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Chapter IX

LOOKING TO SEE: SOME EXPERIMENTS ON THE BRAIN MECHANISMS OF ATTENTION IN PERCEPTION[1]

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...a survey of the phenomena of learning suggests that other variations in addition to repetitive facilitation must be taken into account as important factors in determining the rate of ...discrimination learning. One of those variables is the ease with which figural organization can be imposed upon physically independent items in the stimulus.

KARL S. LASHLEY
The Neuropsychology of Lashley, p. 430

The brain's isocortex may conveniently be divided into 1) those "sensory and motor" areas which rather directly receive an input from, and send an output to, peripheral structures, and 2) others more intrinsic and usually called "association," which do not. The chapters that precede this one have documented some of the relationships between perception and the sensory areas. My purpose here is to discuss the functions of these other more intrinsic cortical systems (fig. IX.1).

THE INTRINSIC PROCESSING CORTEX

Clinically a variety of amnestic syndromes are observed when these intrinsic processing systems are disrupted. Ordinarily the syndromes have been viewed as disturbances in the "integrative" or "associative" functions of the brain. Recent research, however, has developed data which cannot easily be understood in this fashion. Rather, it seems that mnemic functions may depend as much on recoding as on associative storage (24). This research has developed an important innovation in views: the associative view had made of the intrinsic processing systems relatively passive receptacles of information relayed from the sensory areas; the coding view is derived from facts which indicate that the intrinsic processing cortex actively regulates ongoing processes within the sensory systems. Let me review this evidence.

The results of the first series of experiments which were undertaken showed that the mnemic function could be subdivided. Essentially the posterior part of the intrinsic processing (the "association") cortex was found to be involved in recognition (2); the frontal part, in recall (29).

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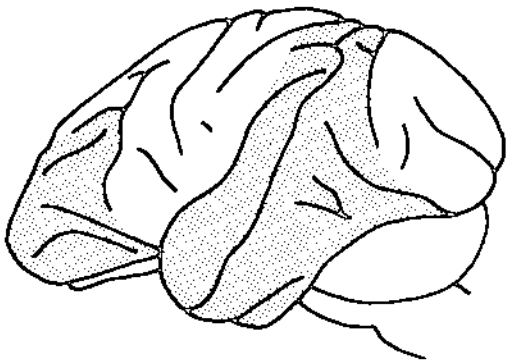

Fig. IX.1. Outline of the monkey brain with central processing cortex stippled. Note that there is a frontal and a posterior division.

The cortex serving recognition memory was in turn found to be divisible according to sense modality: the posterior parietal cortex functions selectively in somesthesis (20, 40); the anterior temporal cortex in gustation (1); the middle temporal cortex in audition (7, 39); and the inferior temporal cortex in vision (5, 16, 18, 22).

These results were gratifying but a surprising anatomical fact became evident almost immediately. The intrinsic processing areas were not located where we had expected them to be, *i.e.*, adjacent to the sensory cortex of the modality concerned. Rather, a considerable span separated the primary sensory system and its associated processor.

The importance of this anatomical separation was brought home to us by yet another experimental result. Implicit in finding the intrinsic processing cortex where we did, was the fact that one could with impunity remove the cortex between the sensory receiving area and associated intrinsic processor. More and more extensive removals were made to test the reliability of this fact (4) and in a recent study we were able to take this type of experiment to its logical extreme: we radically disconnected the striate from the inferior temporal cortex by extensively removing all of the peristriate cortex (fig. IX.2). Despite the massive removals which often inadvertently invaded the primary visual system, visual recognition of patterns (discrimination of the numerals 3 from 8) remained intact (31).

These results alone, were there no others, would call into question the ordinary views of the functions of the intrinsic processing cortex. But there is more. Intersensory associations do not seem to be effected in these locations (8, 9, 38); the evidence in monkey, at least, is that such associations take place within the primary sensory systems *per se* (30, 35). Nor is the idea tenable that the intrinsic processing cortex becomes in-

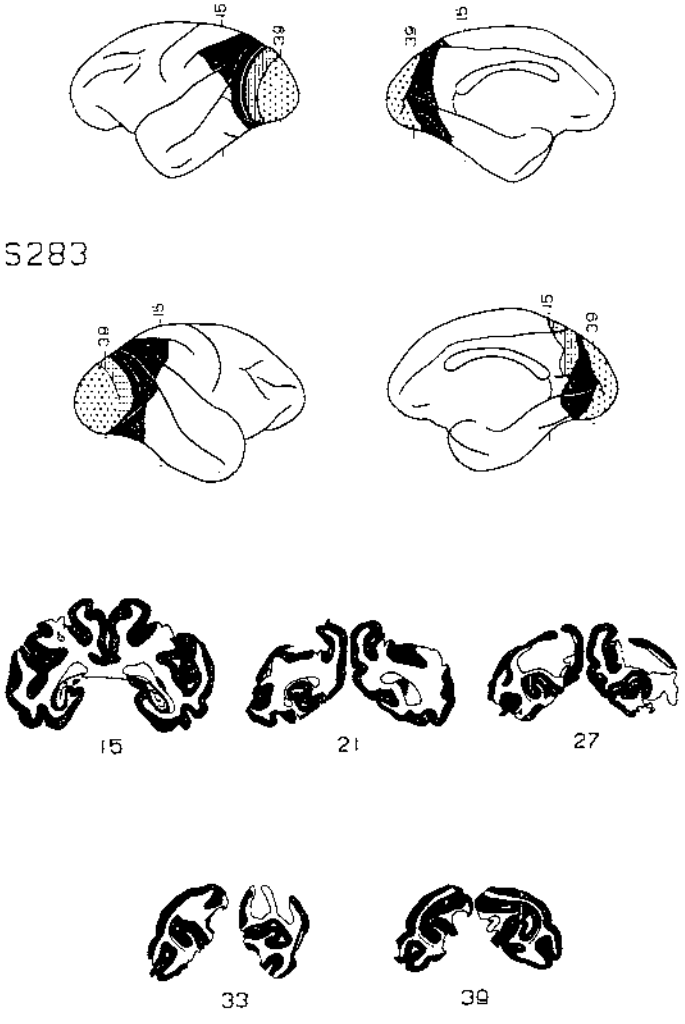

Fig. IX.2. Reconstruction of prestriate lesion in subject 283.

formed by way of a series of corticocortical relays, each of which extracts a progressively higher abstraction from the input. This does not mean that such abstracting relays do not exist: I want to emphasize only that, even if they do, they cannot at present account for the data in hand.

Let me illustrate. We thought that perhaps a corticocortical relay route might be involved in setting down the memory trace even if, once established, retrieval during recognition could be accomplished by some other mechanism. We therefore prepared groups of monkeys by crosshatching the inferior temporal cortex. We fully expected the crosshatches

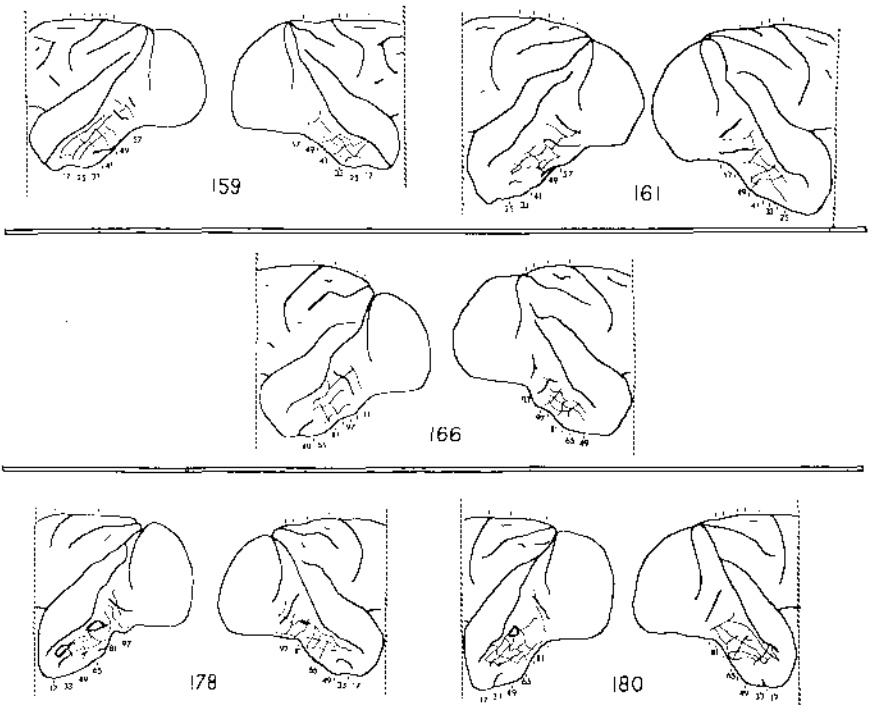

Fig. IX.3. Reconstructions of the crosshatch lesions of subjects 159, 161, 166 (original learning) and 178 (retention). *Fine lines* indicate the lesions.

to impair the learning of a pattern discrimination even if learned recognition remained intact (fig. IX.3). However, if anything, the crosshatched group learned the task more rapidly than did the controls. By contrast, undercutting drastically disrupted both learning and recognition performance (27) (fig. IX.4).

The possibility that the undercuts disconnected U fibers connecting the inferior temporal with the striate cortex must be considered. But, as far as is known today, all corticocortical connections of the striate visual area relay within the peristriate cortex which, as already noted, can be totally removed without impairing recognition. This leaves as the most likely explanation of the research results, the view that corticosubcortical rather than corticocortical connections are important to the functioning of the intrinsic processing cortex.

Corticosubcortical connections can work in two directions. There is evidence that sensory specific signals reach the central processing cortex (12, 37). As yet, however, we do not know how complex these signals are or by what pathways they arrive. The best guess at the moment, based on the evidence available, is that the signals are relatively simple and

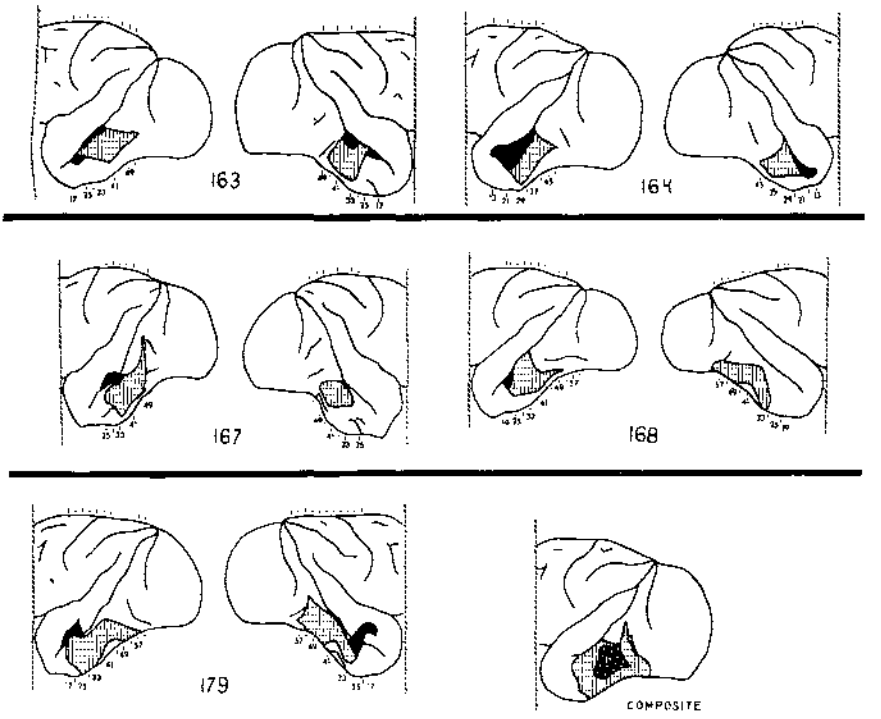

Fig. IX.4. Reconstructions of the undercut lesions of subjects 163, 164, 167, 168 (original learning) and 179 (retention). *Black* indicates superficial cortical damage; *stripes* indicate the deep lesion.

that they come by way of the intrinsic nuclei of the thalamus. Work is underway in several laboratories to clarify this important point.

Our own efforts have been directed largely to investigations of the corticofugal, the efferent, connections from the intrinsic processing cortex. The reasons for this concern stem from the results of analysis of the impairment of recognition produced when the intrinsic processing cortex is damaged.

DISCRIMINATION, DETECTION AND IDENTIFICATION

For the most part, the impairment produced is proportional to the difficulty of the recognition task: difficulty is measured by the number of trials taken by normal monkeys to master the problem. Task difficulty can be manipulated in a variety of ways: for instance, cue differences can be minimized and the impairment proportionally made worse or better (17). But this is not the whole story. Under certain circumstances the cues can be left identical but the responses demanded for recognition altered. A monkey with an inferior temporal lobe lesion may be able

to discriminate between an ashtray and a tobacco tin when they are presented to him simultaneously but, when he has to go to a cup on the right when the ashtray is presented and to a cup on the left when the tobacco tin is before him, he may fail miserably (28). This experiment, especially, made it seem that the impairment was related to the complexity of the entire task and not only the differences among cues, a conclusion supported by an earlier observation. I had noticed that, despite grave difficulties in problem solving, monkeys with lesions of the intrinsic processing cortex could track perfectly. They would snatch gnats out of the air within minutes of failing a + versus [ ] discrimination.

If indeed the impairment were related to the complexity of the task, this could be directly assayed by altering the number of alternatives among which choice has to be made (21). These alternatives can be specified by the number of separate cues or, perhaps more interestingly, among the alternative distinctive features which comprise a single cue (3). The results of these experiments confirmed the hypothesis that the deficit produced by lesions of the central processing cortex are directly related to the complexity of the task and of the number of distinctive features which identify a cue. In fact the results were such that it became clear that the process depends on the ability of the intact, normal subject to handle *more* information at any moment than his lesioned counterpart—more alternatives are able to be kept in mind, as it were. This makes it unlikely that the normal identification process is abstractive in the sense that some features are singled out to the exclusion of others, *i.e.*, that a progressive discard of information is operative. At the purely behavioral level also, the evidence is that discrimination learning (identification) is based largely on progressive differentiation (11), not on associative abstraction.

Several important neurobehavioral experiments remain to be done to clarify the steps involved in the identification process. The question is as yet unanswered as to whether there is a central mechanism that simply extracts the information "same or different" independent of the more complex identification mechanisms. A further question concerns the influence of complexity *per se:* does it interfere with the detectability of the cues or does complexity primarily affect the way in which the organism responds to the cues? These experiments are now underway. If preliminary results are available by the time this volume goes to press, a footnote describing them will be appended.

A CORTICOFUGAL SENSORY SYSTEM

An active identification process of the kind demanded by the above results implies an operation on input. This operation could hardly be

performed locally within the intrinsic processing cortex; it is more likely to be effected by operations which this cortex might exert efferently on the primary sensory system. The quantitative relationship between difficulty of task and amount of impairment, the fact that under some circumstances, *e.g.*, tracking, recognition seems not at all affected by lesions of the intrinsic processor, suggests that the operations critical for recognition are not performed in the intrinsic processing cortex *per se*. It seems, instead, that the operations performed by the intrinsic processing cortex allow recognition to take place in the face of circumstances that would in the absence of this cortex defeat the mechanism. Thus it becomes likely that an important aspect of the mechanism depends on corticofugal, efferent influences which the intrinsic processing cortex could exert on the associated sensory system.

We therefore undertook a series of experiments to demonstrate whether such efferent influences exist, the pathways they utilize and something of the functions exercised by their operation.

The existence of efferent influences exerted by the intrinsic processing cortex on the input mechanism is most dramatically shown in experiments in which the visual receptive fields of units in the optic nerve and lateral geniculate nucleus are changed by electrical stimulation of the central processing cortex (34) (fig. IX.5). The possible pathways utilized by these influences have been mapped (19, 32). Especially important are connections to the colliculi, to the intrinsic nuclei of the thalamus and to the putamen. The fact that two of these pathways involve structures thought to be primarily motor is consonant with the conclusion derived from the neurobehavioral analysis that these intrinsic processing systems provide an actuating mechanism necessary to making identifications.

ATTENTION

Further specifications of this mechanism come from experiments in which recovery cycles in the sensory systems were manipulated by making simulations or excisions of the intrinsic processing cortex (6, 33, 34). These experiments were performed on unanesthetized subjects and recovery speeded or slowed depending on the manipulation performed (fig. IX.6). Electrical stimulation of the sensory specific intrinsic processing cortex speeds recovery; stimulation of the frontal cortex retards the function.

The interpretation of these results comes by way of yet another set of experiments. These make use of the fact that a potential can be evoked in the striate cortex by stimulation of the lateral geniculate nucleus in the awake monkey. The amplitude of this "probe" potential can be varied by engaging the monkey's attention. Thus, when the subject sits in an enclosed box, the potential evoked by a particular stimulus

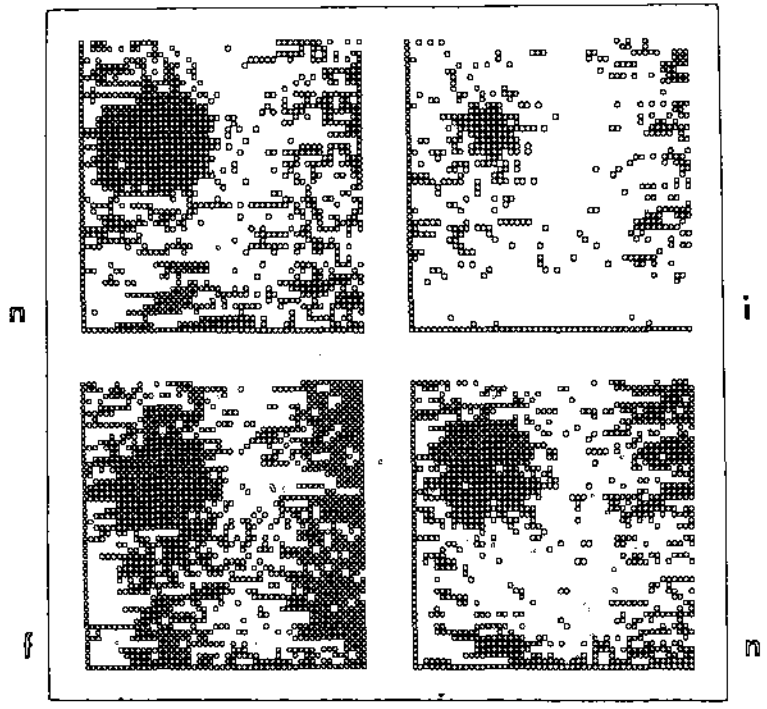

Fig. IX.5. Receptive field maps from a lateral geniculate unit, *n, top left:* control; *i:* mapped while inferotemporal cortex was being stimulated; *f:* mapped during frontal cortex stimulation; *n, bottom right:* final control. A third control was taken between the i and the f maps and was not included because it was not significantly different from the first and the last. Note that inferotemporal stimulation decreases the size of the "on" center; frontal cortex stimulation, while not really changing the circular part of the receptive field, brings out another region below it. The level of activity shown is 3 standard deviations above the normal background for this unit.

intensity is relatively small; when the box is opened and the monkey looks about, the amplitude of the potential evoked by the same stimulus intensity is much larger. Using the size of the probe-evoked response as an indicator of attention, we found that the changes in recovery cycle produced by stimulation of the sensory specific processing cortex could be obtained only when the monkey was *not* attending (10). Attention and the excitation of the intrinsic processing cortex seemed, as it were, in competition for control of the amplitude of the probe response. We concluded, therefore, that the intrinsic processing cortex effects identifications by activating an attention process (fig. IX.7).

The term attention is used in this volume with a variety of meanings and for each meaning a neural mechanism can be and has been proposed.

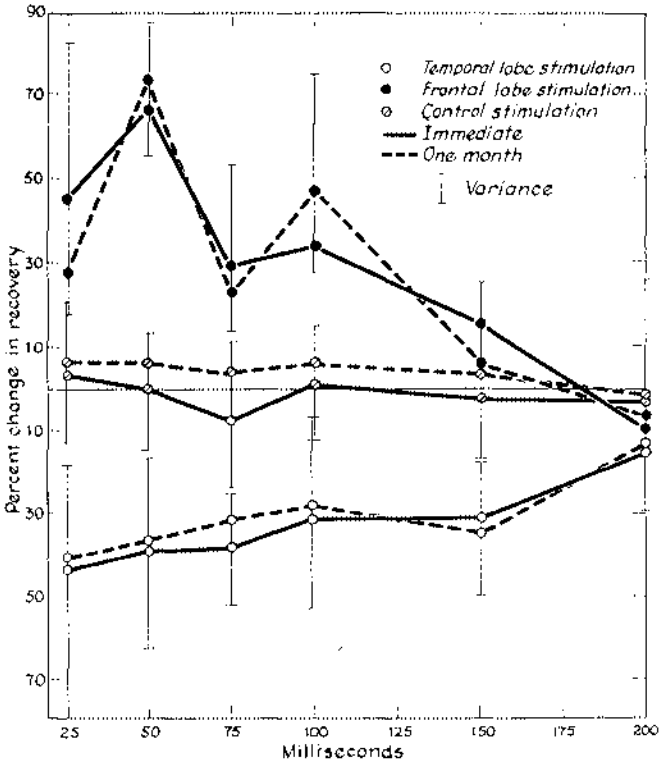

Fig. IX.6. The change in recovery of a response to the second of a pair of flashes compated with prestimulation recovery function. Control stimulations were performed on the parietal cortex. Records were made immediately after the onset of stimulation and weekly for several months. The response *curves* obtained immediately after onset and after 1 month are presented. *Vertical bars* represent variability of the records obtained in each group of four monkeys.

Attention can mean vigilance, a monitoring of internal and external events (14, 15). As reviewed elsewhere (25), the limbic forebrain seems especially adapted to such monitoring processes. Attention can also mean a focusing down, concentrating, on one or another aspect of a situation to the neglect of others. We have good reason to believe that the frontal part of the intrinsic processing cortex is directly concerned in such a mechanism of "concentration." Not only do neurobehavioral data show the increased distractibility, the stimulus binding, that occurs when the frontal cortex is damaged (26, 23). Also, the changes in recovery within the sensory systems produced by frontal stimulation indicate that the sensory channel becomes synchronized, allowing more of it to be given over to a particular item being processed at any moment.

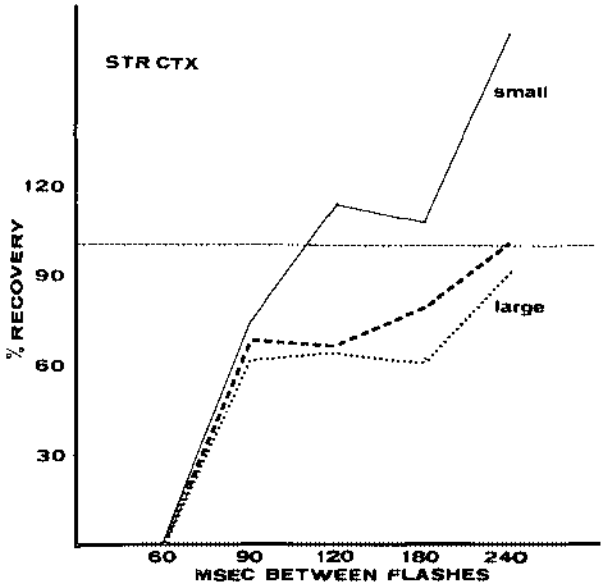

Fig. IX.7. Comparison of flash recovery functions obtained when the probe stimulation of the lateral geniculate nucleus results in "small" (*solid line*) or "large" (*dotted line*) striate cortex response. Control without probe stimulation is indicated by *dashed line*.

Finally, attention can mean the opening up to the variety and richness inherent in every situation—the active idling mechanism used by the psychiatrist's third ear to search, sample and identify incongruities and similarities appearing before him. It is this form of attention which, on the basis of the evidence presented, seems to be enhanced by the operation of the posterior part of the intrinsic processing cortex. The recovery cycle data bear out this conclusion: desynchronization of the sensory channel occurs when this cortex is stimulated; *i.e.*, a greater number of items can be processed at any moment (33). It is as if the sensory systems were fitted with a zoom lens: the frontal cortex provides a long focus which enlarges the item of interest and reduces depth of field; conversely, the posterior processing cortex provides a short focus which allows a much larger field to be sharply imaged. The effects of having the zoom stuck in the long or short focus position due to damage of the processing cortex should show up in differences in distribution of eye movements—differences which are now measurable thanks to the advent of the eye camera. Bagshaw and I are now engaged with Mackworth (see Chapter XIV) in studies to determine the nature of such differences.

What then are the conclusions relevant to perception to be derived

from these studies? The ordinary perceptual operations necessary to tracking and responding to the presence of stimulus characteristics remain intact when the intrinsic processing cortex is damaged. Such damage, however, does alter an active attentive process effected through efferent, corticofugal influences on the sensory systems. In the absence of the frontal cortex the primate becomes overly distractible and stimulus bound. When the posterior part of the central processing cortex is removed the opposite occurs: a paucity of information processing results. This paucity is manifest in restrictions on the number of cues sampled. It seems reasonable to conclude from these data that the operation of the intrinsic processing cortex ordinarily enriches perception.

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