of fish." *Psychol. Forsch.* **31**: 44–51.

Iwai, E., and M. Mishkin. 1968. "Two visual foci in the temporal lobe of monkeys." In *Neurophysiological Basis of Learning and Behavior,* N. Yoshii and N. A. Buchwald, eds. Osaka University Press.

Jarvis, C. D., and M. Mishkin. 1975. "Responses of inferior temporal neurons to visual discriminanda." *Soc. Neurosci. Abstr.* **1**: 61.

———. 1977. "Responses of cells in the inferior temporal cortex of monkeys during visual discrimination reversal." *Soc. Neurosci. Abstr.* **3**: 564.

Jarvis, C. D., M. Mishkin, M. Shinohara, O. Sakurada, M. Miyaoka, and C. Kennedy. 1978. "Mapping the primate visual system with the [^{14}C] 2-deoxyglucose technique." Soc. *Neurosci. Abstr.* **4**: 632.

Jones, B., and M. Mishkin. 1972. "Limbic lesions and the problem of stimulus-reinforcement associations." *Exp. Neurol.* **36**: 362–377.

Jones, E. G., and T. P. S. Powell. 1970. "An anatomical study of converging sensory pathways within the cerebral cortex of the monkey." *Brain* **93**: 793–820.

Keating, E. G. 1975. "Effects of prestriate and striate lesions on the monkey's ability to locate and discriminate visual forms." *Exp. Neurol.* **47**: 16–25.

Keating, E. G., and J. A. Horel. 1972. "Effects of prestriate and striate lesions on performance of simple visual tasks." *Exp. Neurol.* **35**: 322–336.

Kennedy, C., M. H. Des Rosiers, J. W. Jehle, M. Reivich, F. Sharp, and L. Sokoloff. 1975. "Mapping of functional neural pathways by autoradiographic survey of local metabolic rate with [^{14}C]deoxyglucose." *Science* **187**: 850–853.

Kennedy, C., C. D. Jarvis, O. Sakurada, and M. Mishkin. 1978. "A delineation of the visually responsive loci of the temporal lobe by means of [^{14}C]deoxyglucose." *Neurology* **28**: 366.

Kimura, D. 1963. "Right temporal-lobe damage." *Arch. Neurol.* **8**: 264–271.

Kuypers, H. G. J. M., M. K. Szwarcbart, M. Mishkin, and H. E. Rosvold. 1965. "Occipitotemporal corticocortical connections in the rhesus monkey." *Exp. Neurol.* **11**: 245–262.

LaMotte, R. H., and C. Acuña. 1978. "Defects in accuracy in reaching after removal of posterior parietal cortex in monkeys." *Brain Res.* **139**: 309–326.

Lansdell, H. 1968. "Effect of extent of temporal lobe ablations on two lateralized deficits." *Physiol. Behav.* **3**: 271–273.

Lashley, K. S. 1948. "The mechanism of vision. XVIII. Effects of destroying the visual 'associative areas' of the monkey." *Genet. Psychol. Monogr.* **37**: 107–166.

Lund, J. S., R. D. Lund, A. E. Hendrickson, A. H. Bunt, and A. F. Fuchs. 1975. "The origin of efferent pathways from the primary visual cortex, area 17, of the macaque monkey as shown by retrograde transport of horseradish peroxidase." *J. Comp. Neurol.* **164**: 287–304.

Maunsell, J. H. R., J. L. Bixby, and D. C. Van Essen. 1979. "The middle temporal area (MT) in the macaque: Architecture, functional properties, and topographic organization." *Soc. Neurosci. Abstr.* **5**: 796.

McFie, J., M. F. Piercy, and O. L. Zangwill. 1950. "Visual-spatial agnosia associated with lesions of the right cerebral hemisphere." *Brain* **73**: 167–190.

Meadows, J. C. 1974. "The anatomical basis of prosopagnosia." *J. Neurol. Neurosurg. Psychiatr.* **37**: 489–501.

Mesulam, M.-M., G. W. Van Hoesen, D. N. Pandya, and N. Geschwind. 1977. "Limbic and sensory connections of the inferior parietal lobule (area PG) in the rhesus monkey: A study with a new method for horseradish peroxidase histochemistry." *Brain Res.* **136**: 393–414.

Meyer, D. R., H. F. Harlow, and H. W. Ades. 1951. "Retention of delayed responses and proficiency in oddity problems by monkeys with preoccipital ablations." *Amer. J. Psychol.* **44**: 391–396.

Milner, A. D., E. M. Ockleford, and W. Dewar. 1977. "Visuo-spatial performance following posterior parietal and lateral frontal lesions in stumptail macaques." *Cortex* **13**: 350–360.

Milner, B. 1958. "Psychological defects produced by temporal-lobe excision." *Res. Publ. Assoc. Res. Nerv. Ment. Dis.* **36: 244–257**.

———. 1968. "Visual recognition and recall after right temporal-lobe excision in man." *Neuropsychologia* **6: 191–209**.

Mishkin, M. 1966. "Visual mechanisms beyond the striate cortex." In *Frontiers in Physiological Psychology,* R. Russell, ed. New York: Academic.

———. 1972. "Cortical visual areas and their interactions." In *Brain and Human Behavior,* A. G. Karczmar and J. C. Eccles, eds. Berlin: Springer.

Moffett, A. M., and G. Ettlinger. 1970. "Tactile discrimination performance in the monkey: The effect of unilateral posterior parietal ablations." *Cortex* **6: 47–67**.

Moffett, A., G. Ettlinger, H. B. Morton, and M. F. Piercy. 1967. "Tactile discrimination performance in the monkey: The effect of ablation of various subdivisions of posterior parietal cortex." *Cortex* **3: 59–96**.

Mountcastle, V. B., J. C. Lynch, and A. Georgopoulos. 1975. "Posterior parietal association cortex of the monkey: Command functions for operations within extrapersonal space." *J. Neurophysiol.* **38: 871–908**.

Nauta, W. J. H., and P. A. Gygax. 1954. "Silver impregnation of degenerating axons in the central nervous system: A modified technique." *Stain Tech.* **29: 91–94**.

Newcombe, F., and W. R. Russell. 1969. "Dissociated visual perceptual and spatial deficits in focal lesions of the right hemisphere." *J. Neurol. Neurosurg. Psychiat.* **32: 73–81**.

Pandya, D. N., and H. G. J. M. Kuypers. 1969. "Cortico-cortical connections in the rhesus monkey." *Brain Res.* **13: 13–36**.

Pasik, P., T. Pasik, W. S. Battersby, and M. B. Bender. 1958. "Visual and tactual discriminations by macaques with serial temporal and parietal lesions." *J. Comp. Physiol. Psychol.* **51: 427–436**.

Petrides, M., and S. D. Iversen. 1979. "Restricted posterior parietal lesions in the rhesus monkey and performance on visuospatial tasks." *Brain Res.* **161: 63–77**.

Pohl, W. 1973. "Dissociation of spatial discrimination deficits following frontal and parietal lesions in monkeys." *J. Comp. Physiol. Psychol.* **82: 227–239**.

Pribram, H. B., and J. Barry. 1956. "Further behavioral analysis of parieto-temporo-preoccipital cortex." *J. Neurophysiol.* **19: 99–106**.

Pribram, K. H., D. N. Spinelli, and S. L. Reitz. 1969. "The effects of radical disconnexion of occipital and temporal cortex on visual behaviour of monkeys." *Brain* **92: 301–312**.

Ratcliff, G., and G. A. B. Davies-Jones. 1972. "Defective visual localization in focal brain wounds." *Brain* **95: 49–60**.

Ratcliff, G., and F. Newcombe. 1973. "Spatial orientation in man: Effects of left, right, and bilateral posterior cerebral lesions." *J. Neurol. Neurosurg. Psychiat.* **36: 448–454**.

Ratcliff, G., R. M. Ridley, and G. Ettlinger. 1977. "Spatial disorientation in the monkey." *Cortex* 13:62–65.

Ridley, R. M., and G. Ettlinger. 1973. "Visual discrimination performance in the monkey: The activity of single cells in infero-temporal cortex." *Bruin Res.* 55: 179–182.

————. 1975. "Tactile and visuo-spatial discrimination performance in the monkey: The effects of total and partial posterior parietal removals." *Neuropsychologia* 13: 191–206.

Riopelle, A. J., H. F. Harlow, P. H. Settlage, and H. W. Ades. 1951. "Performance of normal and operated monkeys on visual learning tests." *J. Comp. Physiol. Psychol.* 44: 283–289.

Robinson, D. L., M. E. Goldberg, and G. B. Stanton. 1978. "Parietal association cortex in the primate: Sensory mechanisms and behavioral modulations." *J. Neurophysiol.* 41: 910–932.

Rocha-Miranda, C. E., D. B. Bender, C. G. Gross, and M. Mishkin. 1975. "Visual activation of neurons in inferotemporal cortex depends on striate cortex and forebrain commissures." *J. Neurophysiol.* 38: 475–491.

Rockland, K. S. 1979. "Cortical connections of the occipital lobe in the rhesus monkey." Ph.D. diss., Boston University.

Rockland, K. S., and D. N. Pandya. 1979. "Laminar origins and terminations of cortical connections of the occipital lobe in the rhesus monkey." *Bruin Res.* 179: 3–20.

Rolls, E. T., S. J. Judge, and M. K. Sanghera. 1977. "Activity of neurones in the inferotemporal cortex of the alert monkey." *Brain Res.* 130: 229–238.

Rolls, E. T., D. Perrett, S. J. Thorpe, D. Puerto, A. Roper-Hall, and S. Maddison. 1979a. "Responses of neurons in area 7 of the parietal cortex to objects of different significance." *Bruin Res.* 169: 194–198.

Rolls, E. T., M. K. Sanghera, and A. Roper-Hall, 1979b. "The latency of activation of neurones in the lateral hypothalamus and substantia innominata during feeding in the monkey." *Bruin Res.* 164: 121–135.

Sanghera, M. K., E. T. Rolls, and A. Roper-Hall. 1979. "Visual responses of neurons in the dorsolateral amygdala of the alert monkey." *Exp. Neurol.* 63: 610–626.

Schneider, G. E. 1967. "Contrasting visuomotor functions of tectum and cortex in the golden hamster." *Psychol. Forsch.* 31: 52–62.

Seacord, L., C. G. Gross, and M. Mishkin. 1979. "Role of inferior temporal cortex in interhemispheric transfer." *Bruin Res.* 167: 259–272.

Semmes, J. 1967. "Manual stereognosis after brain injury." In *Symposium on Oral Sensation und Perception,* J. F. Bosma, ed. Springfield, Ill.: Thomas.

Semmes, J., S. Weinstein, L. Ghent, and H.-L. Teuber. 1963. "Correlates of impaired orientation in personal and extrapersonal space." *Bruin* 86: 747–772.

Spiegler, B. J., and M. Mishkin. 1978. "Evidence for the sequential participation of inferior temporal cortex and amygdala in stimulus-reward learning." *Soc. Neurosei. Abstr.* 4: 263.

Stanton, G. B., W. L. R. Cruce, M. E. Goldberg, and D. L. Robinson. 1977. "Some ipsilateral projections to areas PF and PG of the inferior parietal lobule in monkeys." *Neurosci. Lett.* 6: 243–250.

Stein, J. 1978. "Effects of parietal lobe cooling on manipulative behaviour in the conscious monkey." In *Active Touch,* G. Gordon, ed. Oxford: Pergamon.

Sugishita, M., G. Ettlinger, and R. M. Ridley. 1978. "Disturbance of cage finding in the monkey." *Cortex* 14: 431–438.

Sunshine, J., and M. Mishkin. 1975. "A visual-limbic pathway serving visual associative functions in rhesus monkeys." *Fed. Proc.* 34: 440.

Trevarthen, C. B. 1968. "Two mechanisms of vision in primates." *Psychol. Forsch.* 31: 299–337.

Ungerleider, L. G., and B. A. Brody. 1977. "Extrapersonal spatial orientation: The role of posterior parietal, anterior frontal, and inferotemporal cortex." *Exp. Neurol.* 56: 265–280.

Ungerleider, L. G., and M. Mishkin. 1978a. "Interactions of striate and posterior parietal cortex in spatial vision." *Soc. Neurosci. Abstr.* 4: 649.

———. 1978b. "The visual area in the superior temporal sulcus of *Macaca mulatta:* Location and topographic organization." *Anat. Rec.* 190: 568.

———. 1979a. "Three cortical projection fields of area 17 in the rhesus monkey." Soc. *Neurosci. Abstr.* 5: 812.

———. 1979b. "The striate projection zone in the superior temporal sulcus of *Macaca mulatta:* Location and topographic organization." *J. Comp. Neurol.* 188: 347–366.

Ungerleider, L. G., and K. H. Pribram. 1977. "Inferotemporal versus combined pulvinar-prestriate lesions in the rhesus monkey: Effects on color, object, and pattern discrimination." *Neuropsychologia* 15: 481–498.

Van Essen, D. C., J. H. R. Maunsell, and J. L. Bixby. 1979. "Areal boundaries and topographic organization of visual areas V2 and V3 in the macaque monkey." *Soc. Neurosci. Abstr.* 5: 812.

Weller, R. E., and J. H. Kaas. 1978. "Connections of striate cortex with the posterior bank of the superior temporal sulcus in macaque monkeys." *Soc. Neurosci. Abstr.* 4: 650.

Weller, R. E., J. Graham, and J. H. Kaas. 1979. "Cortical connections of striate cortex in macaque monkeys." *Invest. Ophthalmol.* (ARVO suppl.) 18: 157.

von Bonin, G., and P. Bailey. 1947. *The Neocortex of Macaca Mulatta.* Urbana: University of Illinois Press.

von Bonin, G., H. W. Garol, and W. S. McCulloch. 1942. "The functional organization of the occipital lobe." *Biol. Symp.* 7: 165–192.

Wilson, M. 1957. "Effects of circumscribed cortical lesions upon somesthetic and visual discrimination in the monkey." *J. Comp. Physiol. Psychol.* **50:** 630–635.

———. 1965. "Tactual discrimination learning in monkeys." *Neuropsychologia* **3:** 353–361.

———. 1978. "Visual system: Pulvinar-extrastriate cortex." In *Handbook of Behavioral Neurobiology,* vol. 1: *Sensory Integration.* R. B. Masterton, ed. New York: Plenum.

Wilson, M., J. A. Stamm, and K. H. Pribram. 1960. "Deficits in roughness discrimination after posterior parietal lesions in monkeys." *J. Comp. Physiol. Psychol.* **53:** 535–539.

Wurtz, R. H. 1969. "Visual receptive fields of striate cortex neurons in awake monkeys."]. *Neurophysiol.* **32:** 727–742.

Zeki, S. M. 1969. "Representation of central visual fields in prestriate cortex of monkey." *Brain Res.* **14:** 271–291.

———. 1971a. "Convergent input from the striate cortex (area 17) to the cortex of the superior temporal sulcus in the rhesus monkey." *Brain Res.* **28:** 338–340.

———. 1971b. "Cortical projections from two prestriate areas in the monkey." *Brain Res.* **34:** 19–35.

———. 1976. "The projections to the superior temporal sulcus from areas 17 and 18 in the rhesus monkey." *Proc. Roy. Soc.* **B 193:** 199–207.