Visual Functions of Inferotemporal Cortex

By

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With 5 Figures
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Introduction

Traditionally, the cerebral cortex on the inferior surface of the temporal lobe was considered part of the “silent” or “association” areas of the brain. The term “association cortex” was first used by FLECHSIG [63] to describe cortical regions that became myelinated relatively late. He thought that “association” among the senses occurred here. In subsequent decades association cortex became the presumed site of “association of ideas” and then of association or linkage between stimuli and responses in learning.

More recently, association cortex has been defined as cortex that is neither sensory nor motor in function, but, defined in this way, the extent of association
cortex continues to diminish as more and more "supplementary" motor and sensory cortical areas are described. Today it is clear that the cortex on the inferior convexity of the temporal lobe, inferotemporal cortex, is involved exclusively in visual functions. Its neurons have visual receptive fields and highly specific response properties. Ablation of inferotemporal cortex produces a deficit in visually guided behavior that seems to be a "higher order" dysfunction: visual learning ability is impaired, but basic visuo-sensory functions remain unaltered.

This chapter\(^1\) begins with a review of the behavioral effects of ablation of inferotemporal cortex since these results pose the questions for the anatomy and physiology of this area. The second section summarizes the anatomical connections of inferotemporal cortex, and in the third section the evidence on the functional significance of these connections is examined. In the fourth section, electrophysiological experiments on inferotemporal cortex are discussed with emphasis on recent single neuron recordings. The fifth section considers inferotemporal cortex in the more general context of vertebrate visual systems. In the final section, some brief speculations on the visual functions of inferotemporal cortex are offered.

This chapter deals almost exclusively with the monkey\(^2\), since this is the only animal in which the functions of inferotemporal cortex have been extensively studied.

**Behavioral Effects of Removal of Inferotemporal Cortex**

**Discovery and Localization of the Inferotemporal Syndrome**

In the late 1930’s, Klüver and Bucy [105—107] reported a bizarre constellation of behavioral changes following bilateral removal of the temporal lobe of monkeys. These monkeys failed to recognize objects visually, showed "insatiable visual curiosity", and were deficient in learning and remembering visual discrimination habits. Klüver and Bucy termed these symptoms "psychic blindness". In addition, the temporal lobectomized monkeys compulsively touched and mouthed objects, were unnaturally tame and docile, and showed abnormal sexual behavior\(^3\). These effects of temporal lobectomy became known as the Klüver-Bucy syndrome. The Klüver-Bucy syndrome was fractionated by Karl Lashley’s associates and their students in the 1950’s. That is, they showed that its components could be produced independently by smaller temporal lobe lesions. The impairment on visual discrimination tasks followed lesions of the neocortex of the temporal lobe, whereas the remaining aspects of the syndrome resulted from destruction of rhinencephalic structures of the temporal lobe, particularly the amygdala [148, 14, 32]. Subsequent studies demonstrated that "inferotemporal cortex" (comprising most

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\(^1\) This chapter was completed in March 1970 and is based on the literature up to that date.

\(^2\) In all the "monkey" studies cited herein, when authors gave the species, it was almost always *Macaca mulatta*. Exceptions include apparent use of *M. speciosa*, *Papio papio*, *P. porcarius* and *P. cynocephalus*. No differences among these species in regard to inferotemporal function have been reported.

\(^3\) Essentially similar observations had been made 50 years earlier by Brown and Schaefer [19]. However, they had attributed their findings to widespread vascular involvement rather than to the temporal lobectomy itself. At this time even the site and existence of a cortical visual area and a cortical auditory area were subjects of bitter controversy; thus, the apparently "generalized dementia" produced by temporal lobe lesions generated little interest.
of the middle and inferior gyri of the temporal lobe and approximating Area TE of von Bonin and Bailey [16] was crucial for normal visual discrimination learning [128, 132]. By contrast, lesions of the superior temporal gyrus (Areas TA, TB, TC), of the temporal pole (Area TG) and of the fusiform and hippocampal gyri (Area TF) had no effect on visual discrimination learning [e.g., 129, 146]. These areas are shown in Fig. 1. In some of the earlier studies, the inferotemporal lesions encroached on the visual radiations which course through the subcortical white matter of the temporal lobe, raising the possibility that this damage might have been the basis of the resulting visual impairments. However, it was soon clear that damage to the optic radiations is neither necessary nor sufficient to produce the severe deficit of visual learning that follows inferotemporal lesions. Indeed large lesions of striate cortex, including the projection of the central 10° of the retina, have virtually no effect on the learning of visual discrimination tasks [e.g., 207, 44].

![Diagram of lateral view of cerebral hemisphere of Macaca mulatta showing sulci and cortical areas discussed in the text. The capital letters designate cytoarchitectonic areas of von Bonin and Bailey [16]. The circumstriate belt is shown after Kuyper et al. [109]. Von Bonin and Bailey [16] divide this region into an Area OB, adjacent to Area OC, and a more rostral Area OA. The dashed lines indicate the approximate borders of the projection of foveal striate cortex onto the circumstriate belt; much of this projection lies in the lunate, inferior occipital and superior temporal sulci [45, 210]. INTRAPAR. S.: intraparietal sulcus; PRINC. S.: principal sulcus; LAT. F.: lateral fissure; SUP. TEM. S.: superior temporal sulcus; INF. OCC. S.: inferior occipital sulcus]

The effects of lesions of the tissue between inferotemporal cortex and striate cortex, that is, lesions of prestriate cortex or the circumstriate belt (see Fig. 1), were somewhat less clear. Some investigators reported no changes in visual discrimination performance after prestriate lesions, whereas others reported small deficits [112, 33, 61, 123, 159, 204, 2, 3, 158, 152, 60, 201, 209]. In all cases the deficits were far less severe than those following inferotemporal lesions. However, in these studies, the prestriate lesions were subtotal, particularly sparing buried cortex on the ventrolateral surface. The effects of removing this tissue are discussed below (pp. 461—463).
The visual functions of inferotemporal cortex can be interfered with by methods other than bilateral ablation. Visual habits are impaired both by electrical stimulation of inferotemporal cortex [35, 37] and by epileptiform discharges induced by alumina cream implantation [180]. Unilateral lesions are ineffective, except in “split-brain” animals (in which both the optic chiasm and corpus callosum are sectioned), and then only when visual input is restricted to the eye ipsilateral to the temporal lesion [54, 24].

In summary, inferotemporal cortex is necessary for normal visual discrimination learning.

Behavioral Analysis of the Inferotemporal Syndrome

The inferotemporal deficit is exclusively visual. Learning and retention of olfactory, auditory, tactile, and gustatory discrimination problems are unaltered by inferotemporal lesions [20, 21, 141, 149, 193, 196, 14, 129].

The earliest investigators of monkeys with inferotemporal lesions were struck by the normal behavior of their monkeys outside formal learning situations. Lesioned monkeys that were incapable of learning visual discrimination tasks could pick gnats out of the air with an accuracy at least as great as that of their observer [146]. More formal tests have continued to fail to find any “strictly sensory” impairment after inferotemporal lesions. Inferotemporal lesions have no deleterious effect on minimum separable visual acuity [192], on the integrity of the visual fields [43, 44], on the critical flicker frequency [184], on the threshold for detection of a brief visual stimulus [10], or on backward masking by pattern and by flash [10]. Inferotemporal lesions usually slow the training procedure necessary to obtain these psychophysical functions, but the asymptotic performance is always indistinguishable from that of normal animals.

Of course, the negative data obtained so far are not exhaustive and do not rule out some more subtle sensory changes. They are sufficient, however, to make it highly unlikely that a simple sensory impairment is the principal cause of the visual discrimination impairment after inferotemporal lesions. Thus, the inferotemporal syndrome seems to fit Freud’s [65] classic definition of agnosia: a modality-specific disturbance in “higher-order” or recognition function in the absence of any elementary sensory disturbances sufficient to account for the recognition deficit.

Data are sparse on the effects of inferotemporal lesions on visuomotor integration. In perhaps the only formal test of complex hand-eye coordination, Butler [25] showed that inferotemporal lesions did not alter a monkey’s ability to track and seize a rapidly and erratically moving peanut. We have shown that monkeys with inferotemporal lesions have altered patterns of eye movements during visual discrimination performance. In a two-choice situation they tend to look longer at the discriminanda and shift their gaze between the discriminanda less than normal monkeys [137, 138]. Moreover, when staring at one discriminandum they show a greater rate of change of fixation within its borders [195]. These results have two possible types of interpretations. One is that the oculomotor changes reflect a dysfunction in scanning or information gathering that is the cause of all the other aspects of the inferotemporal syndrome, thereby disqualifying it as a pure agnosia. Alternatively, the abnormal pattern of eye movements may be a consequence of
some unknown perceptual or mnemonic deficit. If the animal cannot "recognize" a stimulus, it may try to look at it longer or differently.

What types of visual discrimination problems are impaired by inferotemporal lesions? Among the deficits repeatedly reported are ones involving hue, brightness, two-dimensional patterns, and three-dimensional objects, whether the discrimina-nanda are presented simultaneously or successively [150], are few or many [144], or are presented in discrete trials or with free operant procedures [30]. Inferotem-poral lesions impair both the retention of visual discriminations acquired prior to surgery and the acquisition of new visual discriminations following surgery [e.g., 41]. They impair both the learning of individual problems and the rate at which monkeys improve their performance on successive problems, i.e., acquire visual discrimination learning sets [e.g., 129].

The visual discrimination impairment that follows inferotemporal lesions is not an absolute one. Its occurrence and severity depends on the "difficulty" of the discrimination, where difficulty of a task is measured by the number of trials a normal monkey, with a comparable training history, requires to learn the discrimi-nation [e.g., 128, 132, 150, 131]. Difficulty of task usually depends on the magnitude of physical differences between the discriminaanda and upon the training procedures used. A visual discrimination task which normal monkeys learn in one to 30 trials will be learned in a similar number of trials by monkeys with inferotemporal lesions. However, a task that normal monkeys learn in 300–500 trials, animals with inferotemporal lesions usually fail to learn in a thousand or more trials.

There are three interesting exceptions to the generalization that the severity of the inferotemporal deficit is a function of task difficulty. The first is that preoperative overtraining on a specific discrimination eliminates the usual deficit on postoperative retention of that discrimination [122, 136, 38]. The second exception is that temporal lesions in infant monkeys do not alter their subsequent ability to learn visual discriminations [153]. The third case in which inferotemporal lesions do not impair acquisition of difficult visual discrimination tasks is when shock is used for punishment of incorrect responses [116]. (In all previous studies, food or water was used to reward correct responses.) Presumably, in these three cases, other mechanisms are able to compensate totally for the absence of inferotemporal cortex. Whether the compensation is similar in all three cases, and whether it involves the surrounding tissue [cf. 69] or subcortical structures is unknown.

The inferotemporal deficit is a permanent one: monkeys with this lesion remain impaired for years when compared to control animals with the same training history. What about retention of discrimination tasks acquired after operation? Are animals with inferotemporal lesions impaired in remembering a discrimination after it is learned? Apparently not. If animals with inferotemporal lesions and control animals are trained to a criterion of 18 correct out of 20 responses, the animals with inferotemporal lesions take longer than controls to successively reach more stringent criteria of 45 correct out of 50, and 90 correct out of 100. This phenomenon is often reflected in a more negatively accelerating learning curve in animals with inferotemporal lesions. However, once a visual discrimination is well established after operation by postcriterial overtraining (or, equivalently, a very stringent learning criterion), subsequent retention of the task is normal [74].
Many investigators have attempted to determine the “nature” or “basis” of the visual impairment that follows inferotemporal lesions by using tests that afford more analytical possibilities than the usual two-choice visual discrimination learning task. These tests include stimulus equivalence tests [20–29, 205, 95], stimulus generalization tests [30], discrimination reversal tasks [73], matching-to-sample tasks [22], partial reinforcement schedules [116, 117], size constancy tests [93], concurrent discrimination tasks [97, 41], variations in stimulus redundancy [199, 200], visual memory tests [205, 202, 94, 96], and bar pressing motivated by visual incentives [183, 114]. The results of these experiments have been used to support specific theories of inferotemporal function such as the notion that this cortex subserves visual attention functions or visual memory functions. (See discussions by Butter [26], Weiskrantz [190, 191], Pribram [144–147], and M. Wilson [198].) However, it is not clear whether these findings indicate anything specific about the inferotemporal deficit. Rather, the changes in stimulus equivalence, stimulus generalization, and so on may simply reflect the impaired learning of visual discriminations after inferotemporal lesions, whatever the physiological basis of this impairment may be.

In summary, inferotemporal lesions produce a severe impairment in the postoperative acquisition and retention of a variety of visual discrimination habits; this impairment exists in the apparent absence of any visual sensory losses or deficits on non-visual tasks.

Are There Analogous Syndromes in Other Modalities?

Other regions of “association cortex” in the monkey also produce modality-specific deficits in discrimination learning. Posterior parietal lesions impair somesthetic discrimination learning, superior temporal lesions impair auditory discrimination learning, and temporal pole lesions impair olfactory discrimination learning. Are these deficits analogous to the inferotemporal syndrome, and has their study thrown light on the general function of these “uni-modality learning areas” of the brain?

Ablation of posterior parietal cortex (sparing the somesthetic cortex of the post-central gyrus) impairs the postoperative retention and acquisition of tactile discrimination tasks and leaves performance in other types of learning unaffected [59, 13, 8, 196, 197, 149, 133, 141, 203]. As in the case of inferotemporal lesions, the severity of the deficit depends on the difficulty of the task. In the only study of somesthetic thresholds after posterior parietal lesions, there was no evidence for a massive loss in roughness discrimination thresholds [203]. Thus, in these regards, the posterior parietal deficit in somesthesia appears parallel to the inferotemporal deficit in vision, as M. Wilson [196, 197] and Pribram [146] have suggested.

However, in addition to their tactile discrimination impairment, animals with posterior parietal lesions usually show “awkwardness and poverty of movement; gross inaccuracy in reaching for objects and often striking spatial disorientation” [172]. On the basis of these associated symptoms and of the effects of parietal lobe damage in man, Ettlinger and his colleagues [8, 59, 133] have argued that “selective motor retardation” is the basis of the impairment on tactile discrimination,
whereas Semmes [171, 172] has suggested that it reflects a combination of somesthetic, sensory, and spatial orientation dysfunctions. Tactile discrimination tasks involve active palpating of the discriminanda, just as visual discrimination involves an active scanning. Thus, it is conceivable that the abnormal eye movements after inferotemporal lesions (p. 454) are parallel to the much more prominent motor and orientation dysfunctions after posterior parietal lesions.

Lesions of the superior temporal gyrus in the monkey that spare auditory cortex (i.e., lesions of Area TA) produce a deficit in the postoperative acquisition and retention of auditory discrimination tasks, while leaving discrimination in other modalities unaffected [193, 50, 167]. In the only threshold determination after such superior temporal lesions, decreased sensitivity to the duration of an auditory sequence was found [40]. Unfortunately, comparable data are not available for the effects of inferotemporal lesions on the equivalent visual threshold. More disquieting is the fact that the effects of ablation of primary auditory cortex itself are still contradictory and confusing [11, 50, 62, 99, 118, 119, 163, 185, 189].

Finally, lesions that include the temporal pole impair postoperative acquisition and retention of olfactory discrimination, and not other learning tasks [20, 21, 129]. The critical lesions may include the anteromedial allocortex of the temporal lobe. Little else is known about this deficit.

In summary, lesions of posterior parietal cortex, of superior temporal cortex, and of the temporal pole each produce modality-specific deficits in discrimination learning. Whether these deficits are analogous to the inferotemporal one is not yet clear.

**The Effects of Temporal Lobe Damage in Man**

The only primate besides the macaque in which the effects of temporal lobe damage have been extensively studied is man. The human brain is unique in its lateralization of function: unilateral lesions produce highly specific effects which depend on the side of removal. Damage to the dominant left temporal lobe produces language disturbances. In striking contrast, as Milner and her colleagues have persuasively demonstrated, cortical excision in the right temporal lobe produces a constellation of deficits on non-verbal tasks, both auditory [173, 125, 104] and visual.

Milner [126] divides the nonverbal visual deficits that follow right temporal lobe damage into ones of “perception” and ones of “memory”. Among the perceptual changes are deficits in tachistoscopic recognition [104, 52], in dot numerosity [104], in the classification of faces in which normal contour lines are eliminated [110], and in the detection of small differences in complex patterns [121]. The visual memory losses are apparently more striking than these perceptual effects [126]. They include impaired recall of geometric drawings but not of stories or word pairs [126], impaired recognition of faces [126] and nonsense figures [104], but not of words, numbers, or nonsense syllables.

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4 Deficits on auditory discrimination tasks have also been reported after dorsolateral frontal cortex lesions. This deficit, however, is not modality-specific and appears dependent on particular testing techniques [15, 193, 161, 75, 78, 72, 189, 9, 113].
The perceptual and memory deficits are apparently independent of damage to the underlying visual radiations. At least, the perceptual deficits are also independent of associated hippocampal damage [127]. Thus, as Milner [124, 126] has suggested, these visual deficits appear to be similar to the inferotemporal syndrome found in monkeys. Of course, to establish this similarity conclusively, it would be necessary to show that lesions of the right superior temporal gyrus in man led only to the auditory dysfunctions, and that lesions of the right middle and inferior gyri led only to the visual dysfunctions, and such data are unlikely to be forthcoming.

In summary, excision of the cortex of the right temporal lobe of man produces visual deficits that seem similar to those following bilateral inferotemporal lesions in the monkey.

**Anatomy of Inferotemporal Cortex**

**Cytoarchitectonic Studies**

In the studies reviewed in the last section, the inferotemporal lesions approximated Area TE, defined by von Bonin and Bailey [16] on cytoarchitectonic grounds. However, there was considerable variation in the caudal extent of these lesions and, indeed, there is some question about the caudal border of Area TE.

Von Bonin and Bailey place the border between Area TE and the circumstriate belt (which they divide into Areas OA and OB) at about 2–6 mm. anterior to the dorsal termination of the inferior occipital sulcus, but in their text they stress the difficulty of distinguishing these two areas, and in their color plate they reflect this by blending the color designations of these areas along their border (see Fig. 1). In the chimpanzee, they [17] distinguish a separate area between TE and OA which they call “TEO(PH)” and which, in man, corresponds with von Economo’s Area PH and Brodmann’s Area 37. Petr et al. [143] and McCulloch [120], on the basis of strychnine neuronography in the macaque, also designated this temporal-preoccipital border region as Area 37. The reality of such an intermediate Area is supported by the fact that the commissural connections of this transitional zone are different from those of Area TE proper. The former involve the corpus callosum and the latter the anterior commissure [181, 64, 194]. The importance of this problem, as will be seen later, is that the effects of removing this transitional zone and adjacent OA tissue are different from those produced by Area TE lesions (pp. 462–463 and footnote 6).

In summary, studies of the effects of removing inferotemporal cortex involved lesions of the cytoarchitectonic Area TE of von Bonin and Bailey. However, the exact posterior border of TE is unclear, and some investigators have designated an area intermediate between TE and the circumstriate belt termed TEO, PH, or Area 37.

**Afferent Connections of Inferotemporal Cortex**

The existence of cortico-cortical connections from lateral striate cortex to the circumstriate belt (prestriate cortex) and from the circumstriate belt to Area TE have been established by Nauta staining methods [109, 139]. The details of the pro-

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Brodmann’s Areas 20 and 21 are similar except that they include the temporal pole [18].
jections of lateral striate cortex onto the circumstriate belt have recently been described by Zeki [210–213] and Cragg and Ainsworth [45] who also used modified Nauta methods. The projections are multiple and complex and involve several synapses within the circumstriate belt. The foveal portions of these projections include the banks of the ventral portion of lunate sulcus, the posterior bank of the caudal portion of the superior temporal sulcus, the posterior bank of the ascending inferior occipital sulcus, and the cortex between these sulci (see Fig. 1). Thus, Cowey and Gross [41] termed this area of ventrolateral prestriate cortex “foveal prestriate cortex”. Foveal prestriate cortex, in turn, projects in a non-retinotopic fashion to Area TE [39].

The only other cortico-cortical inputs to inferotemporal cortex established by Nauta methods are from the ventral portion of dorsolateral frontal granular cortex [139, 109] and from the opposite inferotemporal cortex. The latter projection is carried by the anterior commissure [194, 64].

The major subcortical projection to inferotemporal cortex that has been demonstrated by retrograde degeneration studies is one from the pulvinar [31, 41]. The pulvinar, in turn, receives afferents from the superior colliculus [100, 130] and probably from striate cortex [135, 12]. Both anterograde and retrograde studies have failed to demonstrate any projections from the lateral geniculate body to inferotemporal cortex.

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Fig. 2. Major connections of inferotemporal cortex in Macaca mulatta. The large arrowheads indicate retinotopic projections. Each arrowhead is not intended to indicate a single synapse. (VL putamen: ventrolateral putamen.)
The pulvinar projects in a topographically organized fashion to the entire posterior association cortex (superior temporal cortex, inferotemporal cortex, the circumstriate belt, and posterior parietal cortex) [31]. After lesions of Area TE, retrograde degeneration is largely confined to the posterior and ventral portions of *n. pulvinaris lateralis*. By contrast, lesions of foveal prestriate cortex produce degeneration confined to the posterior three quarters of *n. pulvinaris inferior* [31, 98, 41].

In summary, inferotemporal cortex receives projections from the circumstriate belt, lateral frontal cortex, the pulvinar, and the contralateral inferotemporal cortex (see Fig. 2).

**Efferent Connections of Inferotemporal Cortex**

Inferotemporal cortex projects both to the circumstriate belt and to the ventral portion of dorsolateral frontal cortex [139, 109]. Both connections are approximately reciprocal. The inferior portions of inferotemporal cortex project to the entorhinal area and to the periamygdaloid cortex [194]. The entorhinal area, in turn, projects to the hippocampus [4].

Whitlock and Nauta [194] have demonstrated a number of subcortical projections from inferotemporal cortex. They made relatively small lesions, and compared the projections of the anterior and posterior parts of the inferior temporal gyrus. The interest of this comparison to the present discussion is that their posterior lesion was in the transitional zone between Areas TE and OA. Both their anterior and posterior sites send projections to the tail of the caudate nucleus, the caudoventral putamen, the dorsomedial nucleus of the thalamus, and the pulvinar. However, the anterior site sends much more massive projections to the laterobasal nuclei of the amygdala, and only the posterior site sends projections to the superior colliculus. Many of these projections, including the anterior-posterior differences, were also found in an electrophysiological study by Reitz and Pribam [155].

In summary, inferotemporal cortex sends efferents to the circumstriate belt, the frontal lobe, and to several subcortical structures, including basal ganglia, the dorsomedial nucleus, the pulvinar, and the superior colliculus (see Fig. 2.).

**The Anatomic Basis of the Visual Functions of Inferotemporal Cortex**

In the previous sections the anatomic relations of inferotemporal cortex and the evidence for its exclusively visual function were summarized. This section will examine the anatomic basis of these visual functions.

**Prestriate Cortex as the Afferent Route to Inferotemporal Cortex**

Although striate cortex does not project to inferotemporal cortex, striate cortex does send fibers to the circumstriate belt and, in turn, the circumstriate belt projects to inferotemporal cortex (pp. 458–459). Thus, it has been suggested that inferotemporal cortex might receive visual information by way of prestriate cortex [35, 129, 169, 77].
The first clear support for this hypothesis was a study in which Mishkin [129] indirectly but totally interrupted the cortico-cortical pathway from striate to inferotemporal cortex. He did so by making three successive lesions. First, he totally removed striate cortex on one side, then inferotemporal cortex on the contralateral side, and finally he severed the corpus callosum. Thus, after the third lesion, the remaining inferotemporal cortex could not receive information from striate cortex over the occipito-temporal cortico-cortical pathway. Animals with these three lesions (but not animals with only two of them or with control lesions) showed the full inferotemporal deficit on visual discrimination performance. Ettinger [58] and Reit [134] obtained similar results in studies differing from Mishkin's principally in the use of unilateral optic tract section rather than unilateral striate cortex removal. Reit, however, interpreted her results as supporting Pribram's "efferent" model (see p. 464).

A major difficulty for the view that inferotemporal cortex receives visual information by way of prestriate cortex has been the repeated finding that large prestriate lesions, which should have interrupted this pathway, had little or no effect on visual discrimination learning (see p. 453). However, in all these studies (including one by Pribram et al. [152] which involved by far the most extensive prestriate lesions yet reported), the lesions were not complete. They invariably failed to remove completely and bilaterally the depths of at least one of the following sulci: the lunate sulcus, the inferior occipital sulcus, and the ventral bank of the posterior part of the superior temporal sulcus. These areas include a major portion of the projection of striate cortex onto the circumstriate belt [45, 210].

Recently, Iwai and Mishkin [97], Mishkin [130], and Cowey and Gross [41] showed that relatively small prestriate lesions could produce a visual pattern discrimination impairment more severe than the one usually seen following inferotemporal lesions. These lesions were confined to ventrolateral prestriate cortex, including the anterior bank of the lunate sulcus, a small portion of the ventral bank of the posterior part of the superior temporal sulcus, the ascending portion of the inferior occipital sulcus, and extending ventrally to the occipitotemporal sulcus (see Fig. 1). Much of this cortex is buried in sulci and had never been totally removed in previous studies. The importance of this tissue is presumably that it includes the entire projection of foveal striate cortex onto prestriate cortex. Foveal prestriate cortex, in turn, projects to inferotemporal cortex. Thus, a crucial pathway for normal functioning of inferotemporal cortex appears to be the foveal portion of the projection of striate cortex onto the circumstriate belt. Further support for this view is our finding (p. 469)

* We have called these lesions "foveal prestriate lesions" because they include the entire area of prestriate cortex to which foveal striate cortex selectively projects [210, 45]. Simply to call them "pre striate cortex" lesions or "posterior inferotemporal cortex" lesions is misleading because they include far less than the former and far more than the latter. Iwai and Mishkin [98, 130] have called essentially the same lesion one of "Area TEO". Note that since the anterior border of foveal prestriate cortex is still unspecified, it is possible that the deleterious effects of "foveal prestriate lesions" could have been due, at least in part, to the removal of tissue between Area TE and the foveal projection onto prestriate cortex, rather than entirely to the ablation of the latter area. That is, a functionally and anatomically discrete area may exist between Area TE and the circumstriate belt.
that the visual receptive fields of neurons in inferotemporal cortex all include the fovea and are dependent on striate cortex.

For three reasons, foveal prestriate lesions probably do not totally disconnect inferotemporal cortex from visual afferents. The first is that total removal of the circumstriate belt, including all of foveal prestriate cortex, has a somewhat more deleterious effect on visual discrimination than removal of foveal prestriate cortex alone [130]. Second, although the visual receptive fields of single neurons in inferotemporal cortex invariably include the fovea, they extend at least 5° and often over 30° from fovea (p. 466). Third, as described in the next section, on certain visual tasks, inferotemporal lesions produce greater deficits than do foveal prestriate lesions; this would argue against inferotemporal cortex receiving all of its visual input from foveal prestriate cortex. These considerations suggest that when foveal prestriate cortex is removed, inferotemporal cortex still receives some information from striate cortex by way of "extra-foveal" prestriate cortex.

In summary, a severe impairment in visual discrimination follows interruption of the occipital-temporal pathway by means of foveal prestriate lesions. These results support the hypothesis that inferotemporal cortex receives visual information from striate cortex over this pathway.

**Comparison of the Effects of Foveal Prestriate and Inferotemporal Cortex Lesions**

As mentioned in the previous section, it was shown by Iwai and Mishkin [97, 130] and by us [41] that foveal prestriate lesions produce a more severe impairment on postoperative acquisition and retention of visual pattern discrimination tasks than do lesions confined to inferotemporal cortex. However, the difference between the effects of the two lesions is not simply a quantitative one. On some visual tasks, animals with foveal prestriate lesions are more impaired than those with inferotemporal lesions, and the converse is true on other types of visual problems. Specifically, foveal prestriate lesions produce greater impairment on three-dimensional object discrimination [79], on two-dimensional pattern discrimination [97, 130, 41], and on pattern stimulus equivalence tests [97]; they also cause greater disruption of discrimination performance by introduction of irrelevant visual stimuli [79]. By contrast, inferotemporal lesions produce greater impairment on color discrimination [79], on concurrent discrimination\(^7\) [97, 130, 24, 41], greater retardation in visual learning by the use of partial rather than continuous reinforcement schedules [117], greater interference in retention of visual problems after interpolation of dissimilar material [79], and greater improvement with the addition of punishment for incorrect responses [116].

Thus, the impairments produced by foveal prestriate lesions appear to involve tasks for which foveal information is particularly important, such as pattern discrimination. On the other hand, animals with inferotemporal lesions are more affected by manipulation of reinforcement, motivation and mnemonic variables; for example, they are more disrupted by partial reinforcement, more improved by

\(^7\) A concurrent discrimination involves the parallel learning of several discrimination tasks, each of which would be extremely simple if presented alone. The pairs of discriminanda are intermingled from trial to trial in every training session until all the discriminations are learned.
punishment, and have greater difficulty on the concurrent discrimination in which pro- and retroactive interference among the discriminanda are maximal [72a].

It is difficult, at this time, to categorize the contrasting deficits following inferotemporal and foveal prestriate lesions in psychological terms, let alone neurophysiological ones. The impairment after inferotemporal lesions is clearly not a simple sensory one. Furthermore, the constellation of behavioral changes it produces implies an associative type of deficit rather than an attentional or perceptual one. On the other hand, the pattern of deficits after foveal prestriate lesions suggests some sensory, attentional or perceptual dysfunction rather than any alteration in associative functions. Unfortunately there are insufficient data on the sensory status of monkeys with foveal prestriate lesions to determine whether their difficulty is due to sensory loss or, alternatively, to some attentional or perceptual deficit [cf. 41, 97, 130].

In summary, ablation of foveal prestriate cortex and of inferotemporal cortex both impair visual discrimination learning, but somewhat differently. The deficit after foveal prestriate lesions appears to be more a sensory, perceptual, or attentional one. By contrast, inferotemporal lesions produce an impairment relatively more sensitive to associative factors.

Functions of the Subcortical Connections of Inferotemporal Cortex

The pulvinar projects to inferotemporal cortex and to the circumstriate belt and, in turn, the pulvinar receives fibers from both the superior colliculus and striate cortex (p. 459). In both the cat and squirrel monkey, the pulvinar is responsive to visual stimuli [e.g., 182, 71, 115]. Thus, a superior colliculus-pulvinar-cortex route could be a source of visual information for inferotemporal cortex in addition to the occipital-temporal pathway. However, bilateral lesions of the pulvinar, even when total, do not affect visual pattern discrimination learning [34, 35, 175]. Furthermore, large lesions of the pulvinar do not increase the visual discrimination impairment produced by subtotal lesions in the circumstriate belt [35].

Thus, this afferent pathway is not necessary for the normal learning of conventional pattern discrimination tasks. Does it convey some other type of visual information to inferotemporal cortex? In a subsequent section we will cite evidence that the superior colliculus is involved in visual orientation and localization and suggest that this type of information may be conveyed to inferotemporal cortex by the colliculus-pulvinar projection. We will argue that inferotemporal cortex integrates the pattern analysis mechanisms of the geniculostriate system with the visual localization mechanisms of the superior colliculus (pp. 471—472).

Inferotemporal cortex sends major projections to the amygdala, the superior colliculus, the basal ganglia, and, by way of the entorhinal area, to the hippocampus (p. 460). The amygdala has been repeatedly implicated in reinforcement mechanisms [168], the superior colliculus in orientation functions [166], the basal ganglia in motor control [47], and the hippocampus in learning and memory [78, 170]. Thus, it is possible that the subcortical projections to these structures are involved in reinforcement, orientation, response, and storage aspects of visual discrimination, respectively.
Lesions in each of these structures have been reported to impair certain types of visual discrimination tasks [e.g., 7, 6, 178, 51, 55]. Although the resulting deficits appear to be different from each other and from the inferotemporal deficit, there has been relatively little systematic comparison among them. Indeed, unlike the inferotemporal syndrome, the behavioral changes after amygdala, superior colliculus, basal ganglia, and hippocampal lesions are probably not specifically visual changes. Rather, these areas may play particular roles in discrimination of all stimulus modalities, the amygdala in reinforcement, the superior colliculus in orientation, the basal ganglia in motor control, and the hippocampus in some storage mechanism. On this view, posterior parietal cortex (necessary for tactile learning), superior temporal cortex (necessary for auditory learning), and the temporal pole (necessary for olfactory learning), should send projections to all or some of these structures. Similarly, we predict that destruction of each of these structures should have a supramodal effect on one aspect of discrimination learning such as reinforcement, localization, response control, and storage. (However, if the terminations of the corticofugal projections onto these structures are spatially separate, then it might be possible to produce modality-specific impairments by discrete lesions. This may be the case for the basal ganglia [47, 51].) Unfortunately, the anatomical and ablation data to test these speculations are not yet available. (A somewhat similar hypothesis has been proposed by Geschwind [68].)

A rather different view of the functions of inferotemporal cortex and its subcortical efferents from that presented in this chapter has been proposed by Pribram and his colleagues [e.g., 176, 177, 146, 147, 152]. They have suggested that the role of inferotemporal cortex in visual discrimination lies in exerting a tonic inhibitory influence on the visual system by means of subcortical efferents from inferotemporal cortex. The principal experimental support for Pribram's "efferent model" was the demonstration that electrical stimulation of inferotemporal cortex depresses the excitability of striate cortex, as measured by recovery cycles of striate evoked responses to paired light flashes [176]. Pribram suggested that inferotemporal lesions would have the opposite effect, i.e., increased excitability of striate cortex. We directly tested this hypothesis and failed to find any effects of bilateral inferotemporal lesions on the recovery cycle of striate evoked responses [169]. Extensive undercutting of inferotemporal cortex [151, but cf. 35] does impair visual discrimination learning, but this is consistent with both Pribram's view of the role of subcortical efferents as well as the speculations presented above. Furthermore, this effect might have been due to interference with cortico-cortical U-fibers or with subcortical afferent fibers rather than subcortical efferents [151].

The ubiquity of reciprocal connections in the nervous system, the numerous centrifugal projections that modulate sensory input, and the importance of feedback mechanisms in those parts of the nervous system that we are beginning to understand, certainly make it likely that activity in inferotemporal cortex does affect lower levels of the visual system. However, there appears to be no clear evidence that this is the sole or most important function of inferotemporal cortex.

In summary, there are few hard data on the specific functions of the subcortical connections of inferotemporal cortex. The projection from the pulvinar is not necessary for the usual visual discrimination tasks, but may convey other types of
visual information to inferotemporal cortex. Several of the subcortical structures to which inferotemporal cortex projects are necessary for normal visual discrimination learning, but it is unlikely that these structures are exclusively visual in function. Finally, the evidence does not support the hypothesis that the main function of inferotemporal cortex is to modulate visual input.

**Physiology of Inferotemporal Cortex**

**Gross Evoked Potentials and EEG**

We have recorded evoked responses to light from monopolar and bipolar electrodes placed on inferotemporal cortex in unanesthetized animals [188, 67]. These responses are small, and thus usually must be averaged in order to be detected. The response to a brief diffuse light flash consists of a negative deflection peaking at 44 msec followed by a complex positive wave lasting up to 400 msec after the stimulus. This late complex wave is much more labile than both the early negative component and the entire visual evoked response recorded from striate cortex under identical conditions. The effects on the late component of the inferotemporal visual evoked response of unilateral striate cortex ablation are different from those of unilateral optic tract section, suggesting a subcortical as well as striate contribution to this part of the response [188].

Chow [36] has reported a transient decrease in amplitude of inferotemporal EEG in the course of acquisition of visual discrimination tasks. Stamm and Mahoney [179] found bursts of 6–8 per second activity in inferotemporal EEG associated with the early stage of learning a visual discrimination. We have occasionally seen a similar phenomenon [67]. However, as Chow [36] has pointed out, all these changes may simply reflect changes in arousal level rather than any specific learning mechanism.

In our laboratory, responses evoked at inferotemporal cortex by small illuminated patterns serving as discriminanda were recorded during the acquisition and performance of a number of visual discrimination tasks [67]. The situation was such that the monkey's state of arousal and attention and its fixation of the discriminanda were kept constant. The averaged responses from different pairs of electrodes on inferotemporal cortex were somewhat different. However, from a given pair of electrodes there was no discernible effect on the evoked response of (a) the meaning of the stimulus, (b) the physical pattern of the stimulus, (c) the correctness of the response following the stimulus, or (d) the stage of learning. These negative results do not prove that inferotemporal cortex has no role in visual learning. Rather, they may indicate that gross evoked responses, or the particular behavioral paradigm used, may not be the appropriate way of demonstrating any plastic functions that this tissue may have.

In summary, gross evoked responses to visual stimuli can be recorded from macroelectrodes on inferotemporal cortex, but they do not appear to be altered in the course of visual learning.

**Properties of Single Neurons in Inferotemporal Cortex**

For several years we have been studying the properties of single neurons in inferotemporal cortex of the macaque. These neurons respond to visual but not to
auditory, somesthetic or olfactory stimuli [72a, 77, 80, 81]. Thus the results of single neuron recording confirm the view, derived from lesion studies, that inferotemporal cortex is involved in visual functions.

In monkeys anesthetized with nitrous oxide and oxygen we were able to plot discrete receptive fields for most of the neurons responsive to light [72a, 77, 80]. The receptive fields always included the fovea. In contrast to receptive fields in the geniculostriate system, those of inferotemporal neurons were not invariably confined to the contralateral half-field: 56% of them extended well into both visual half-fields, 35% were confined to the contralateral half-visual field and 9% to the ipsilateral one. Most of the bilateral receptive fields had their centers in the contralateral half-field. The receptive fields were very large for cortical neurons: their median size was 418 degrees² with an interquartile range of 150–1410 degrees² (see Figs. 3–5).

Fig. 3. Coronal section in plane of electrode pass (arrow) in inferotemporal cortex showing approximate location of seven representative cells recorded on that pass and the size and retinal location of their receptive fields. The receptive fields recorded at increasing depth are shown clockwise starting from the top left. The cross in each receptive field figure represents the horizontal and vertical meridians of the visual field. The scale, in degrees of visual angle, is the same for all fields. The broken lines indicate that the field boundary extended to the edge of the tangent screen. The first, second, third and seventh receptive fields were plotted with various black stimuli, the others with various light stimuli. In each case, the stimuli used to define the field were the most adequate found. A: allocortex; Cd: caudate nucleus; CC: corpus callosum; GLD: dorsal lateral geniculate; H: hippocampus; Pul. I: inferior pulvinar; TA, TE, TF and TH: cytoarchitectonic areas of von Bonin and Bailey. [GROSS, ROCHA-MIRANDA, AND BENDER, unpublished data]

Perhaps the most salient characteristics of the neurons we studied were the heterogeneity of their properties. The strength or existence of response to a visual stimulus varied as a function of some or all of the following parameters of the
stimulus: direction and speed of movement, size and shape, contrast and wavelength.

Almost all the units responded more vigorously to a moving stimulus than a stationary one. For the great majority of the neurons a white, dark or colored slit about 1° in width was a more adequate stimulus than other rectangular or circular stimuli. Responses were almost always stronger when the movement of the slit was orthogonal to its long axis. About half of the units were sensitive to the direction of movement (Fig. 4). Most of these were “bidirectional”, i.e., they fired

![Diagram](image)

Fig. 4. Receptive field and responses of an inferotemporal neuron which showed “bidirectional sensitivity”. The histograms indicate frequency of firing of the unit as a function of retinal locus of a 1° x 70° white slit moving at 5°/sec in the direction indicated above each histogram. Each histogram was generated by seven sweeps of the stimulus. For the eight histograms, the vertical scale indicates number of neuron discharges and the horizontal scale degrees of visual angle; the middle of each horizontal scale (0°) represents the center of gaze. The receptive field of this unit is shown in the center of the array of histograms. Plus (+) in all parts of the figure indicates upper or right of the visual field; minus (-) indicates lower or left; UL, upper left; LR, lower right; LL, lower left; UR, upper right. The lower part of the figure shows the discharges of an isolated unit to a single sweep of the stimulus in the indicated direction on an expanded time scale. The lines below each trace correspond to the horizontal extent of the receptive field. The marker indicates 8°. The histograms and trace in which the arrow is shown on the left were generated from left to right whereas the converse was true where the arrow is shown on the right. See also legend to Fig. 3. [80]
equally well to both directions of movement in the optimal orientation of the stimulus and had null directions 90° to the optimal one, but some were “unidirectional”, i.e., they fired best to one direction of movement and had null directions 180° from the optimal one. Neurons were found that were color sensitive. Of these, neurons preferring red stimuli were the most common.

Fig. 5. Receptive field and responses of an inferotemporal neuron which did not respond differentially to the orientation or direction of movement of a 1° × 70° white slit. Each histogram was generated by 10 sweeps of the stimulus moving in the indicated direction at 6.7°/sec. Note that the response is vigorous when the slit enters the receptive field but declines before the slit reaches the opposite border. See also legends to Fig. 3 and 4. [80]

A few dozen of the neurons we studied responded best or only to a highly complex stimulus, such as the shadow of a monkey hand, the shadow of a hemostat forceps, a bullet shaped form and other specific patterns. The existence of these cells with highly specific trigger features raises several questions about the response properties of all our neurons. They suggest the possibility that we may never have found the appropriate trigger feature for many of our cells. Thus, a (more typical) neuron that responded best to a 1° × 5° red slit oriented at 45° within its receptive field may not have been “coding” this size, shape, color and orientation. Rather, its trigger feature might have been a far more specific, complex and perhaps meaningful stimulus that we never used and that happened to share some of the parameters of the stimulus we did use. Thus in searching for the adequate stimulus and
systematically varying the length, width, color, etc. of a slit we may have been like Kipling’s proverbial blind men examining an elephant. Another possibility suggested by the highly specific trigger features that we did find for a minority of cells is that adequacy of a stimulus for inferotemporal neurons may be a function of the meaning of a stimulus as well as of the physical parameters of the stimulus actually falling on the retina.

As first steps in directly testing the hypothesis (pp. 460–462) that inferotemporal cortex further process the outputs of the geniculostriate system we studied the effect of total unilateral-removal of striate cortex and of complete section of the corpus callosum and anterior commissure [72a]. According to this hypothesis, after a unilateral striate lesion, inferotemporal neurons should be responsive only to stimulation of the half-field contralateral to the lesion. Similarly, after the commissural section, inferotemporal neurons should be responsive only to stimulation of the half-field contralateral to the recording electrode. Both predictions were confirmed: all receptive fields were unilateral and in the predicted half-field in contrast to the normal animal in which over half the receptive fields were found to extend into both visual half-fields.

Although these results established the dependency of the visual responses of inferotemporal neurons on striate cortex they are in themselves compatible with a subcortical route (involving the pulvinar) from striate cortex to inferotemporal cortex as well as with the hypothesized striate-circumstriate-inferotemporal route (Fig. 2). Preliminary data indicate that partial pulvinar lesions do not eliminate the visual responsiveness of inferotemporal neurons although they modify it [72a]. Thus the cortico-cortical occipito-temporal route appears both necessary for the visual responsiveness of inferotemporal neurons and sufficient for at least some of the visual properties of these cells.

The hypothesis that inferotemporal cortex further processes outputs of the circumstriate belt provides an explanation for two prominent properties of inferotemporal units, viz., the invariable inclusion of the fovea in the receptive fields and the existence of bilateral and ipsilateral receptive fields. The inclusion of the fovea would derive from the fact that inferotemporal cortex receives a heavy projection from the portion of the circumstriate belt (“foveal prestriate cortex”) onto which the foveal representation in striate cortex projects [45, 210]. The ipsilateral and bilateral receptive fields would derive from the connections of the two circumstriate belts through the splenium of the corpus callosum [181] or the connections of the two inferotemporal cortices through the anterior commissure [64] or both connections.

How does inferotemporal cortex process the information it receives from the circumstriate belt? One possibility is that inferotemporal cortex is a further stage in the hierarchy of visual mechanisms shown by Hubel and Wiesel to extend from the retina through the geniculostriate system to the circumstriate belt [87–89]. The successive transformations of the visual input that Hubel and Wiesel have proposed occur in this system involve two chief principles. The first is increasing generalization across the retina: cells at higher levels can be driven by their adequate stimulus over wider regions of the retina. The second is increasing specificity of the adequate stimulus: orientation of a slit is not critical for ganglion or lateral geniculate cells but is critical for cortical cells; length of a slit
is critical for hypercomplex but not for simple or complex cortical cells. **Hubel**
and **Wiesel** suggest that convergence of outputs from cells at a lower level under-
lies these transformations.

Virtually all inferotemporal neurons appear to continue the first trend: their
receptive fields were much larger than those of complex and hypercomplex neurons
with fields in comparable retinal areas. Some inferotemporal neurons appear to
continue the second trend: they had more specific trigger features than have been
reported for complex or hypercomplex cells. Many cells, however, appeared to be
less sensitive to such stimulus parameters as length, width, and orientation than
cells in striate and prestriate cortex (e.g. Fig. 5). This apparent lack of specificity
may have been because these cells had complex and specific trigger features that
we never found. The existence of other cells in our sample with very complex
trigger features supports this possibility. The observation that three-dimensional
objects were far more adequate stimuli than two-dimensional patterns for some
neurons also suggests that a wider range of stimuli might have revealed a greater
stimulus specificity.

It is also possible that “stimulus adequacy” for some inferotemporal neurons
may depend on factors other than retinal stimulus. One such factor might be the
orientation of the animal relative to the stimulus. This possibility is suggested by
the tectofugal input to inferotemporal cortex since the tectum appears to be
involved in orientation functions (pp. 471–472). A second factor might be the mean-
ing of the stimulus for the animal. This possibility is suggested both 1) by the
dependence of the discrimination deficit that follows inferotemporal lesions
on prior experience and on the training situation and 2) by the incredible specificity
of the trigger feature of some of the inferotemporal neurons we studied. That is,
adequacy of a stimulus for inferotemporal neurons may, in part, depend on its
meaning: they may, in Konorski’s terms, be “gnostic units” [108].

In summary, the present results demonstrate that inferotemporal cortex
neurons receive specific and complex visual information. The visual responsiveness
of these neurons is dependent on striate cortex and they probably receive visual
information over a cortico-cortical route from striate cortex to the circumstriate
belt and then to inferotemporal cortex. Some of the properties of inferotemporal
neurons suggest that the processing of information in inferotemporal cortex con-
tinues the trends seen in the geniculostriate system. However, it is also possible
that new types of integration occur in inferotemporal cortex — that the activity
of inferotemporal units depends on more than the retinal stimulus. For example,
it may also depend on information received from the tectofugal system about the
location of the stimulus relative to the animal and on the significance of the
stimulus for the animal.

**Phylogeny of Inferotemporal Cortex**

In the first part of this section, inferotemporal cortex is related to major
features and trends in the development of vertebrate nervous systems. The next
section considers the cat, the species whose central visual mechanisms have been
most intensively studied, and points out that their organization is quite different
from that in the monkey and that there is no area in the cat yet described which is comparable to inferotemporal cortex in the monkey.

**Vertebrate Visual Systems and Inferotemporal Cortex**

Two major trends may be discerned in the evolution of vertebrate visual systems. The first is one of encephalization, the development of diencephalic and eventually telencephalic visual mechanisms. In lower vertebrates, the optic tectum appears to be the primary central visual mechanism. In reptiles and birds, the optic tectum projects to a large thalamic nucleus, nucleus rotundus, which certainly in birds and probably in reptiles is a visual structure [102, 157]. Indeed, in pigeons, the principal behavioral effect of its destruction is an impairment in visual discrimination performance [86]. In turn, at least in birds, nucleus rotundus has a telencephalic projection to the ectostriatum, which also has visual functions [157, 101, 156]. In a number of mammals, tectothalamic projections to nucleus lateralis posterior or the pulvinar have been described [134, 186, 164, 1, 100, 130]. This thalamic area is responsive to visual stimuli [e.g., 115, 71] and may be homologous to nucleus rotundus. In turn, lateralis posterior or the pulvinar (some authors reserve the latter term for primates) projects to the temporal lobe and circumstriate belt [31, 111]. Destruction of these cortical areas in the hedgehog [82], the tree shrew [174, 103] and, of course, the monkey, impairs visual discrimination learning. Thus, characteristic of birds and mammals is a tectal-thalamic-“association” cortex system having visual functions and whose cortical termination area is necessary for normal visual learning.

The other major trend is the development of a second visual system paralleling the phylogenetically older tectofugal one, namely the geniculostriate system involving an optic tract projection to the dorsal portion of the lateral geniculate body which, in turn, projects to striate cortex. As one ascends the vertebrate series and perhaps even within the mammalian order, an increasing proportion of optic tract fibers terminate in this newer system and the relative behavioral effects of its destruction seem to increase. These two visual systems converge in the circumstriate belt and temporal cortex and in the posterior thalamus of a variety of mammals from the opposum to the monkey. In the rhesus monkey, striate cortex projects retinotopically onto prestriate cortex, and prestriate cortex projects non-retinotopically onto inferotemporal cortex. The pulvinar projects to both inferotemporal and prestriate cortex, but whether it is retinotopically organized is not known. Finally, inferotemporal cortex projects back to both the pulvinar and the circumstriate belt (p. 460 and Fig. 2). Thus, anatomically, inferotemporal cortex is in a unique position to integrate the tectofugal and geniculofugal visual systems.

What are the relative roles of these two systems? **Schneider** [165, 166], on the basis of his comparison of the behavioral effects of tectal and striate cortex lesions in the golden hamster, has proposed that the tectum is involved in the location of and orientation toward visual stimuli, whereas the striate cortex is involved in the specific identification of objects. He further suggests that this association of localizing functions with the tectum and identifying functions with the geniculostriate system may be valid for other vertebrates besides the hamster. (Similar ideas have been proposed for other species by **Trevathan** [187],
Snyder and Diamond [174], and Held [85]. If this were true for the monkey, it would imply that the integration of localization and identification of visual stimuli might be a prime function of inferotemporal cortex. The demonstration by Hubel and Wiesel [87, 88, 89] of a hierarchy of pattern analysis mechanisms in the geniculate-striate cortex-circumstriate belt projection supports a stimulus-identifying function for this system in the macaque.

The functions of the primate tectum are more obscure. In the monkey, superior colliculus lesions have been reported to have no effect on the discrimination of visual pattern, total luminous flux, color, flicker, or the detection of movement [140, 5, 162]. However, they do appear to impair severely the discrimination of rate of movement [5]. The effects of inferotemporal or pulvinar lesions on such a discrimination problem have not been studied. Nor have there been any systematic studies of the ability of animals with damage to the colliculus, the pulvinar, or inferotemporal cortex to locate visual stimuli in relation to their body or head position. However, in the immediate postoperative period, monkeys with superior colliculus lesions have been observed to have considerable visual difficulties including an inability to localize visual stimuli. These dysfunctions appear to be transient except for a persistent reduction in spontaneous eye movements, although normal eye movements can still be elicited by visual stimuli [5, 46, 162, 142]. Additional but indirect evidence for a role of the superior colliculus in localizing functions comes from studies in which the entire striate cortex is removed: the ability to localize but not to identify visual stimuli remains intact [48, 49, 92, 91]. However, at least some of this residual function after total striate lesions may be due to the accessory optic system [140] or the pregeniculate body rather than to the superior colliculus or pulvinar.

In summary, inferotemporal cortex has two-way connections with both the phylogenetically older tectofugal visual system and the newer geniculostriate visual system. This puts inferotemporal cortex in a unique position to integrate the results of processing of visual information in these two systems. The geniculate-striate cortex-prestriate cortex hierarchy of processing mechanisms is involved in the analysis and synthesis of information about the retinal locus, size, shape, color, and movement of visual stimuli. By contrast, the functions of the superior colliculus and pulvinar are unclear. At least the superior colliculus may be involved with orientation toward visual stimuli.

Comparison of Inferotemporal Cortex with Non-Striate Cortical Visual Areas in the Cat

The visual system of the cat appears to be rather differently organized from that of the monkey, particularly at the cortical level. For example, whereas the sole cortical projection of the dorsal lateral geniculate body in the monkey is to Area 17 (striate cortex), in the cat it projects to Areas 17, 18, and 19, and to the lateral-middle suprasylvian gyrus, also known as Area Vss or the Clare-Bishop area [66, 70, 206].

There are two areas in the cat which might be compared to inferotemporal cortex in the monkey. The first is the lateral middle suprasylvian gyrus, which is a specifically visual area distant from striate cortex. However, unlike inferotemporal cortex, it (a) receives a direct projection from the lateral geniculate body, (b)
its removal does not seem to impair pattern discrimination learning [84, 83] and (c) although its neurons do have relatively large receptive fields, they do not invariably include the center of the retina, and their properties are more similar to those of complex and lower-order hypercomplex cells of Areas 17, 18 and 19 than to those of neurons in inferotemporal cortex [90, 208].

The second comparison that might be made is between inferotemporal cortex in the monkey and the “association areas” or “non-primary areas” of the cat (e.g., pericruciate, anterior marginal, anterior and posterior middle suprasylvian cortices). All these areas, however, unlike inferotemporal and superior temporal cortex, are polysensory; they respond to auditory and tactile stimuli as well as visual ones (see review by BUSER and BIGNALL [23]). (The anterior middle suprasylvian area does seem to have a “preponderance” of visual input; its units have large receptive fields and some of them require specific types of visual stimuli [53, 56, 57].) Thus, not only does the term “association cortex” convey nothing about function, it actually is used to refer to two fundamentally different types of cortex in the cat and monkey: one polysensory and the other unisensory.

In summary, no cortical areas in the cat have been described which are similar to inferotemporal cortex in the monkey in terms of anatomy, electrophysiology or the behavioral effects of ablation.

Synthesis and Speculation

In these days of domination of sensory physiology by single unit analysis, it is important to note that the guiding questions for the anatomy and physiology of inferotemporal cortex are still those raised by ablation experiments. Bilateral removal of inferotemporal cortex results in a severe deficit in learning new visual discrimination problems and in remembering ones learned prior to operation. This deficit is not only a purely visual one, but appears to be a purely gnostic one as well. Neither elementary sensory functions, nor any other aspect of visually guided behavior, except discrimination learning, is altered after inferotemporal lesions.

If monkeys with inferotemporal lesions do not have a sensory deficit, then what is wrong with them, or to reformulate the question, what are the “higher order” functions of inferotemporal cortex? Is the inferotemporal disorder one of visual “perception,” visual “integration,” visual “association,” or visual “memory,” or perhaps of some less medieval and more fashionable category like visual “information processing,” visual “selective attention,” visual “storage,” or visual “information retrieval”? It is far from obvious that the use of such terms advances our knowledge of this part of the brain in any fundamental fashion. Of course, these and similar terms are all we have, and they often have considerable heuristic or descriptive value. However, after over twenty years of dispute on the applicability of these and other logical (at best) categories to the inferotemporal syndrome, it now seems more profitable to concentrate on the anatomic and physiologic basis of the syndrome and on the functioning of intact inferotemporal cortex. Hopefully, such analyses will lead to more biological categories than those listed above or, perhaps, when successful, they will remove the need for such categories.

The integrity of the pathway from striate cortex to inferotemporal cortex by way of synapses in the circumstriate belt is necessary for normal discrimination learning. Furthermore, the responsiveness to visual stimuli of neurons in infero-
temporal cortex is dependent on striate cortex. Thus, inferotemporal cortex appears to be a processing station for visual input from the geniculostriate system. This “serial processing” model is further supported by the properties of the neurons in inferotemporal cortex. They have receptive fields and responsive properties that suggest their generation, at least in part, by converging input from neurons in the circumstriate belt.

Is inferotemporal cortex solely a mechanism for further analysis of visual input from prestriate cortex? In addition to the input it receives from the geniculostriate system, inferotemporal cortex receives a projection from the pulvinar which, in turn, receives fibers from the superior colliculus. Although nothing is known about the nature of the information transmitted along this pathway, it is clear that the superior colliculus processes visual information and that, at least in other species, the superior colliculus is involved in visual orientation mechanisms. Thus, it is conceivable that inferotemporal cortex receives, by this route, information about the location of visual stimuli in behavioral space.

Finally, there are several reasons why inferotemporal cortex may be involved in more than the further processing of geniculostriate output and perhaps tectal-pulvinar output. The severity of the effects of inferotemporal lesions on discrimination learning depends on several variables besides the physical nature of the discriminanda, such as the animal’s prior experience, the training procedure used, and the type of reinforcement. Furthermore, the impairment is one of rate of learning, not an absolute inability to distinguish stimuli. Thus, the activity of inferotemporal neurons may depend on more than the physical stimuli falling on the animal. Inferotemporal cortex may be involved in the storage of visual information or in interactions with structures that are concerned with mnemonic processes.

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