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New Dimensions in the Functions of the Basal Ganglia*

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INTRODUCTION

Research in psychopathology is distinguished by the fact that it partakes in three basic areas of observation and experimentation: clinical, behavioral, and biological. As a rule two avenues of investigation of psychopathological problems are open to us, each addressing the three basic elements. These avenues are the direct, which uses clinical material, and the indirect, which employs models. The nature of the distinction between direct clinical observation and model building in the biochemical aspects of psychopathological research is familiar, as attested by such phrases as *"in vivo"* and *"in vitro."* Less well understood is the distinction in the neurobehavioral and neurophysiological approaches to psychopathology. Especially lacking has been the detailing of the development of neurobehavioral and neurophysiological models of clinical problems. I want therefore to describe one program of research in which such models were developed and to issue a brief "progress report" on the state of that program.

Thirty years ago I began research aimed at studying the role of cerebral cortex in human clinical disorders. Along with Bucy (4), I had published observations of a case of localized facial sweating because of an oligodendroglioma situated in the precentral motor cortex. These observations led to the belief, contrary to ones then current, that the cortex, not the hypothalamus, might be the "head ganglion" of the autonomic nervous system. I was in neurosurgical practice with J. G. Lyerly and helped devise a "superior" approach to the frontal cortex in order to assure a more restricted procedure when lobotomy was performed for obsessions, compulsions, and depressions. The "standard" Freeman and Watts procedure was proving to have unwanted cognitive side effects, was shown to invade "Broca's area" (19), and was much in need of careful quantitative evaluation. But perhaps of more lasting significance, I was able to work with Karl Lashley at the Yerkes Laboratories for Primate Biology in an attempt to make animal *models* of the clinical disorders presumably produced by cerebral dysfunction. Such models would allow precise specification of lesions and quantitative testing of behavior over prolonged periods.

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A large surface of primate cortex was at the time "silent" to experimental manipulation. Lashley was making restricted resections of frontal, parietal, and temporal cortex with little apparent effect. I therefore took the opposite approach and made very large lesions of the frontal lobes, of the temporal lobes, and of the entire extent of the "association cortex" lying between the primary sensory projection areas. Because medial and basal forebrain structures such as cingulate cortex, precuneus, and hippocampus might be responsible for effects such as the Klüver-Bucy syndrome or the apraxias, the limbic portions of the cerebral mantle were excised in many of the experiments.

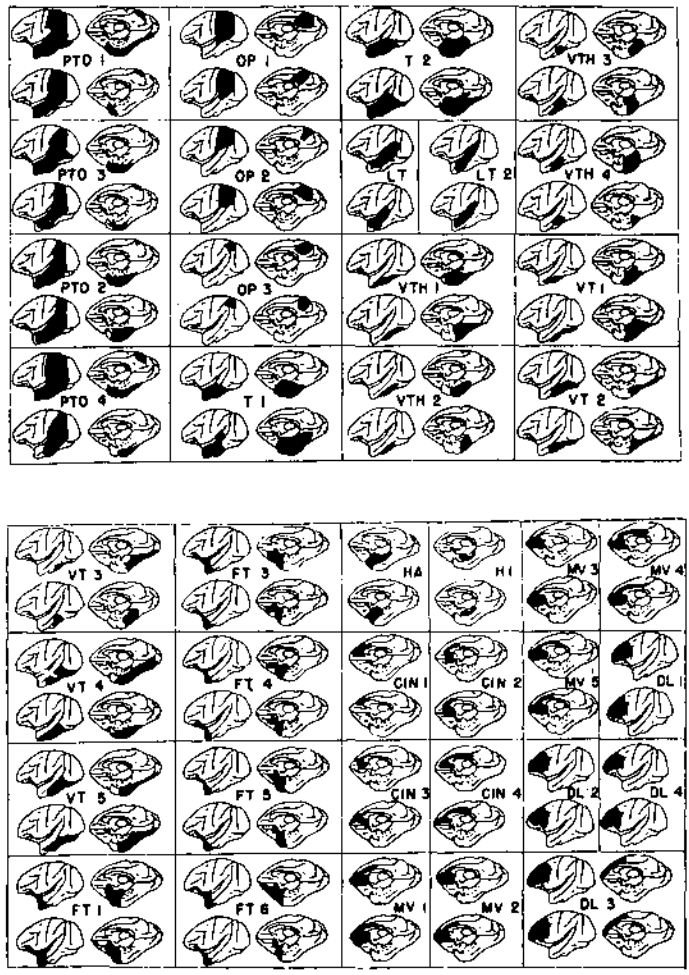

FIG. 1. Schematic reconstructions of the locus and extent of brain resections performed on 40 monkeys used for the multiple dissociation of brain lesion effects on behavior.

In order to assess the effects of this variety of large resections, investigators devised a battery of some 30 behavioral tests to study the monkeys. These tests ranged from the recording of general locomotor activity and performance of latch box manipulations, through a variety of sensory preference and discrimination tasks, to higher order delay and matching from sample procedures (Fig. 1).

A method was then devised (24) to relate the effects of the lesions to one or another of the tests. An arbitrary criterion was established for "failure" in a task: when postoperatively a monkey took more trials to master the task than he had needed preoperatively, a deficit was declared. Then all of the cortical resections that had produced such a "deficit" were spatially summed—as was the spatial extent of all of the resections that had produced "no deficit." The intercept of the two sums was then diagrammed by overlay and this intercept considered the "locus" responsible for the deficit. The conclusion was tested by restricting the next set of lesions to such loci and showing by multiple "double dissociations" that, in fact, the localization held up. By now approximately 1,500 monkeys have been studied in this fashion in my laboratory alone, and I feel we know a great deal about cerebral function in behavior as a result (Table 1, and Figs. 2, 3).

These initial studies that provided brain-behavior correlations were, of course, only the first steps in the program of research. What did a specific correlation mean? Two types of questions need to be answered. First, what is the *behavioral* significance of the sign that has become pathognomonic of the cerebral pathology? This is much like asking what the Babinski sign tells us about the behavioral development of a child. The second type of question deals with the *neural* mechanism responsible for the production of the sign. Thus we take the Babinski reflex to be an indicator of myelinization of the pyramidal motor system. What neural pathways are involved in the ability or disability to perform a visual discrimination?

As a final stage in the research, of course, the fruits of all of these studies

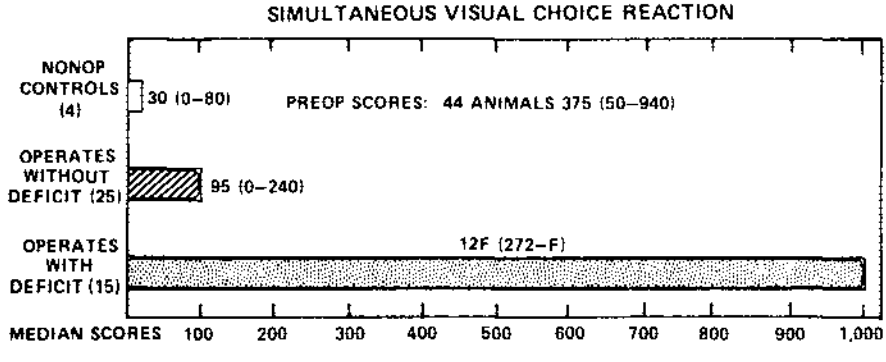

FIG. 2. Summary graph of Table 1.

TABLE 1. *Simultaneous visual choice reaction*

Operates Without Deficit			Operates With Deficit		
	Pre	Post		Pre	Post
OP_1	200	0	PTO_1	120	272
OP_2	220	0	PTO_2	325	F
OP_3	380	0	PTO_3	180	F
LT_1	390	190	PTO_4	120	450
LT_2	300	150	T_1	940	F
H_1	210	220	T_2	330	F
HA	350	240	VTH_1	320	F
FT_1	580	50	VTH_2	370	F
FT_3	50	0	VTH_3	280	F
FT_4	205	0	VTH_4	440	F
FT_5	300	200	VT_1	240	F
FT_6	250	100	VT_2	200	F
DL_1	160	140	VT_3	200	890
DL_2	540	150	VT_4	410	F
DL_3	300	240	VT_5	210	F
DL_4	120	100			
MV_1	110	0			
MV_2	150	10	Nonoperate Controls		
MV_3	290	130			
MV_4	230	10	C_1	790	80
MV_5	280	120	C_2	230	20
CIN_1	120	80	C_3	750	20
CIN_2	400	60	C_4	440	0
CIN_3	115	74			
CIN_4	240	140			

Table of "deficit" and "no-deficit" performances on the visual choice reaction, which is shown to be selectively impaired by inferotemporal cortical resections on the basis of these data (see Fig. 3).

performed on nonhuman primates must be reflected back to the clinic and the human condition.

DISCOVERY OF THE VISUAL FUNCTIONS OF THE INFEROTEMPORAL CORTEX

To illustrate this program of investigation, I will focus on one cortical area, that lying on the inferior surface of the temporal lobe. I will present some of the highlights that changed the course of research when they occurred and end in what might be considered a progress report on current endeavors.

The inferior convolution of the temporal lobe was discovered by the multiple dissociation technique to be responsible for deficits in visual discrimination performance (see review in ref. 24). At the animal level, this was a welcome discovery because it parcelled out one part of the Klüver-Bucy

TABLE 2. *Baboons: Postoperative visual discrimination scores. Scores are no. trials preceding 90 correct in 100*

Pre-op Median Subjects	350 ▽ △	100 □ ○	20 D L	0 R G
T 1	F	760	F	750
T 2	F	F	F	540
VTH 1	F	F	F	790
VTH 2	F	F	F	100
VTH 3	F	F	F	990
VTH 4	F	F	0	10
LT 1	190	20	0	0
LT 2	150	450	0	0

F = Failure in 1,000 trials (Underlined)__ = initial acquisition

Table showing deficits in a variety of visual choice procedures in monkeys with resections of inferotemporal cortex.

syndrome from the rest. Klüver had shown that monkeys with temporal lobectomies were, aside from hyperoral and hypersexual, also "psychically blind" – i.e., they suffered from a visual agnosia. In our early experiments we were able to establish not only that the visual portion of the syndrome could be separated from the rest [which was attributable to amygdala resections

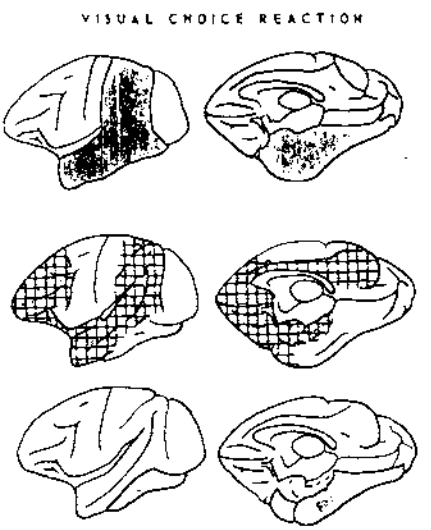

FIG. 3. Summary schematic reconstruction of the sums of the locus and extent of brain resections that produce the deficit (*top*) and those that do not (*middle*) in the visual choice procedure. Bottom diagrams show the intercept of these two sums and "localize" the effective lesion to the inferotemporal cortex.

TABLE 3. *Double dissociation between the effects of resections of parietal and of inferotemporal cortex*

	Preop. Learning	Preop. Retention	Postop. Retention	Postop. Learning
	SOMESTHETIC DISCRIMINATION			
P Group A	408	5	687	–
P Group B	–	–	–	821
IT Group A	410	54	19	–
IT Group B	–	–	–	439
	VISUAL DISCRIMINATION			
P Group A	–	–	–	331
P Group B	320	0	194	–
IT Group A	–	–	–	1000f
IT Group B	518	0	1000f	–

Parietal resections produce a somatosensory deficit but visual performance remains intact. Inferotemporal resection, conversely, leave somatosensory functions intact while impairing visual performances. The lesions producing these deficits did not invade the primary thalamocortical sensory systems.

(29)] but also that the visual agnosia was, in fact, visual and not global—e.g., not somatosensory, gustatory, or auditory (3), as shown in Tables 2 and 3.

On the clinical level, the finding that the inferotemporal cortex of monkeys is critical to visual discrimination performance was also important. First, visual symptoms associated with temporal lobe lesions had always been attributed at least in part to invasion of the optic radiations—even anteriorly placed lesions were assumed to produce their effect by interrupting Meyer's loop, a portion of the radiations assumed to sweep anterior to the temporal horn of the lateral ventricle in order to account for temporal lobe visual deficits. The existence of Meyer's loop was brought into question by the animal studies, and clinical neuropsychologists began to look seriously at the effects of temporal lobe lesions *per se* on visual performance and not as reflections of damage to the geniculostriate system. Thus Milner (20) was led to conclude that human temporal lobe cortex was in fact involved in making visual discriminations much as was monkey temporal cortex, but that in man the nonverbal visual functions were served by the right hemisphere. This was the first demonstration of hemispheric specialization rather than dominance.

WHAT IS A VISUAL AGNOSIA?

The question that immediately arose was how to distinguish the visual functions of the temporal cortex from those of the occipital geniculostriate system. Initially, a series of studies argued the issue in terms of changes in

visual field. Battersby (1) claimed that changes in the visual field did occur, although Mishkin and Pribram (22) failed to note any such changes. The argument was reminiscent of classic ones based on clinical observations [e.g., by von Monakov (40)] and more currently pursued by Bay (2). Battersby did not mean to suggest that spatial scotoma resulted from the temporal lobe lesions. Rather, his point was that some as yet unspecifiable change in the total visual field accounted for the discrimination difficulty. As we shall see, subsequent research has borne out this early intuition.

Meanwhile our research suggested that the discrimination deficit produced by inferotemporal cortex resections was more akin to a motor than to a sensory difficulty. For instance, we trained monkeys to discriminate between a tobacco tin and an ashtray. Although deficient, animals with inferotemporal cortex resections could learn this very easy visual discrimination. We then changed the situation from one in which the cues (tobacco tin and ashtray) were presented simultaneously for choice between them to a situation in which they appeared successively. Thus the tobacco tin signaled that one behavioral response—e.g., reaching toward it or going to a cup on the right—needed to be made, whereas the ashtray signaled that a different response—e.g., withholding reach or going to the left—would be rewarded. Monkeys with inferotemporal cortex resections were markedly

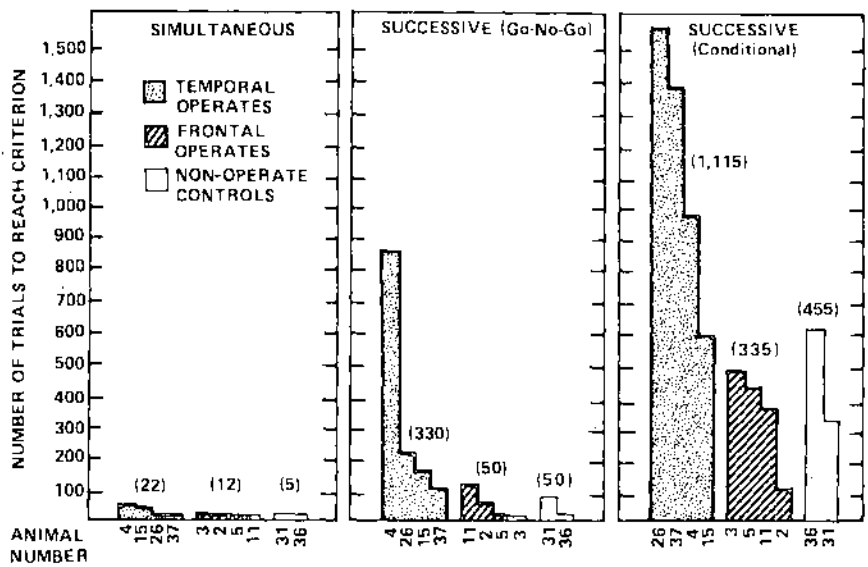

FIG. 4. Graph showing the relatively excellent performance of monkeys with resections of inferotemporal cortex on a simultaneous visual discrimination and markedly deficient performance on two forms of the successive version of the *same* task. These results suggest that the difficulty experienced by the lesioned monkeys is not so much an inability to distinguish cues as it is their inability to use the distinction.

deficient in performing the successive versions of the discriminations, and it could readily be shown that this was not because of any disability in distinguishing the cues, since the monkeys repeatedly performed well—even on the same day—in the simultaneous version of the task (28) (Fig. 4).

In another study (25) the monkeys were asked to choose among many objects. In this experiment the deficit of the animals with inferotemporal cortex lesions was shown to be dependent on a deficiency in the number of such objects they sampled. This finding was extended by Butter (6) to show that in the two-choice discrimination, the monkeys with the inferotemporal cortex lesions failed to sample as many features of the cues presented as the control monkeys. He showed this by eliminating first one and then additional features of cues that were being discriminated, and he demonstrated that the behavior of the monkeys with the lesions broke down much before that of their controls.

This result suggested that the sampling deficit shown by monkeys with inferotemporal cortex lesions need not be expressed by a motor response but could be "attentional." Evidence was therefore at hand to suggest a rapprochement between the view that inferotemporal cortex resections interfered either with visuomotor or visuosensory performance. Perhaps the difficulty was better characterized as "attentional." "Attention" is a central cognitive process that, if we could better understand it, might account for the agnosias produced by cortical lesions.

AN APPROACH TO THE NEUROPHYSIOLOGY OF SELECTIVE ATTENTION

Unfortunately, the term "attention" is used to cover a variety of psychophysiological processes such as a phasic response to input, a tonic level of readiness, or a more selective process involving choice [see, e.g., reviews by Kahneman (15) and by Pribram and McGuinness (27)]. When we assign the effects of inferotemporal cortex resections (on the number of features of cues sampled) to problems in attention, we need, therefore, to distinguish clearly which form of attention we mean. Sampling is dependent on selection and choice. Thus, selective attention involves the inferotemporal cortex.

We set out to investigate the relationship between inferotemporal cortex and selective attention by using a situation that would necessitate different central processes even though identical cues were presented to the eyes of the monkeys. This was made possible by using colored patterns—i.e., cues with several obvious features—and reinforcing one feature (e.g., the color) at one time and another feature (e.g., the pattern) at another. Thus the feature attended depends on the reinforcing contingencies operating at the time of testing (Fig. 5).

Rather than making lesions in the temporal lobe, we implanted electrodes

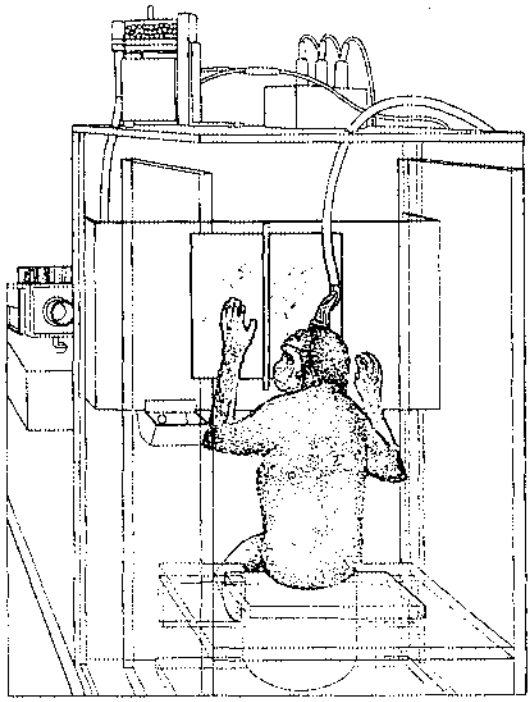

FIG. 5. View of a monkey performing a successive visual choice reaction while his brain electrical activity is being recorded.

and made records of the brain electrical activity evoked by the cue presentation (a brief flash of the colored pattern on a screen) and by the response (depression of either of two panels) of the monkey. A series of experiments using this technique (8,23,26,33) showed that the electrical activity of the inferior temporal cortex is distinctly different when the monkey attends the color and when he attends the pattern features of the cue. This difference becomes manifest approximately 50 to 100 msec before the monkey makes any overt behavioral response and is often, although not always, absent at the time of or shortly after the cue presentation. Overtraining predisposes to an early, cue-dependent difference in the brain electrical record, which then can become manifest in the occipital cortex as well (Fig. 6).

Another set of electrophysiological experiments related the activity of the inferotemporal cortex to attention. For reasons discussed below, we were investigating the effects of chronic electrical stimulation of inferotemporal cortex on recovery cycles within the geniculostriate visual system. After publishing our initial interesting results (36), we went through a 2-yr series of disappointing attempts at replication. Finally, we found that the electrical

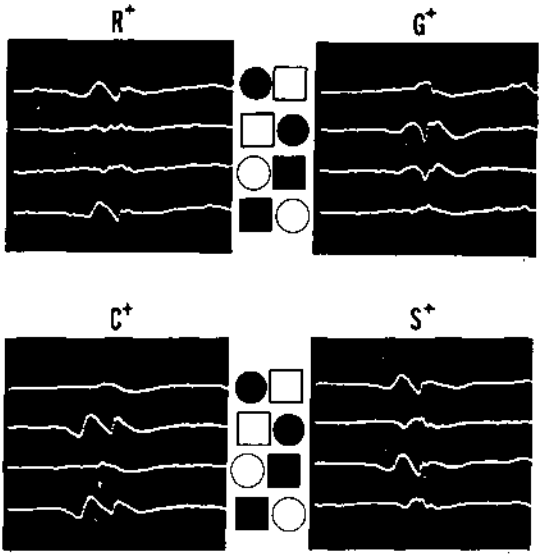

FIG. 6. Computerized averages of the electrical activity recorded from the inferotemporal cortex of a monkey solving a visual choice problem in a situation similar to that shown in Fig. 6. In this experiment, however, a simultaneous choice between colored circles (●) and stripes (□) is offered, and the monkey's response is shaped by reward contingencies; 500 msec of record is shown and the monkey's response occurs midway (at 250 msec) of the recording. Note that when colors (red R+; green G+) are rewarded the temporal lobe electrical activity reflects the position of the colors irrespective of the shape of the cues (i.e., records 1 and 4 and records 2 and 3 look alike). When the shape of the cues is rewarded (circles C+; stripes S+), the temporal lobe electrical activity reflects the position of these shapes irrespective of their colors (i.e., records 1 and 3 and records 2 and 4 now look alike).

stimulation of inferotemporal cortex interacted with the attention being paid spontaneously by the monkeys to one or another visual or auditory cue to produce a ceiling effect. When we brought the monkeys' attentional processes under strict control by eliminating distractors, the original finding was readily replicated (10).

When the results of the experiments described so far are summarized, a common thread can be discerned. Resections of the inferotemporal cortex produce visual discrimination deficits. These deficits are modality-specific and other modality-specific deficits are obtained from other restricted resections of posterior "association" cortex (e.g., audition from superior temporal cortex; taste from anterior temporal cortex; somatosensory signals from parietal cortex). The discrimination deficit is not due, however, to any disturbance in distinguishing cues. Rather, the deficit reflects a disordered selection process. Thus cognitive difficulties (e.g., tobacco tin signifies go to the right cup or key signifies lock) and attentional disturbances (color has been paying off) are produced.

THE PARADOX OF MODALITY-SPECIFIC OUTPUTS

Recall that this program of research can be analyzed in several stages. The first of these was correlational: a unique relationship between a cerebral locus and a behavioral sign was established. Next, the behavioral significance of that sign was explored. Thus the deficit in visual discrimination produced by inferotemporal cortex lesions was found to be owing to a disordered selection process limited to the visual mode and manifested in cognition and attention. We are now, therefore, ready to ask the third question: By what neural mechanism is the selection process organized?

The classic answer to this question, an answer still commonly held to be true, is that a hierarchy of abstractive steps characterizes the processing of "information" from the primary geniculostriate (or other sensory) system. These steps are thought to take place in progressively remote locations of the perisensory cortex until high-order abstractions are achieved in the association cortex [see, e.g., Luria (17) and Jones (14)]. For the most part these abstractive steps are assumed to rely exclusively on corticocortical connections with at best only minimal involvement of corticosubcortical pathways.

Unfortunately for these classic views, our laboratory experiments with animal models have time and time again produced data that are difficult to encompass in this fashion. Most critical has been the finding that extensive resections of the peristriate cortex fail to interfere, except temporarily, with visual discrimination performance (30) (Fig. 8).

This does not mean that such perisensory cortex resections have no effect.

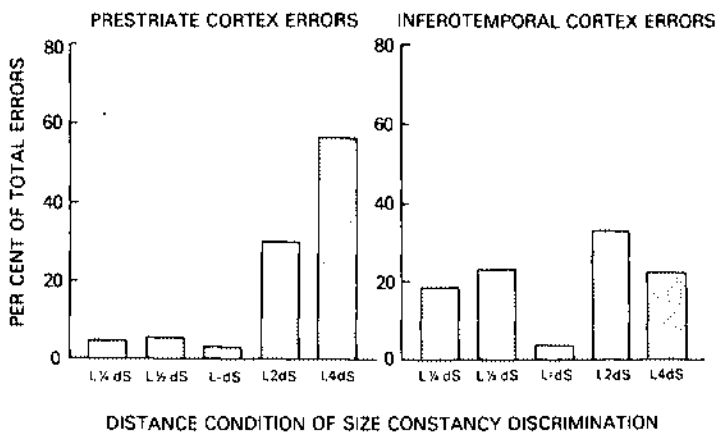

FIG. 7. Graph showing that after resections of prestriate cortex, monkeys rely on the retinal image size projected by a cue and ignore distances. ($L^1/_4dS$, $L^1/_2dS$, etc. are specific cue combinations.) Monkeys with resections of inferotemporal cortex do not adopt this "retinal-image-size" strategy—their failure is owing to a relatively random selection of cues.

On the contrary, peristriate lesions produce, for example, a monkey that is unable to display perceptual size constancy and instead responds to retinal image size (38). However, disordered size constancy is not necessarily a "step" involved in the selection process necessary to make visual discriminations. Rather, the deficit is distinct and can be clearly dissociated from that which characterizes lesions of the inferotemporal cortex (Fig. 7).

Recent anatomical studies have delineated three separate "visual systems." In addition to the geniculostriate, there is a collicular-peristriate and a pretectile-inferotemporal system (11,14). The thalamic connection of these extrageniculate systems is the pulvinar. One possibility for explaining the visual specificity of the functions of the inferotemporal cortex, therefore, is its pulvinar input. This possibility has been tested (7,21) and found wanting. Even extensive lesions of pulvinar fail to impair visual discrimination performance [unless cues are presented with a brief tachistoscopic flash (18)]. Thus the pulvinar input to the inferotemporal cortex is even less important than the peristriate input in explaining visual specificity.

The suggestion was therefore made (13) that perhaps the conjunction of inputs from peristriate and pulvinar becomes essential. In a study just completed, Ungerleider and I (39) have tested this alternative and found that even such drastic surgery does not produce a deficit comparable to that which follows inferotemporal cortex lesions.

In sharp contrast to these largely negative results, when pathways under-

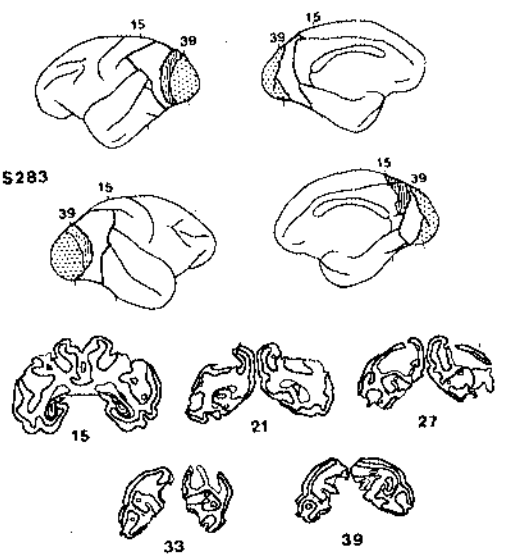

FIG. 8. Reconstruction of a resection of prestriate cortex that gives rise to a deficit in size constancy but does not severely and permanently impair other visual choices as does resection of inferotemporal cortex.

lying inferotemporal cortex are sectioned the full-blown deficit in visual discrimination behavior results. Further, the same result is obtained when lesions are made in the tail of the caudate nucleus and ventral putamen, the basal ganglia nearest the inferotemporal cortex (5,32).

Destructions were attempted in these nuclei of the basal ganglia because fiber tracts were traced to there from the inferotemporal cortex both neurohistologically (41) and electrophysiologically (31). The weight of this evidence thus suggests an important role for the basal ganglia in the selection process ascribed to the inferotemporal cortex (Fig. 9).

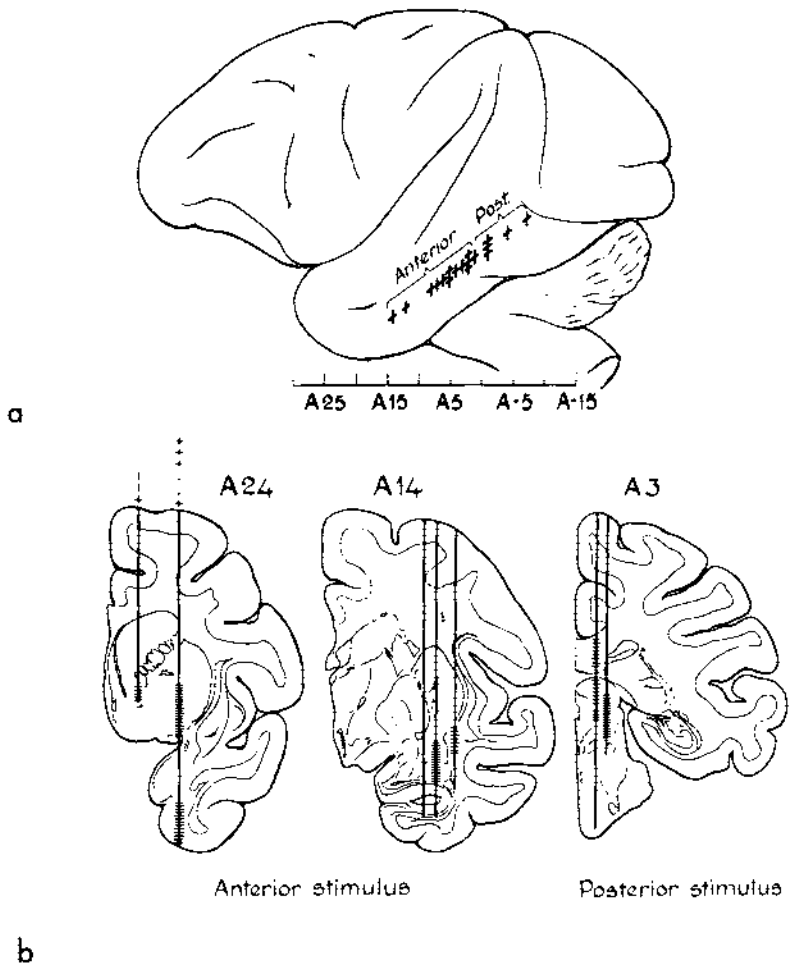

FIG. 9. a: Location of electrode placements used to stimulate inferotemporal cortex in order to trace subcortical efferent pathways. **b:** Location of responses evoked from electrical stimulations of inferotemporal cortex. Note heavy involvement of basal ganglia (especially putamen).

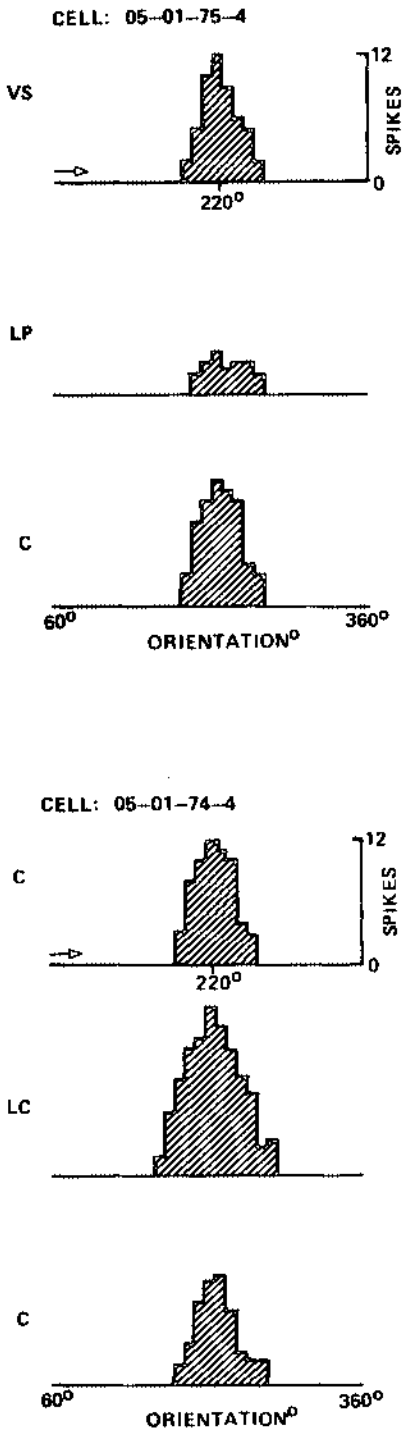

FIG. 10. a: Poststimulus histogram showing a tuning curve of a receptive field sensitivity to a line orientation at 220°. Recording is from a single unit in the visual cortex (of a cat). VS is the initial response of the unit, LP shows the effects of electrical stimulation of the putamen, and C shows the control histogram obtained after cessation of the electrical stimulation. **b**: Same as **a** except that electrical stimulation is now of the caudate nucleus. Note that the effect is opposite that obtained when putamen is stimulated.

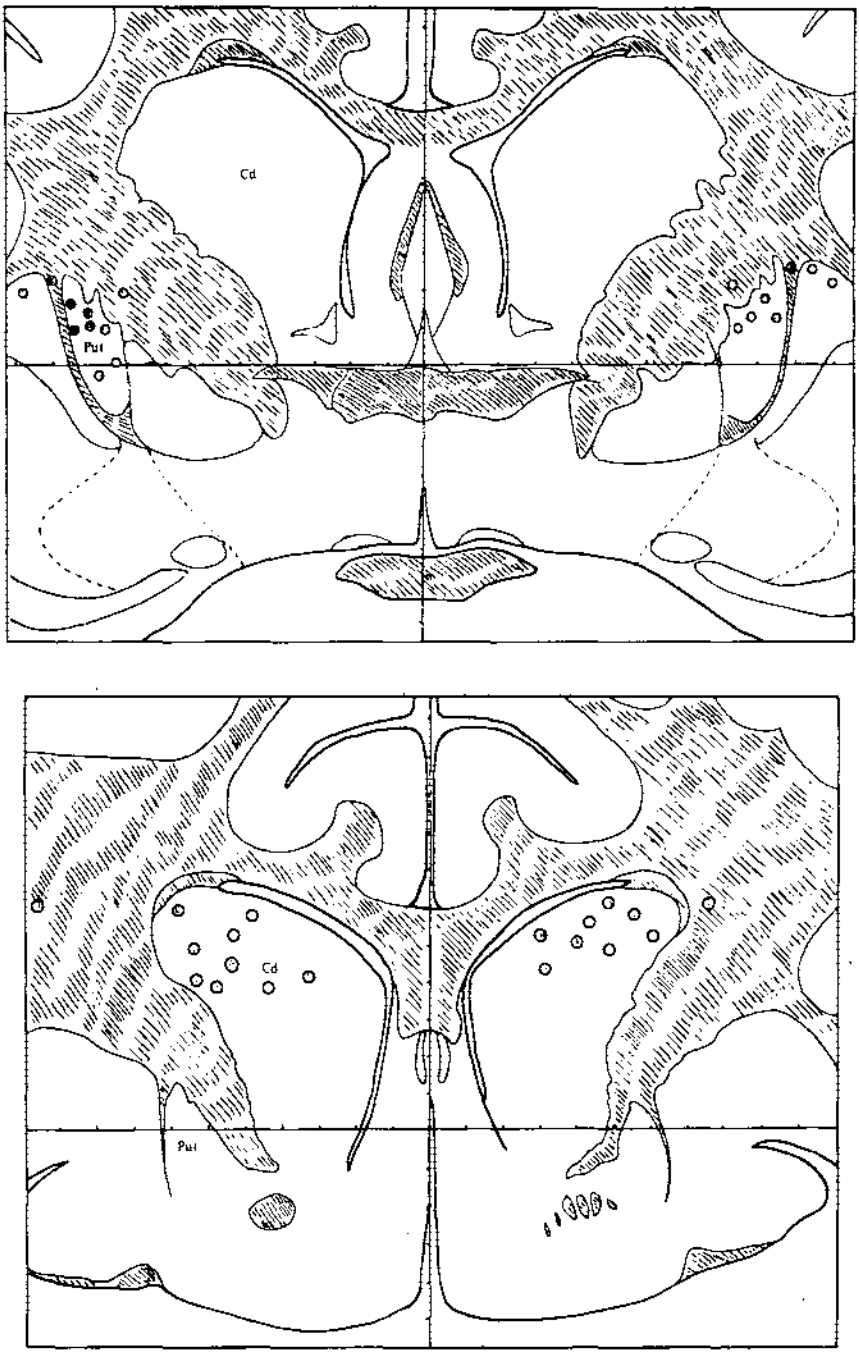

FIG. 11. a,b: Location of the electrodes used to stimulate the basal ganglia to obtain the changes in the visual receptive fields shown in Fig. 12. Cd, caudate; Put, putamen.

To complete the circuit, there must be connections from the basal ganglia to the geniculostriate visual system. (Connections to extrageniculate visual pathways would not help since these have already been ruled out as critical.) We have considerable indirect evidence that the basal ganglia are capable of influencing visual processing. Most recently we have electrically stimulated basal ganglia to elicit changes in visual receptive fields recorded from cells in the lateral geniculate nucleus (35) and the striate cortex (16). These changes are similar to those produced by changes in the inferotemporal cortex (37). (See Figs. 10 and 11.)

By what neuroanatomical pathways can the basal ganglia influence visual processes? We do not yet know but are actively searching, using the new autoradiographic techniques. Two hypotheses are of special interest. One concerns a thalamic "gate," the other involves the substantia nigra.

The thalamic gating of sensory input can readily occur through excitation of the thalamic reticular nucleus. Skinner and Yingling (34,42,43) have shown that such excitation modifies unit activity in primary sensory thalamic nuclei. Further, activity in the thalamic reticular nucleus is itself under the control of at least two systems. One reaches it through the median forebrain bundle from the mesencephalic reticular core. The other pathway constitutes a major portion of the thalamic peduncle. What is unknown is whether there are major basal ganglia contributions to this peduncle. Electrophysiological evidence has suggested that there are (9). Now anatomical verification is being sought.

Another major alternative is a route through the substantia nigra. When I recently attempted to trace the connections from inferotemporal cortex by means of autoradiography, my preparations revealed an odd artifact. Radioactive granules appeared on the surface of most of the cells of the substantia nigra bilaterally. This was true for both short time course (24 to 72 hr) proline-injected brains and long time course (20 days) leucine material. Such radioactive granules were absent from the nigra when frontal or motor cortex was injected. The granules looked much more like those obtained in retrograde preparations, such as when horseradish peroxidase is used, than the usual antegrade slides.

Thus I end this chapter with a puzzle, perhaps even an artifactious one. Nonetheless, the finding, if it proves to be real, may be important because Graybiel (12) has just established (using autoradiography) a point-to-point connectivity between substantia nigra and the superior colliculus. We cannot therefore as yet ignore a possible connection between inferotemporal cortex and the nigra.

CONCLUSION

By whatever route, the importance of the basal ganglia in processing sensory input (as well as motor function) is becoming evident. An intimate

functional relationship between association cortex and basal ganglia is established and has been traced here with respect to the inferotemporal cortex, the tail of the caudate nucleus, and the ventral portion of the putamen. These structures in turn have been shown to influence receptive field properties in the primary sensory system. We now need to trace the pathways by which these influences are mediated and to clarify the precise nature of the changes.

When answers to these questions are available, perhaps we can begin to understand why patients with lesions in homologous regions of cortex suffer agnosias and pay such a high attentional price in making even the simplest selective discriminations.

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