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THE ORGANIZATION OF MEMORY IN NON-HUMAN PRIMATE MODEL SYSTEMS

Karl H. Pribram

Neuropsychology Laboratories
Departments of Psychology and of
Psychiatry and the Behavioral Sciences
Stanford University
Stanford, CA 94305

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INTRODUCTION

The Problems:

It is scarcely more than three decades since Lashley, in "The Search for the Engram" continuing the theme of his 1929 "Brain Mechanisms and Intelligence" declared his failure to locate the memory trace. By contrast, today the evidence from carefully performed clinical neuropsychological studies (which continue a 19th century tradition) provide a plethora of localizable disturbances of memory processes. However, interpretation of the data from these studies often remains difficult because lesions in humans do not carve the brain according to functionally operating anatomical systems. Nor do these studies completely contradict Lashley: deficits are not limited to specific memory traces (e.g. recognition of grandmother) but involve classes of memory processes. We are thus faced with the problem of reconciling the nature of engram storage with the nature of memory processing.

Further, the attempt to characterize and classify processes has served up series of dichotomies which vary from investigator to investigator. What is the relationship between procedural and referential processes? Between episodic and working memory? And between all of these and others such as semantic processing? There is thus at present no agreement as to the relationship among dichotomous formulations - only a monumental tower of Babel to confuse those not directly involved in the research.

Some of these problems can be addressed by studying non-human primate models. There is a relevant body of such evidence which is currently ignored in studies made on human neuropsychological patients. In this chapter I shall review this evidence which suggests that the various

aspects of memory can be arranged hierarchically according to the brain systems which have been identified to be involved. The specifics of the hierarchy proposed will most likely be subject to changes as new evidence acrues. However, I will maintain that the complexities of memory will not be understood until the relationships among them and to brain systems becomes clarified.

At least seven different memory mechanisms can be identified. At base these involve perceptual and motor skills on one hand and the episodic processing of novel (registration) and of familiar (extinction) events on the other. Skills are enhanced by search and sampling procedures to form referential processes. Episodic processes become sensitive to the spatiotemporal structure which describes the context within which the episodes occur and allows transfer of training in one context to another. In turn referential and contextual processes interact to produce declarative language.

Each of the nodes of the hierarchy has a forebrain system identified with it. Thus perceptual skills involve the primary sensory systems; motor skills, the primary motor systems. Processing novelty involves systems converging on the amygdala; processing the familiar, those converging on the hippocampus. Search and sampling are disturbed by resections of the posterior intrinsic, probabilistic programming by resections of the far frontal cortex. The methods and data from which these conclusions stem are described below.

[Figure 1]

The Multiple-Dissociation Technique

The experimental analysis of subhuman primate psychosurgical preparations has uncovered a host of memory disturbances. The initial technique by which these brain-behavior relationships were established is called the

method of multiple dissociation (Pribram, 1954), akin to what Teuber[4] named the method of "double dissociation" of signs of brain trauma in humans.[5] The multiple dissociation technique depends on classifying the behavioral deficit produced by cortical ablations into yes and no instances on the basis of some arbitrarily chosen criterion; then plotting on a brain map the total extent of tissue associated with each of the categoires ablated:deficit and not ablated:no deficit; and finally finding the intersept of those two areas (essentially subtracting the noes from the yeses-plus-noes.) This procedure is repeated for each type of behavior. The resulting map of localization of disturbances is then validated by making lesions restricted to the site determined by the intersept method and showing that the maximal behavioral deficit is obtained by the restricted lesion. (See Table I and Figure 2.)

Once the neurobehavioral correlation has been established by the multiple dissociation technique, two additional experimental steps are undertaken. First, holding the lesion constant, a series of variations is made of the task on which performance was found defective. These experimental manipulations determine the limits over which the brain/behavior-disturbance correlations hold and thus allow reasonable constructions of models of the psychological processes impaired by the various surgical procedures.

Second, neuroanatomical and electrophysiological techniques are engaged to work out the relationships between the brain areas under examination and the rest of the nervous system. These experimental procedures allow the construction of reasonable models of the functions of the areas and of the mechanisms of impairment.

Two major classes of memory processes have been delineated by the first of these operations: referential and contextual. The following

sections take up these processes while the final sections deal with modeling the neural mechanisms which underlie the processes.

REFERENCE MEMORY AND THE POSTERIAL CORTICAL CONVEXITY

Sensory Specificity:

Between the sensory projection areas of the primate cerebral mantle lies a vast expanse of parieto-temporo-preoccipital cortex. Clinical observation has assigned disturbance of many gnostic and language functions to lesions of this expanse. Experimental psychosurgical analysis in subhuman primates of course, is limited to nonverbal behavior; within this limitation, however, a set of sensory-specific agnosias (losses in the competence to choose among cues) have been produced. Distinct regions of primate cortex have been shown to be involved in each of the modality-specific memory functions: anterior temporal in gustation (Bagshaw & Pribram, 1953), inferior temporal in vision (Mishkin & Pribram, 1954), midtemporal in audition (Weiskrantz & Mishkin, 1958; Dewson, Pribram & Lynch, 1969), and occipitoparietal in somesthesis (Pribram & Barry, 1956; M. Wilson, 1975). In each instance chice reactions learned prior to surgical interference are lost to the subject postoperatively and great difficulty (using a "savings" criterion) in reacquisition is experienced, if task solution is possible at all.

The behavioral analysis of these sensory specific memory deficits has shown that they involve a restriction in sampling of alternatives, a true information processing deficit, a deficit in reference memory. Perhaps the easiest way to communicate this is to review the observations, thinking, and experiments that led to the present view of the function of the inferior temporal cortex in vision.

Search and Sampling:

All sorts of differences in the physical dimensions of the stimulus, for example, size, influences the deficiency in choice which occurs after inferior-temporal lesions (Mishkin & Hall, 1955), but the disability ismore complex - as illustrated in the following story:

One day when testing my lesioned monkeys at the Yerkes Laboratories at Orange Park, Florida, I sat down to rest from the chore of carrying a monkey a considerable distance between home cage and laboratory. The monkeys, including this one, were failing miserably at the visual discrimination tasks being administered. It was a hot, muggy, typical Florida summer afternoon and the air was swarming with gnats. My monkey reached out and caught a gnat. Without thinking I also reached for a gnat - and missed. The monkey reached out again, caught a gnat, and put it in his mouth. I reached out - missed! Finally the paradox of the situation forced itself on me. I took the beast back to the testing room: He was still deficient in making visual choices, but when no choice was involved, his visually guided behavior appeared to be intact. On the basis of this observation the hypothesis was developed that choice was the crucial variable responsible for the deficient discrimination following infero-temporal lesions. As long as a monkey does not have to make a choice, his visual performance should remain intact.

To test this hypothesis, monkeys were trained in a Ganzfeld made of a translucent light fixture large enough so the animal could be physically inserted into it (Ettlinger, 1957). The animal could press a lever throughout the procedure but was rewarded only during the period when illumination was markedly increased for several seconds at a time. Soon response frequency became maximal during this "bright" period. Under such conditions no differences in performance were obtained between infero-temporally lesioned and control animals. The result tended to support the

view that if an inferotemporally lesioned monkey did not have to make a choice he would show no deficit in behavior, since in another experiment (Mishkin & Pribram, 1954) where choice was involved the monkeys failed to distinguish between differences in brightness.

In another instance (Pribram & Mishkin, 1955) we trained the monkeys on a very simple object discrimination test: using an ashtray and a tobacco tin. These animals had been trained for 2 or 3 years prior to surgery and were sophisticated problem-solvers. This, plus ease of task, produced only a minimal deficit in the simultaneous choice task. When given the same cues successively the monkeys showed a deficit when compared with their controls, despite their ability to differentiate the cues in the simultaneous situation.

This result gave further support to the idea that the problem for the operated monkeys was not so much in "seeing" but in being able to refer in a useful or meaningful way to what they had seen previously. Not only the stimulus conditions but an entire range of response determinants appeared to be involved in specifying the deficit. To test this more quantitatively, I next asked whether the deficit would vary as a function of the number of alternatives in the situation (Pribram, 1959). It was expected that an informational measure of the deficit could be obtained, but something very different appeared when I plotted the number of errors against the number of alternatives (see Fig. 3).

If one plots repetitive errors made before the subject finds a peanut - that is, the number of times a monkey searches the same cue - vs the number of alternatives in the situation, one finds there is a hump in the curve, a stage where control subjects make many repetitive errors. The monkeys do learn the appropriate strategy, however, and go on to complete the task with facility. What intrigued me was that during this stage the

monkeys with inferotemporal lesions were doing better than the controls! This seemed a paradox. As the test continued, the controls no longer made so many errors, whereas the lesioned subjects began to accumulate errors at a greater rate than shown earlier by the controls.

When a stimulus sampling model was applied to the analysis of the data, a difference in sampling was found (Fig. 4). The monkeys with inferotemporal lesions showed a lowered sampling ratio; they sampled fewer cues during the first half of the experiment. Their defect can be characterized as a restriction on the number of alternatives searched and sampled. Their sampling competence, their competence to process information, had become impaired. The limited sampling restricted the ability to construct an extensive semantic memory store and to reference that memory during retrieval.

Automatic (Skilled) vs Controlled (Procedural) Processing

There is evidence that, for some tasks at least, memory processes need only the primary projection, input-output systems of the brain. Shiffrin and Schneider (1977; Schneider and Shiffrin, 1977) and Treisman (1977) have developed tasks which differentiate between automatic and controlled processing. They differ in that tasks which can be automatically processed involve overlearned skills in which a choice can procede without serial search (as indicated by short reaction times which are relatively independent of sample size). Thus the number of alternatives from which a cue is chosen has no effect on reaction time since all are processed in parallel. Controlled processing involves an earlier stage of skill and requires a serial search with reaction time dependent on the number of alternatives.

To determine what brain systems were involved in these two types of reference memory tasks, we used a modification of Treisman's displays and

measured the event-related electrical activity recorded from the striate and peristriate cortex, the inferior temporal lobe, far frontal and precentral cortex. The subject had to select a green square from a set of colored squares and diamonds, each of equal contour and luminance when compared to the rewarded cue.

The following display combinations were used in the experiment described here: a) a simple disjunctive display in which the green square had to be identified in a background of eight red diamonds; b) a more complicated disjunctive display in which the green square had to be identified in a background of red diamonds, white circles, and blue triangles not held identical); c) the conjunctive display in which the green square had to be identified in a background of green diamonds, red diamonds and red squares. The results showed that changes in brain electrical responses recorded from the primary sensory areas reflected the number of distinct features in the display. Conversely, changes in potentials recorded from the posterior and, initially, when the task was novel in the far frontal intrinsic association cortex reflected the difficulty of the task as determined by the number of alternatives and the conjunctive/disjunctive dimension of the displays.

Other experiments have allowed us to make a dissociation between the brain electrical activity evoked in the primary sensory projection cortex and the posterior intrinsic association cortex of the temporal lobe (Rothblat & Pribram, 1972; Nuwer & Pribram, 1979). These earlier studies as well as the current ones showed that the brain electrical activity evoked in the primary sensory receiving areas was largely determined by the features in the stimulus display irrespective of whether they were being reinforced, whereas the electrical potential changes evoked in the temporal cortex were primarily related to the cognitive operations involved,

i.e. the choices based on categorizing, pigeon holing (Broadbent, 1974). Clear and consistent involvement of the frontal cortex was found only on occasions when the task was novel or the reinforcing contingencies were shifted between runs. This relationship to novelty is consonant with the results described below.

EPISODIC MEMORY AND THE LIMBIC FOREBRAIN

Contextual Memory:

The second major division of the cerebral mantle to which memory functions have been assigned by clinical observation lies on the medial and basal surface of the brain and extends forward to include the poles of the frontal and temporal lobes. This frontolimbic portion of the hemisphere is cytoarchitecturally diverse. The expectation that different parts might be shown to subserve radically different functions was therefore even greater than that entertained for the apparently uniform posterior cortex. To some extent this expectation was not fulfilled: Lesions of the frontolimbic region, irrespective of location (dorsolateral frontal, caudate, cingulate-medial frontal, orbitofrontal, temporal polar-amygdala, and hippocampal) disrupted "delayed alternation" behavior. The alternation task demands that the subject alternate his responses between two cues (for example, between two places or between two objects) on successive trials. On any trial the correct response is dependent on the outcome of the previous response. This suggests that the critical variable which characterizes the task is its temporal organization. In turn, this leads to the supposition that the disruption of alternation behavior produced by frontolimbic lesions results from an impairment of the process by which the brain achieves its temporal organization. This supposition is only in part confirmed by further analysis: It has been necessary to impose severe restrictions on what is meant by "temporal organization" and

important aspects of spatial organization are also severly impaired. For instance, skills are not affected by frontolimbic lesions, nor are discriminations of melodies. Retrieval of long-held memories also is little affected. Rather, a large range of short term memory processes are involved. These clearly include tasks which demand matching from memory the spatial location of cues (as in the delayed response problem) (Anderson, Hunt, Vander Stoep, & Pribram, 1976) as well as their temporal order of appearance (as in the alternation task: Pribram, Plotkin, Anderson, & Leong, 1977). A similar deficit is produced when, in discrimination tasks, shifts in which cue is rewarded are made over successive trials (Mishkin & Delacour, 1975). The deficit appears whenever the organism must fit the present event into a "context" of prior occurrences, and there are no cues which address this context in the situation at hand at the moment of response.

The Registration of Events as Episodes:

As noted, different parts of the frontolimbic complex would, on the basis of their anatomical structure, be expected to function somewhat differently within the category of contextual memory processes. Indeed, different forms of contextual amnesia are produced by different lesions. In order to be experienced as memorable, events must be fitted to context. A series of experiments on the orienting reaction to novelty and its registration have pointed to the amygdala as an important locus in the "context-fitting" mechanism. The experiments were inspired by the results from Sokolov's laboratory (Sokolov, 1960).

Sokolov presented human subjects with a tone beep of a certain intensity and frequency, repeated at irregular intervals. Galvanic skin response (GSR), heart rate, finger and forehead plethysmograms, and electroencephalograms were recorded. Initially, these records showed the

perturbations that were classified as the orienting response. After several repetitions of the tone, these perturbations diminish and finally vanish. They habituate. Originally it had been thought that habituation reflected a lowered sensitivity of the central nervous system to inputs. But when Sokolov decreased the intensity of the tone beep, leaving the other parameters unchanged, a full-blown orienting response was reestablished. Sokolov reasoned that the central nervous system could not be desensitized but that it was less responsive to sameness: when any difference occured in the stimulus the central nervous system became more sensitive. He tested this idea by rehabituating his subjects and then occasionally omitting the tone beep, or reducing its duration without changing any other parameter. As predicted, his subjects now oriented to the unexpected silence.

The orienting reaction and habituation are thus sensitive measures of the process by which context is organized. We therefore initiated a series of experiments to analyze in detail the neural mechanisms involved in orientating and its habituation. This proved more difficult than we imagined. The dependent variables - behavior, GSR, plethysmorgram, and electroencephalogram - are prone to dissociate (Koepke & Pribram, 1971). Forehead plethysmography turned out to be especially tricky, and we eventually settled on behavior, the skin conductance (GSR), heart and respiratory responses, and the electrical brain manifestations as most reliable.

The results of the first of these experiments (Schwartzbaum, Wilson, & Morrissette, 1961) indicated that, under certain conditions, removal of the amygdaloid complex can enhance the persistence of locomotor activity in monkeys who would normally decrement their responses. The lesion thus produces a disturbance in the habituation of motor activity (Fig. 5).

The results of the experiments on the habituation of the GSR component of the orienting reaction (Bagshaw, Kimble, & Pribram, 1965) also indicate clearly that amygdalectomy has an effect (Fig. 6). The lesion profoundly reduces GSR amplitude in situations where the GSR is a robust indicator of the orienting reaction. Concomitantly, deceleration of heartbeat, change in respiratory rhythm, and some aspects of the EEG indices of orienting also are found to be absent (Bagshaw & Benzies, 1968). As habituation of motor activity (1960 a,b) and also habituation of earflicks (Bateson, 1969) had been severely altered by these same lesions, we concluded that the autonomic indicators of orienting are in some way crucial to subsequent behavioral habituation. We identified the process indicated by the autonomic components of the orienting reaction as "registering" the novel event.

However, the registration mechanism is not limited to novelty. Extending the analysis to a classical conditioning situation (Bagshaw & Coppock, 1968; Pribram, Reitz, McNeil, & Spevack, 1979) using the GSR as a measure of conditioning, we found that normal monkeys not only condition well but produce earlier and more frequent anticipitory GSR's as time goes by. Amygdalectomized subjects fail to make such anticipatory responses. As classical conditioning of a striped muscle proceded normally, it is not the conditioning per se which is impaired. Rather, it appears that registration entails some active process akin to rehearsal - some central mechanism aided by viscero-autonomic processes that maintains and distributes excitation over time.

Behavioral experiments support this suggestion. Amygdalectomized monkeys placed in the 2-cue task described above fail to take proper account of reinforced events. This deficiency is dramatically displayed whenever punishment, that is, negative reinforcement, is used. For in-

stance, an early observation showed that baboons with such lesions will repeatedly (day and day and week after week) put lighted matches in their mouths despite showing obvious signs of being burnt (Fulton, Pribram, Stevenson, & Wall, 1949). These observations were further quantified in tasks measuring avoidance of shock (Pribram & Weiskrantz, 1957). The results of these two experiments have been confirmed in other laboratories and with other species so often that the hypothesis needed to be tested that amygdalectomy produces an altered sensitivity to pain. Bagshaw and Pribram (1968) put this hypothesis to test and showed that the amplitude of GSR to shock is not elevated as it would be were there an elevation of the pain threshold. Rather the threshold is, if anything, reduced by the ablation. This experimental result suggests that amygdalectomy produces its effects by way of a "loss of fear" defined as a disturbance in "registering" the noxious event by placing it in context. In other words, the animal does not <u>remember</u> the noxious event so that its recurrence is experienced as novel and not fear producing.

<u>Processing the Familiar</u>:

Context is not composed solely of the registration of reinforcing and deterrent events. As important are the errors, the non-reinforced aspects of a situation, especially if on previous occasions they had been reinforced. It is resection of the primate hippocampal formation (Douglas & Pribram, 1966) which produces relative insensitivity to errors, to frustrative non-reward (Gray, 1975) and more generally to the familiar, non-reinforced aspects of the environment (the S$\triangle$ of operant conditioning; the negative instances of mathematical psychology). In their first experience with a discrimination learning situation subjects with hippocampal resections show a peculiar retardation provided there are many nonrewarded alternatives in that situation: For example, in an experiment

using a computer-controlled automated testing apparatus (DADTA) the subject faced 16 panels; discriminable cues are displayed on only two of these panels and only one cue is rewarded. The cues are displayed in various locations in a random fashion from trial to trial. Hippocampectomized monkeys were found to press the unlit and unrewarded panels for thousands of trials, long after their unoperated controls ceased responding to these "irrelevant" items. It is as if in the normal subject, a "ground" is established by enhancing "inattention" (extinction) to all the negative instances of those patterns that do not provide a relevant "figure." This "inattention" is an active, evaluating process as indicated by the behavior shown during shaping in a discrimination reversal task, when the demand is to respond to the previously nonreinforced cue: Unsophisticated subjects often begin by pressing on various parts of their cage and the testing apparatus before they hit upon a chance response to the nonrewarded cue.

These and many similar results indicate that the hippocampal formation is part of an evaluative mechanism that helps to establish the "ground", the familiar aspects of context.

The Spatiotemporal Structure of Context:

In some respects the far frontal resection produces memory disturbances characteristic of both hippocampectomy and amygdalectomy, though not so severe. Whereas medial temporal lobe ablations impair context formation by way of habituation of novel and familiar events, far frontal lesions wreak havoc on yet another contextual dimension, that of organizing the spatial and temporal structure of the context (Anderson, Hunt, Vander Stoep, & Pribram, 1976; Pribram, Plotkin, Anderson, & Leong, 1977). This effect is best demonstrated by an experiment which changed the parameters of the classical alternation task. Instead of interposing equal intervals between trials (go right, go left every 5 seconds) in the usual way, couplets of R/L were formed by extending the intertrial interval to 15" before each R trial (R5" L15" R5" L15" R5" L15"...). When this was done the performance of the far frontally lesioned monkeys improved immediately and was indistinguishable from that of the controls (Pribram & Tubbs, 1967; Pribram, Plotkin, Anderson, & Leong, 1977). This result suggests that for the subject with a bilateral far frontal ablation, the alternation task is experienced similarly to reading this page without any spaces between the words. The spaces, like the holes in doughnuts, provide the contextual structure, the parcellation or parsing of events by which the outside world can be coded and deciphered.

Context as a Function of Reinforcing Contingencies:

Classically, disturbance of "working" short term memory has been ascribed to lesions of the frontal pole. Anterior and medial resections of the far frontal cortex were the first to be shown to produce impairment on delayed response and delayed alternation problems. In other tests of context-formation and fitting, frontal lesions also take their toll. Here

also impairment of conditioned avoidance behavior and of classical conditioning and of the orienting GSR is found. Furthermore, as shown in Figure 7, error sensitivity is reduced in an operant conditioning situation. After several years of training on mixed and multiple schedules, the animals were extinguished over 4 hours. The frontally lesioned animals failed to extinguish in the 4-hour period, whereas the control monkeys did (Pribram, 1961).

This failure in extinction accounts in part for poor performance in the alternation already described: The frontally lesioned animals again make many more repetitive errors. Even though they do not find a peanut, they go right back and keep looking (Pribram, 1959).

This result was confirmed and amplified in studies by Wilson (1962) and by Pribram, Plotkin, Anderson, & Leong (1977), in which we asked whether errors followed alternation or non-reinforcement. We devised a situation in which both lids over two food wells opened simultaneously, but the monkey could botain the peanut only if he had opened the baited well. Thus the monkey was given "complete" information on every trial and the usual correction technique could be circumvented. There were four procedural variations: correction-contingent, correction, non-contingent, noncorrection-contingent, and noncorrection-noncontingent. The contingency referred to whether the position of the peanut was altered on the basis of the monkey's responses (correct or incorrect) or whether its position was changed independtly of the monkey's behavior. We then analyzed the relationship between each error and the trial that preceded that error. Table II shows that for the normal monkey the condition of

reinforcement and nonreinforcement of the previous trial makes a difference. for the frontally lesioned monkey this is not the case. Change in location, however, affects both normal and frontal subjects about equally. In this situation, as well as in an automated computer controlled version of the alternatives problem, frontal subjects are simply uninfluenced by rewarding or nonrewarding consequences of their behavior.

In a multiple choice task (Pribram, 1959) (see Figure 8) the procedure calls for a strategy of returning to the same object for five consecutive times, that is, to criterion, and then a shift to a novel item. The frontally lesioned animals are markedly deficient in doing this. Again, the conditions of reinforcement are relatively ineffective in shaping behavior in animals with frontal lesions and the monkeys' behavior becomes nearly random when compared to that of normal subjects (Pribram, Ahumada, Hartog, & Roos, 1964). Behavior of the frontally lesioned monkeys thus appears to be minimally controlled by expected outcomes.

THE MEMORY STORE

Distributed Memory:

As noted in the introduction, the experiments of Lashley had demonstrated that specific memory traces remain intact after extensive resection - up to 85% - of the primary visual cortex. These results make it imperative to assume that input becomes widely distributed. Several mechanisms have been produced to account for such distribution (see review by Pribram, 1982; Murdock, 1979, 1982; Eich, 1982), and how such distributed systems are organized (Edelman, 1974; Cooper, 1973; Pribram, 1966, 1971, 1982). Here I want to present direct evidence that indeed distribution does occur.

We trained monkeys to discriminate between a circle and a set of vertical stripes by pressing the right or left half of a plastic panel upon

which the cues were briefly projected (for 0.01 msec). Transient electrical responses were meanwhile recorded from fine electrode wires and these electrical responses (from single neurons or small neuronal aggregates) were related by computer analysis to the stimulus, response, and reinforcement contingency of the experiments (Pribram, Spinelli, & Kamback, 1967; Bridgeman, 1982). Thus we could distinguish from the record whether the monkey had looked at a circle or at the stripes, whether he had obtained a reward or made an error, and whether he was about to press the right or the left leaf of the panel. Interestingly enough, not all of these brain patterns were recorded from all of the electrode locations: From some, input-related patterns were obtained best; from others, the reinforcement-related patterns were derived; and still others gave us the patterns that were response-related. This was despite the fact that all placements were within the primary visual system, which is characterized anatomically by being homotopic with the retina. It appears therefore not only that optic events are distributed widely over the system but that response and reinforcement-related events reliably reach the input systems. Such results surely shake further one's confidence in the ordinary view that input events must be transmitted to the "association" area for associative memory to be effected.

Semantic Competence:

As noted above, the dimunition in reference memory which follows lesions of the posterior convexity is specific to the sensory mode served by the cortex which has been injured (Warrington et al., 1971; Warrington & Rabin, 1971; Warrington & Weiskrantz, 1973; Gordon, in press) and is therefore often interpreted as a change in channel capacity. Elsewhere I have reviewed the evidence that the neural channels which process sensory input which is involved not channel capacity. Further, the lesion sites

which result in these changes involve the intrinsic (or associational) systems related to specific primary extrinsic sensory systems, and not thesensory systems per se. The question therefore arises as to how competence is altered by lesions of the intrinsic systems of the brain.

One reasonable answer to this question can be framed in terms of coding and retrieval. Changes in competence following lesions to intrinsic cortical systems are changes in the mechanisms that address the long term distributed sotre. This leads to the dilemna that strategies of retrieval must also be represented and stored in the brain or they would not be disturbed by the lesion. It might be argued that such strategies are innate. Be that as it may, the changes in competence following lesions appear to become manifest in behaviors such as language, which is clearly learned. Whatever the innate competence for the behavior, it has been considerably modified by experience. What then are the characteristics of such modifiable competences?

One possible answer to this question is that the intrinsic systems of the forebrain operate corticofugally to preprocess sensory input on its route through the projection systems. In this way the sensory input becomes coded and recoded on the basis of experience.

There are several advantages to this formulation. First, it helps considerably in systematizing data. Second, it answers a persistent unanswered question originally formulated by Von Monakov: can disturbances in memory (such as the agnosias) be completely dissociated from disturbances in sensory functions? Von Monakov asked specifically: 1. Can agnosia occur in the absence of involvement of the primary projection systems? 2. Does agnosia occur in the absence of primary sensory difficulties ? Von Monakov's answer to the second question was an unequivocal "No," an opinion shared by Bay (1964) on the basis of more recent and carefully controlled studies. Von Monakov, after reviewing the anatomical

data also gave a tentative "No" to the first question, although he was neither completely convinced or convincing on this point. The preprocessing formulation and the data presented below indicate that the correct answer is more complex.

THE MECHANISMS OF RE-MEMBERING THE DISMEMBERED STORE

Recovery Functions:

The experimental findings detailed here allow one to specify a possible mechanism to account for the lesion-produced memory deficits: The posterior intrinsic cortex, by way of efferent tracts leading to the basal ganglia and brainstem [most likely to the colliculi or surrounding reticular formation (Pribram, 1958, 1960, 1977)] can be suggested to partition the events that occur in the associated projection cortex and classify, categorize these events according to one or another schema. This suggestion was tested in experiments (Spinelli and Pribram, 1966, 1967) in which the intrinsic cortex was electrically stimulated and records were made of recovery functions and receptive field properties of cells within the projection systems.

Records were made in the awake monkey. Paired flashes were presented, and recordings made from electrodes implanted in the occipital cortex. The response to 50 such paired flashes were accumulated on a computer for average transients. The flash-flash interval was varied from 25 to 200 msec. All records were made from striate (visual) cortex. The top traces were recorded prior to the onset of stimulation and the lower ones after stimulation of the inferotemporal region had begun. Note that with cortical stimulation the recovery function is depressed, that is, recovery is delayed.

Figure 9 shows the average of such effects in five subjects. Chronic stimulation of the inferotemporal cortex produces a marked increase in the processing time taken by cells in the visual system.

A parallel experiment in the auditory system. In this study, made with cats, removals of the auditory homologue of the inferotemporal cortex were performed. This homologue is the insular-temporal region of the cat. Dewson (1964) had shown that its removal impairs complex auditory discrimination (speech sounds), leaving simple auditory discrimination (pitch and loudness) intact. Removal, in addition, alters paired-click recovery cycles recorded as far peripherally as the cochlear nucleus. Bilateral ablation shortens the recovery cycle markedly. Of course, control ablations of the primary auditory projection cortex and elsewhere have no such effect. Thus we have evidence that chronic stimulation of the "association" cortex selectively prolongs, while ablation selectively shortens, the recovery time of cells in the related primary sensory projection system.

Receptive Fields

These results have been extended in both the auditory and visual modes. Electrode studies have shown alternations of visual receptive fields recorded from units at the optic nerve, geniculate and cortical levels of the visual projection systems produced by electrical stimulation of the inferior temporal cortex. The anatomy of the corticofugal pathways of these controls over sensory input also is under study. In the auditory system the fibers lead to the inferior colliculus and from there (in part via the superior olive) to the cochlear nucleus (Dewson, Noble, & Pribram, 1966). In our studies of the visual pathways, the fibers from the inferotemporal cortex lead via the putamen (Reitz and Pribram, 1969) to the

pretectal-collicular region as the site of interaction between the corticofugal control mechanism and the visual input system.

Contextual memory mechanisms have also become subject to neuro-physiological analysis. Again, as in the case of the sensory specific memory processes, cortico-fugal, efferent control mechanisms have been demonstrated. Results obtained in my laboratory show that in many instances these controls are the reciprocals of those involved in the sensory-mode specific processes (Spinelli & Pribram, 1967; Lassonde, Ptito & Pribram, 1981). Others (Skinner & Lindsley, 1973) have shown that the most likely pathways of operation of the fronto-limbic mechanisms involve the brainstem reticular formation. Here, however, as in the case of the sensory specific memory processes, control can be exerted as far peripherally as the primary sensory neuron (Spinelli & Pribram, 1967; Spinelli, Pribram, & Weingarten, 1965).

Channel Redundancy:

In general terms, the operation of efferents from sensory-specific posterior systems tend to reduce, and those from the frontolimbic systems tend to enhance, redundancy in the input channels, that is, the primary projection systems. This presumably is accomplished by inhibition and disinhibition of the ongoing interneuronal regulatory processes within the afferent channels, both those by which neurons regulate the activities of their neighbors and those which decrease a neuron's own activity.

Partitioning must work something like a multiplexing circuit. In neurophysiological terms, when the recovery time of neurons in the sensory projection system is increased by posterior "association" cortex stimulation, fewer cells are available at any given moment to receive the concurrent input. Each of a successive series of inputs thus will find a different set of cells in the system available to excitation. There is a

good deal of evidence that, in the visual system at least, there is plenty of reserve capacity - redundancy - so that information transmission is not, under ordinary circumstances, hampered by such "narrowing" of the channel (Attneave, 1954). Ordinarily a particular input excites a great number of fibers in the channel, ensuring replication of transmitted information. Just as lateral inhibition in the retina has the effect of reducing redundancy (Barlow, 1961), so the operation of the sensory-specific posterior "association" cortex increases the density of information within the input channel.

Conversely, the functions of the frontolimbic mechanism enhance redundancy, making more cells available at any given moment to concurrent input. This diminishes the density of information processed at any moment and enhances temporal resolution.

THE BRAIN MODEL OF THE MEMORY MECHANISM:

The Model:

The evidence presented here makes it not unlikely that one function of the posterior intrinsic and frontolimbic formations of the forebrain is to code events occurring within the input systems. As noted, the distribution of information (dismembering) implies an encoding process that can reduplicate events. Regrouping the distributed events (re-membering) also implies some sort of coding operation - one similar to that used in decoding binary switch settings into an octal format and that into assembly and still higher order programming language. An impaired coding process would be expected to produce grave memory disturbances. Lesion-produced amnesias, reference and contextual, therefore reflect primary malfunctions of coding mechanism and not the destruction of localized engrams.

Concretely, the intrinsic cortex is thus conceived to program, or to structure, an input channel. This is tantamount to saying that the input in the projection systems is coded by the operation of the intrinsic cortex. In its fundamental aspects, computer programming is a coding operation: The change from direct machine operation through assembler to one of the more manipulable computer languages involves a progression from the setting of binary switches to conceptualizing combinations of such switch settings in "octal" code and then assembling the numerical octals into alphabetized words and phrases and finally parcelling and parsing of phrases into sentences, routines, and subroutines. In essence these progressive coding operations minimize interference among the configurations of occurrence and recurrence of the events.

Implications:

The model has several important implications: First, the nonrecovered cells, the ones that are still occupied by excitation initiated by prior inputs, will act as a context or short-term memory buffer, against which the current input is matched. A match-mismatch operation of this sort is demanded by models of the process of recognition and selective attention spelled out on other occasions by Craik (1943), Sokolov (1960), Bruner (1957), MacKay (1956), and myself (1960a, 1963a,b). These "occupied" cells thus form the matrix of "uncertainty" that shapes the pattern of potential information, that is, the "unexpectancy" that determines the selection of input signals that might or might not occur. The normal functions of the posterior cortex are assumed to increase the complexity of this context while those of the frontolimbic systems would simplify and thus allow readier registration and parceling.

Second, in a system of fixed size, reduction of redundancy increases the degree of correlation possible with the set of external

inputs to the system, while enhancement of redundancy has the opposite effect. The number of alternatives or the complexity of the item to which an organism can attend is thereby controlled (Garner, 1962). This internal alteration in the functional structure of the classic sensory projection system thus allows attention to vary as a function of the spatial and temporal resolu- tion that excitations can achieve, with the result that events of greater or lesser complexity can be attended to. The sharper the spatial resolu- tion, the greater the "uncertainty" and, thus, the more likely that any set of inputs will be sampled for information. Conversely, the greater the temporal resolution, the more likely that attention is focused, and that events become grouped, memorable, and certain. In the extreme, the sharpening of the appetite for information becomes what the clinical neurologist calls stimulus-binding. Its opposite is agnosia, the inability to identify events because they fail to fit the oversimplified context of the moment.

Third, this corticofugal model of the functions of the so-called association systems relieves us of the problem of higher and higher order infinite regress - an association area "homunculus" who synthesizes and abstracts from inputs, only to pass on these abstractions to a still higher "homunculus," perhaps the one who makes decisions, etc. Former ways of looking at the input-output relationships of the brain invariably have come up against this problem (implicit or explicit) of "little men" inside "little men."

According to the model presented here, there is no need for this type of infinite regress. The important functions of perception, decision, etc. are going on within the primary sensory and motor projection systems. Other brain regions such as the posterior intrinsic sensory-specific systems and the frontolimbic systems exert their effects by altering the

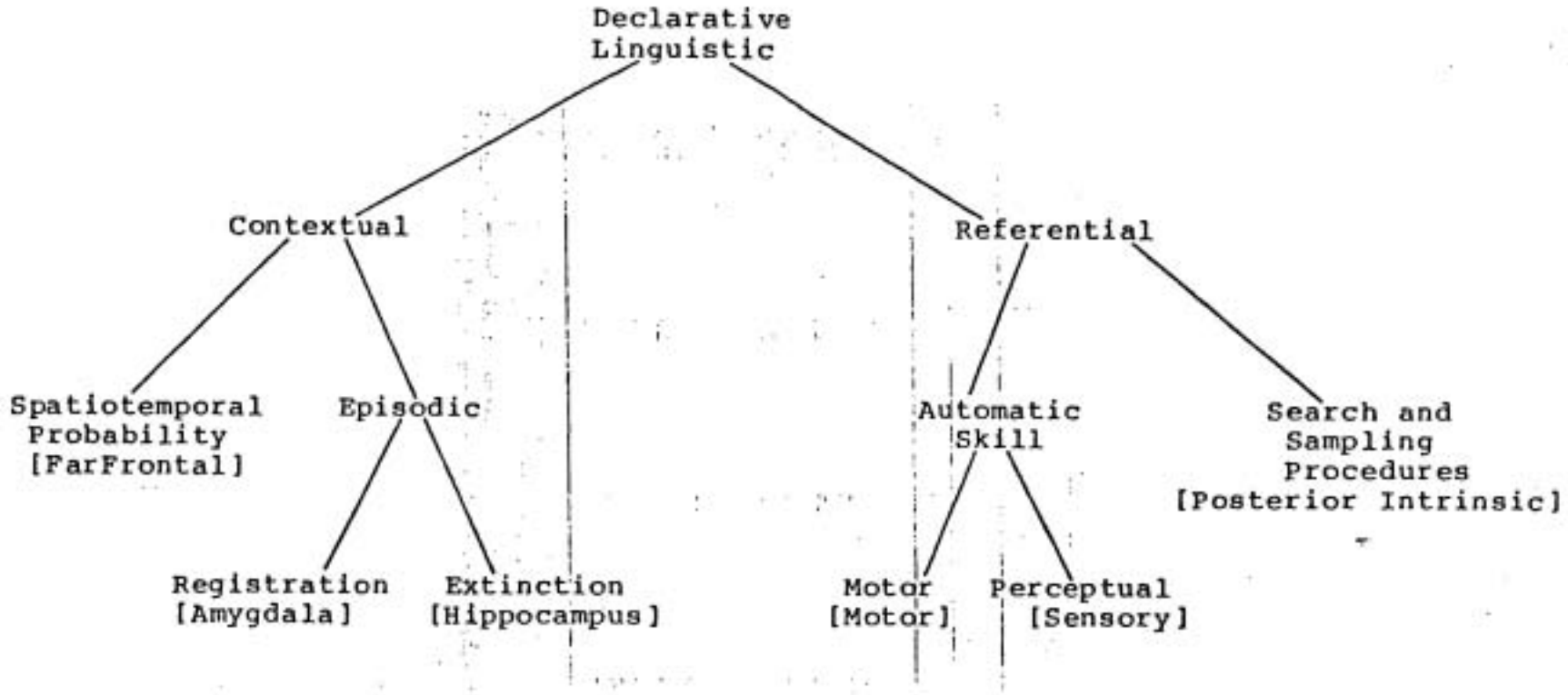
Declarative
Linguistic
Contextual
Referential
Spatiotemporal
Probability
[FarFrontal]
Episodic
Automatic
Skill
Search and
Sampling
Procedures
[Posterior Intrinsic]
Registration
[Amygdala]
Extinction
[Hippocampus]
Motor
[Motor]
Perceptual
[Sensory]

Table 1
Simultaneous Visual Choice Reaction[a]

Operates without deficit			Operates with deficit			Nonoperate controls		
	Pre	Post		Pre	Post		Pre	Post
OP 1	200	0	PTO 1	120	272	C 1	790	80
OP 2	220	0	PTO 2	325	F	C 2	230	20
OP 3	380	0	PTO 3	180	F	C 3	750	20
LT 1	390	190	PTO 4	120	450	C 4	440	0
LT 2	300	150	T 1	640	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						
MV 3	290	130						
MV 4	230	10						
MV 5	280	120						
CIN 1	120	80						
CIN 2	400	60						
CIN 3	115	74						
CIN 4	240	140						

[a]Pre- and postoperative scores on a simultaneous visual choice reaction of the animals whose brains are diagrammed in Fig. 2, indicating the number of trials taken to reach a criterion of 90% correct on 100 consecutive trials. Deficit is defined as a larger number of trials taken in the "retention" test than in original learning. (The misplacement of the score H 1 does not change the overall results as given in the text.)

functional organization of the primary systems. Thus these intrinsic systems are not "association" but "associated" systems; they simply alter the configuration of input-output relationships processed by the projection systems. In computer language the intrinsic systems function by supplying sub-routines in a hierarchy of programs, subroutines contained within and not superimposed from above on the more fundamental processes. In this fashion the infinite higher-order abstractive regress is avoided. One could argue that in its place a downward regress of sub- and subroutines is substituted. I would answer that this type of regress, through progressive differentiation, is the more understandable and manipulable of the two.

A final advantage of the model is that the signal itself is not altered; the invariant properties of a signal are unaffected (unless channel capacity is overreached). It is only the organization of the channel itself -- the matrix within which the signal is transmitted - that is altered. Thus the same signal carries more or less information, depending on the "width" of the channel. Further, the signal carries different meanings, depending on the particular structure or organization of the redundancy of the channel.

Summary

Experimentally produced local forebrain damage does demonstratably impair memory functions. However, the impairment apparently is not so much a removal of localized engrams as an interference with the mechanisms which ordinarily code neural events so as to allow facile storage and retrieval. Thus, the evidence shows that anatomically the memory trace is distributed within a neural system by means of an en-coding process, while as a function of de-coding the distributed, dismembered engram is reassembled, that is, re-membered. Thus, what and whether something is

remembered is in large part dependent on how it is, and that is is, adequately coded.

Two major classes of memory mechanisms were identified: Reference and Contextual. Each of these classes is subdivided into others. Reference memory is composed of sets of sensory specific skilled, automatic processes on the one hand and another set of sensory specific search sampling procedures on the other. Contextual memory is composed of a mechanism which registers episodes, another which processes the familiar and a third which generates, "works" to organize the spatiotemporal structure of context. How these memory mechanisms interact to produce declarative linguistic processes remains to be investigated.

Table II
Percentage of Alternation as a Function of Response and Outcome of Preceding Trial[a]

	Preceding trial[b]			
S	A-R	A-NR	NA-R	NA-NR
Normal				
394	53	56	40	45
396	54	53	36	49
398	49	69	27	48
384	61	83	33	72
Total	55	68	34	52
Frontal				
381	49	51	41	43
437	42	46	27	26
361	49	48	38	35
433	43	39	31	32
Total	46	46	33	33

[a]Comparison of the performance of frontally ablated and normal monkeys on alternations made subsequent to reinforced (R) and nonreinforced (NR) and an alternated (A) and nonalternated (NA) response.

[b]A, alternated; NA, did not alternate; R, was rewarded; and NR, was not rewarded.

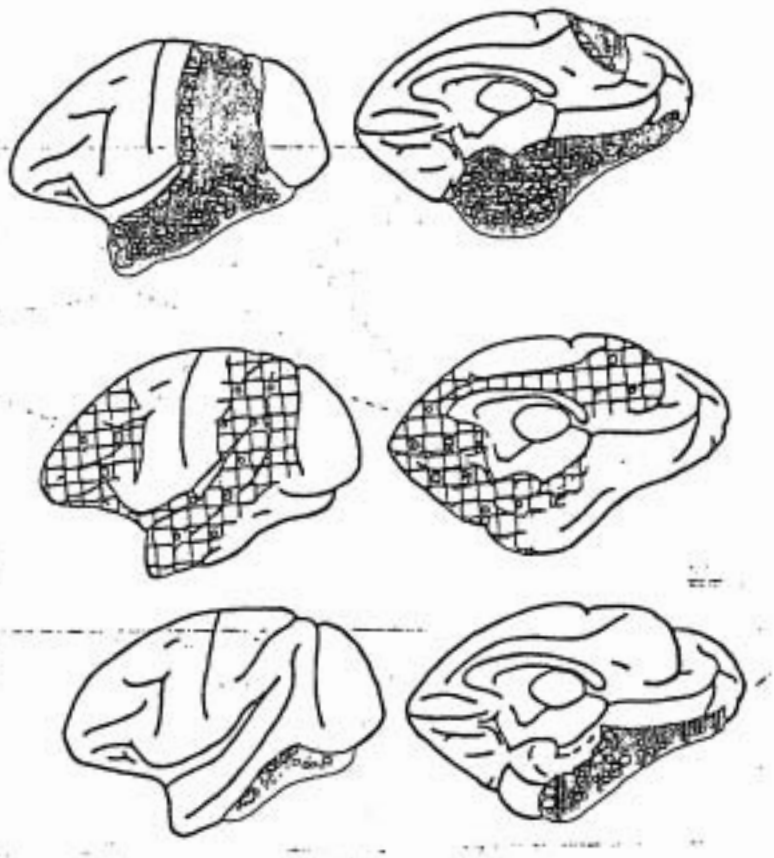

Fig. 2. The upper diagram represents the sum of the areas of resection of all of the animals grouped as showing deficit. The middle diagram represents the sum of the areas of resection of all of the animals grouped as showing no deficit. The lower diagram represents the intersect of the area shown in the black in the upper diagram and that not checkerboarded in the middle diagram. This intersect represents the area invariably implicated in visual choice behavior in these experiments.

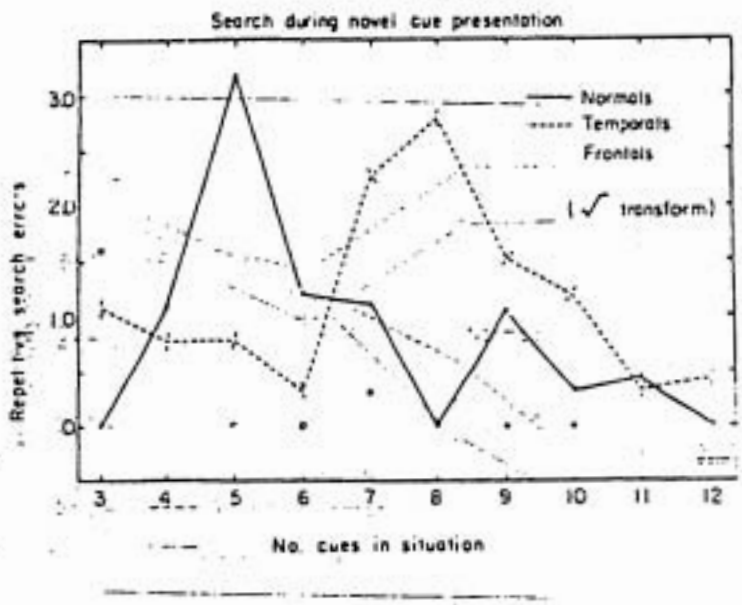

Fig. 3. Graph of the average number of repetitive errors made in the multiple object experiment during those search trials in each situation when the additional, that is, the novel, cue is first added.

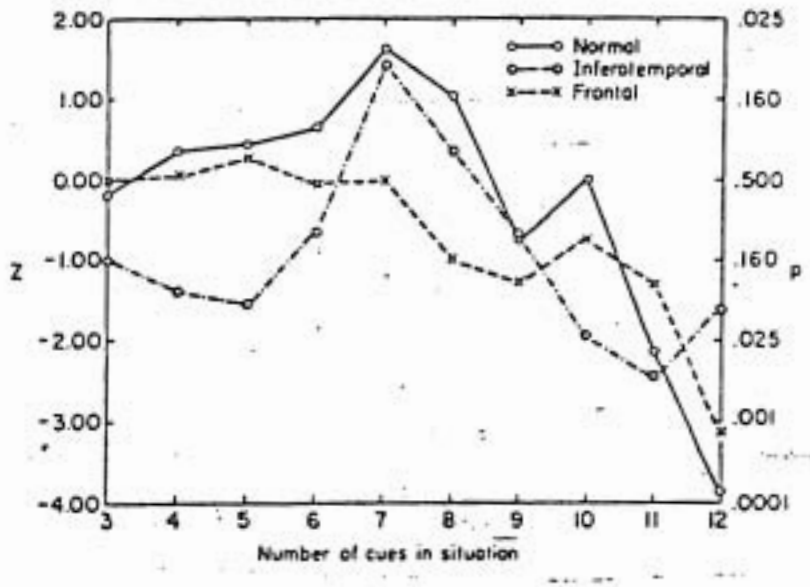

Fig. 4. Graph of the average proportion of objects (cues) that are sampled (except novel cue) by each of the groups in each of the situations. To sample, a monkey had to move an object until the content or lack of content of the food well was clearly visible to the experimenter. As was predicted, during the first half of the experiment the curve representing the sampling ratio of the posteriorly lesioned group differs significantly from the others at the 0.024 level (according to the nonparametric Mann-Whitney U Test).

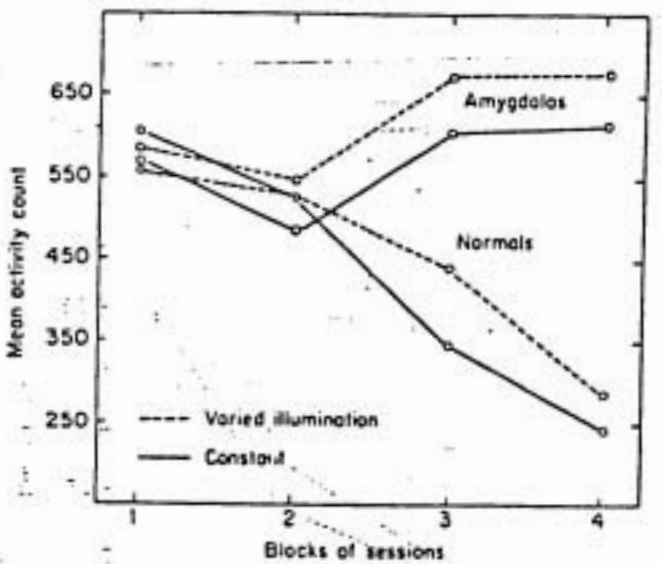

Fig. 5. Postoperative activity scores of normal and amygdalectomized monkeys for successive blocks of three sessions under conditions of constant illumination and more intense, varied illumination.

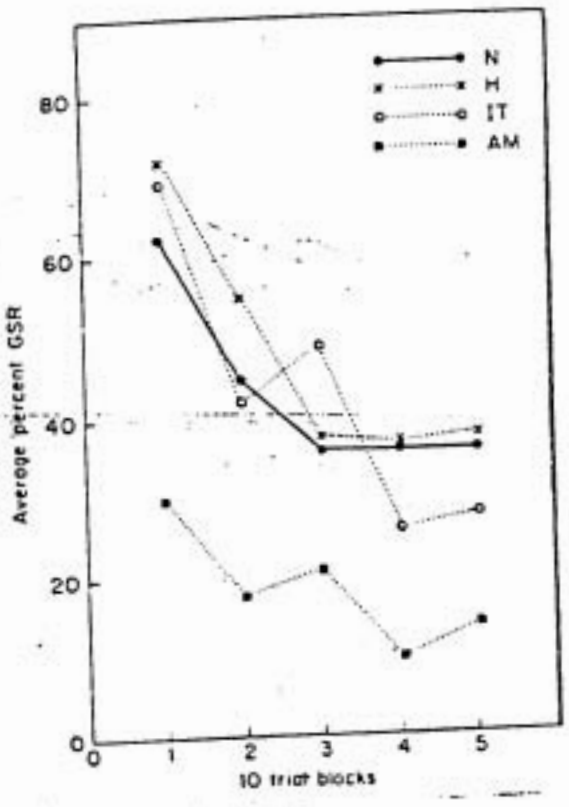

Fig. 6. Curves of percent GSR response to the first 50 presentations of the original stimulus for the normal (N) and three experimental groups (H, IT, AM), i.e., hippocampal, inferior temporal, and amygdala resected monkeys.

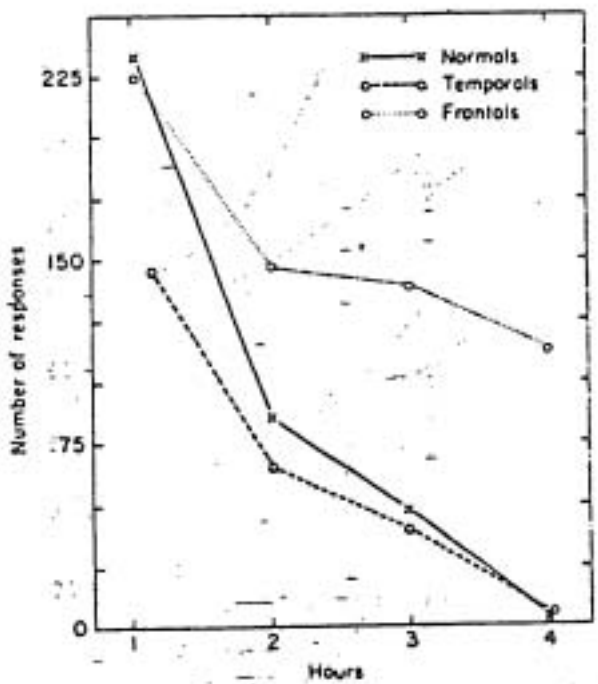

Fig. 7. Graph of performance of three groups of monkeys under conditions of extinction in a mixed schedule operant conditioning situation. Note the slower extinction of the frontally lesioned monkeys.

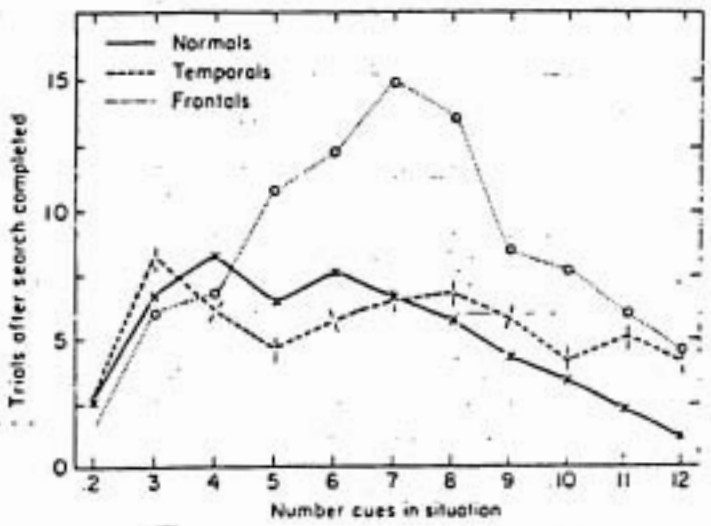

Fig. 8. Graph of the average number of trials to criterion taken in the multiple object experiment by each group in each of the situations after search was completed, that is, after the first correct response. Note the difference between the curves for the controls and for the frontally operated group, a difference that is significant at the .05 level by an analysis of variance ($F = 8.19$ for 2 and 6 df) according to McNemar's procedure performed on normalized (by square root transformation) raw scores.

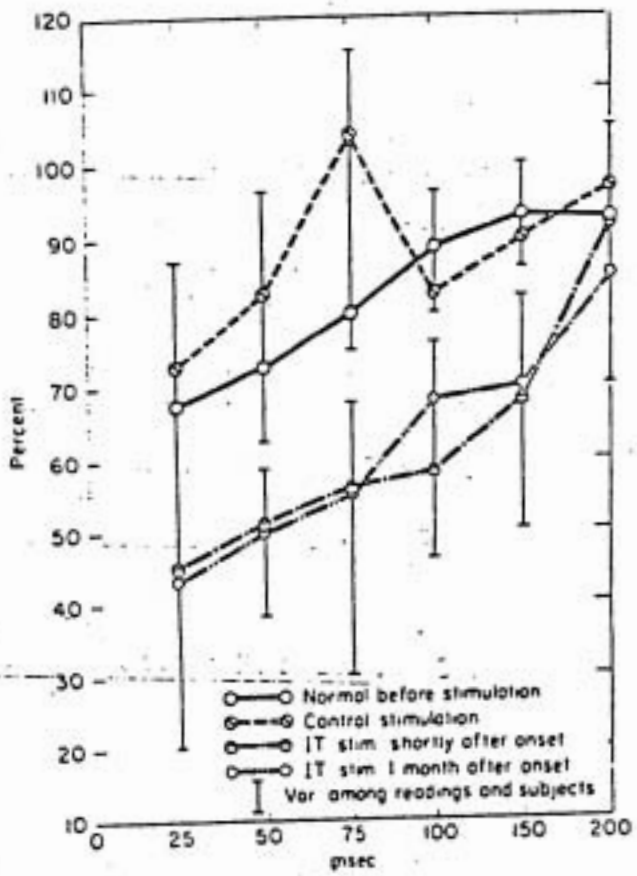

Fig. 9. A plot of the recovery functions obtained in five monkeys before and during chronic cortical stimulation: relative amplitude of the second response as a function of interflash interval.

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