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A NEUROPSYCHOLOGICAL ANALYSIS OF CEREBRAL FUNCTION: AN INFORMAL PROGRESS REPORT OF AN EXPERIMENTAL PROGRAM

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A NEUROPSYCHOLOGICAL ANALYSIS OF CEREBRAL FUNCTION: AN INFORMAL PROGRESS REPORT OF AN EXPERIMENTAL PROGRAM

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I am going to organize what I have to say around three basic questions. Most of the work which is addressed to the first question was done some years ago; most of the work which is related to the last question is now in progress in our laboratories and elsewhere. The questions are: (1) How can one establish and characterize brain-behavior relationships? Specifically, I was interested in establishing characteristic relationships for those parts of the forebrain which, at the time the studies were initiated, were essentially "silent" to experimental analysis. (2) What is the psychological meaning of the brain-behavior relationships thus established? (3) What is the neuro-physiological meaning of these brain-behavior relationships?

PHASE I. THE BRAIN-BEHAVIOR RELATIONSHIP

Around 1946, at the time I began this work, there were two vast expanses of the brain cortex which were essentially "silent" to existing experimental manipulation: the posterior "association" cortex and the frontolimbic systems. No physiological function could be assigned to them, and we did not know — though there was much conjecture on the basis of clinical or anatomical evidence — what their function in behavior might be. Experimental analyses made in the laboratory were sorely needed. We therefore initiated a series of studies using, with few exceptions, subhuman primates — mostly Macacus Rhesus. (Experiments using human subjects will be mentioned as well; these as a rule, were made in order to test whether the results obtained on monkeys could usefully be extrapolated to man).

Techniques: The primary, though not the sole, physiological-anatomical technique used in this early phase of the work, was the ablation method checked histologically. After sacrifice of the animal, serial reconstructions of the lesion were always made and the depth and relation to thalamus or other structures was outlined.

Combined with cortical removals was an extensive behavioral survey of the subject, both pre- and post-operatively. A variety of behavioral techniques was used. For example, a shuttle box was made in which conditioned avoidance behavior was studied. Another was an operant-conditioning situation where the monkey was taught to press levers. In this situation his pressing rate can be controlled by simple cues and by programming the reinforcement. For a large number of experiments we used a Yerkes box in which monkeys could be taught to make visual choices between two alternatives. Finally, we devised a multiple-choice procedure (Fig. 1). A number of cues can be placed over holes in which the rewards are hidden; the position of the cues is randomized from trial to trial.

Our present setup shows considerable refinement of these basic techniques. The multiple-choice procedure has been automated and is programmed by a small general purpose computer (PDP-8) to which is attached some hardware and software which in aggregate is called the DADTA machine (Discrimination Apparatus for Discrete Trial Analysis). This device allows us to perform a great variety of behavioral tasks and automatically records the results of the experiment for us. In addition, DADTA is a much more powerful tool for the analysis of behavior than we had before. Both animals and children (and even adult humans) like to work the device. There is no experimenter directly in the situation, so there is reliability comparable to that obtained with operant equipment. Further, each and every trial, i.e. every panel press, is recorded on punched tape so that computer analysis of the data can be easily obtained. The problem depends strictly on the input program. We can, for instance, program a sequence so the subjects must respond to 1, 3, 5, 7, in that order, before they receive the peanut or a piece of "M & M" candy. There are a variety of such problems that can be presented (40) and I will be talking about some of these.

A quick example of the power and utility of this instrument is in order here. In the old hand-operated Yerkes box, a sophisticated animal — one having been trained for several years — required to discriminate between the numbers 3 and 8, will probably fail to master this in less than 1,000 trials. But with the DADTA, a completely naive animal takes an average of only 250 trials which represents just five days of training. This is an unexpected dividend of the DADTA.

What makes it so much better? There are probably at least two reasons. One is simply that, from the point of view of the subject, it is much more "fun" to manipulate. But probably the most important consideration is that, by changing the position of the cues on *each* trial, we are rid of any position tendency and find, therefore, no position habits. We do not have the confounding of position with the discrimination for which we are testing. Most animals and children will respond to position cues first and only later will "catch on" that this may be irrelevant. But by *initially* changing position from trial to trial, the subject is immediately alerted to the fact that position is irrelevant.

The experiments to which I will have reference were done with no fewer than four animals per group and, as a rule, the experiments have been replicated. If they have not, I will so indicate.

In order to establish the brain-behavior relationships I devised a data-processing technique (Fig. 2) which is called the method of "the intersect of sums" (27). Listed separately are those subjects that had a post-operative deficit on a particular problem (in the example given, a visual choice reaction) ,and those without such deficit. "Deficit" as here used, means either failure to perform the task at criterion in 1,000 trials — 90 correct out of 100 consecutive responses — or to relearn it in the number of trials taken to learn the task pre-operatively (in other words, no savings). The method of intersect of sums was then applied in this fashion: a plot was made of the sum of all the lesions (which had been individually reconstructed) that produced deficit; another plot was made

of the sum of all the lesions that produced no deficit; and the two were superimposed. Here (in Fig. 2) is the remaining cortex (the intersect) upon which we then focused. This infero-temporal region is the critical cortical area concerned in visual choice behavior. No other portion of the "silent" cortex is involved.

The Posterior Intrinsic Cortex and Sensory Specificity: When the "Intersect of Sums" technique was applied to the problem of making neuro-behavioral correlations by including other tasks, the posterior of the silent areas was shown to be divisible into regions, each of which served one or another sense modality; i.e., there is modality specificity within this posterior "association" cortex. An example follows.

After one group of animals was given a parietal lesion and another an inferotemporal lesion, they were tested either for original post-operative retention of a pre-operatively learned task. Both groups were trained on both a visual- and a somesthetic-discrimination task. After the parietal operation the monkeys had difficulty in original learning and retention of the somesthetic discrimination (59). (The apparatus used was an infrared device (6) by which the monkeys' performance was observed, televised and converted into visible light for display. The animals were working in darkness, but we could watch what was going on via a television screen.) On the other hand, visual discrimination remained intact, i.e., the savings criterion was met and orginal learning fell within the scores of the controls.

Conversely, the inferotemporal group performed the somesthetic problem within normal limits, both in learning and during retention, but showed complete failure in learning and remembering the visual discrimination. (See also Pribram and Barry (25)).

In the auditory mode, the data (58) are almost as clear-cut. These data are now being replicated (10). The results show again that inferotemporal lesions result in a visual discrimination deficit; and that, this time, auditory discrimination remains unaffected. Conversely, a mid-temporal lesion, while leaving visual discrimination intact, does produce a deficiency in auditory discrimination. For taste, an anterior temporal locus has been isolated (2, 38) by the similar use of the intersect of sums technique (27).

The question remains whether there are any "supramodality" regions in this posterior cortical region. This question has been experimentally explored but most of the work is as yet unpublished (57). So far there has been no evidence in the monkey that there is a supramodality organization in the posterior "association" cortex (11a, 57). In man, the data from Milner's group in Montreal (18) and Teuber's group at MIT (56) suggest that there might be such a thing as a locus for visuo-somatic spatial organization, or the organization of verbal behavior, irrespective of mode. However, these data are open to other interpretations so that this remains an issue which needs a good deal more investigation, both at the human and subhuman levels.

To summarize: there is a considerable body of evidence that the

posterior "association" cortex of primates contains areas which are modality-specific. Whether some supramodality organization exists in man remains an open question.

The Fronto-limbic Formations and Behavior Sequences: Here (Fig. 3) is the method of the "Intersect of Sums" applied to the delayed response experiment. For the delayed response problem a peanut is shown to the animal over one of the food wells. A screen is interposed while the peanut is hidden in the well; then the sceen is raised, giving the monkey an opportunity to find the peanut. There are variations of the delayed reaction problem that do make a difference (21, 22, 45) but varying the delay period is not an important one (46). More of this in a moment.

(The dotted portions represent experiments that are in the literature, including one of my own, which suggest there may be a deficit obtained from lesions in these locations. This turns out to be an artifact of this particular task since there are control animals who have never been operated on at all — four such animals in my experience — who also show a deficit on this task.)

Another task, closely related to delayed response but not identical, has given somewhat more reliable data. This task is delayed alternation. Performance of this task is impaired whenever a lesion invades frontal or limbic cortex. To perform, the subject must simply alternate his response from trial to trial: right, left, right, left, with a screen interposed between trials. We used a five-second delay, standard-correction technique for both tasks.

This figure (Fig. 4) shows an orbito-insulo-temporal resection. The OIT region includes the amygdala, the anterior portion of the insula and the posterior orbital portion of the frontal lobe. This region receives its projection from the midline, medial macrocellular mediodorsal and medial intralaminar nuclei (16). It can also be differentiated as a unified sector by the method of strychnine neuronography (41, 43). Another such region is the cingulate cortex, which really comprises a good deal of the medial frontal cortex as well as the cingulate gyrus. This region is the projection sector of the anterior nuclear group of the thalamus (39). The anterior nuclei project not only to the thin strip of cortex above the corpus callosum but more widely to the medial cortex anterior to and under the corpus callosum. Both of these regions have become standard ones in our repertoire. Finally, lesions of the hippocampal cortex also lead to difficulty with the delayed alternation problem (47a).

When one varies the method of presenting the delayed response and delayed alternation problems (Fig. 5) one can further differentiate between lesion effects. Frontal and limbic (OIT, cingulate and hippocampal) lesions have different effects on the performance of different variations of the task (42). The effective variation is a change from a left-right to a go no-go procedure. In the go no-go situation the animal is reinforced every alternate time and is expected to stay away from the well on the other times. On one trial the peanut is placed in the well, the screen comes up, the animal responds. On the following trial there is no peanut in the well;

the animal has to learn to withhold his response. If he does not, the non-reinforced trial is repeated until he does withhold. On the next trial, the peanut is again in the well. From the results of an initial experiment, this go no-go form of alternation appears to be more severely impaired than the right-left variation of the task when the lesions are limbic.

On the other hand, when the lesions are of frontal cortex (Fig. 6) the go no-go variation of the procedure turns out to be much easier for the monkey than the right-left variation (22).

To summarize: frontal and limbic lesions produce effects different from those produced by the posterior cortex. I have not reviewed here the evidence that the fronto-limbic defect is not modality-specific but such reviews are in the literature (33, 48). The fronto-limbic effect is demonstrated in a class of tasks of the delayed response and delayed alternation type. Further differentiation can be made between frontal and limbic structures by varying the problem from a right-left to a go no-go procedure. Performance in right-left delay tasks is more seriously disturbed by frontal lesions; performance of go no-go delay tasks apparently suffers most from limbic lesions.

Brain Lesions, Learning and Remembering: But removal of cerebral tissue was not the only tool in our armamentarium. Simultaneously, experiments were carried out in which we placed aluminum hydroxide cream on the cortex or injected it into selected areas of the cortex (26, 28, 53). Multiple foci of altered electrical activity were thus produced, often leading to actual seizure patterns. The behavioral techniques found useful in the ablation experiments were used in these studies as well. When one has trained the animal before the abnormal electrical activity develops, (e.g.) spike or spike and slow-wave complexes), one finds no impairment of behavior (Figs. 7 and 8). The monkey runs along smoothly at criterion, despite the abnormal electrical activity. As in the case of the ablation experiments, the aluminum hydroxide cream implantations were made in each of the regions discussed and performance was recorded for many weeks (52, 53, 54).

On the other hand, if one trains the animals only after the abnormal electrical activity has appeared, a marked change in behavior can be demonstrated (Figs. 9 and 10). Original learning of a particular task is impaired when the electrical activity of the appropriate cortex becomes abnormal. These figures depict visual discrimination and alternation performance following EEG abnormality in the inferotemporal and frontal cortices, respectively. Learning is delayed approximately five-fold. (Note that the slope of the discrimination curve is not drastically changed; rather, the onset of learning is retarded. This finding may be important in uncovering the mechanism which underlies the disturbance). Thus, the acquisition of behavior appears to be highly correlated with what is recorded electrically from the brain, even though there seems to be no such correlation between such electrical changes and performance *per se*, i.e., the ability to remember the problem.

To make the story complete I should mention that converse experimental results have also been obtained. Using the ablation technique, Law-

rence Weiskrants of Cambridge University (58) followed this paradigm: train the animals on a particular day to criterion on a particular discrimination; let's say A versus B. On the following day, test for the retention of A versus B, and teach a new discrimination, C versus D. On day 3 test for retention of C versus D, and teach E versus F. He did this with many variations, always using easily discriminable cues such as junk objects, and showed that after ablation of the inferotemporal cortex learning was unaffected, though remembering suffered severely. In other words, the acquisition of new performance remained unimpaired by the resection, learning rates were identical, summed across days. On the other hand, retention was markedly impaired — that is, from day to day these animals forgot a good deal of what they had learned the day before.

So, in summary, the irritative and the ablative lesions produce different results: the brain's electrical abnormality is correlated with altered acquisition, brain cortex removal with disturbed remembering. I use the word "remembering" here in a sense opposite to dismembering: these animals must put together again — re-trieve or re-construct — elements used to solve problems.

PHASE II. THE PSYCHOLOGICAL SIGNIFICANCE OF THE BRAIN-BEHAVIOR RELATIONSHIPS

Now for the psychological significance of these findings. The question can be put somewhat like this: If one obtains a deficit in color discrimination, does that mean that the animal is color blind? One makes a removal of cortex, and the animal now fails a color discrimination; does that in itself mean the animal is color blind? Obviously not. And just as obviously we needed other kinds of tasks besides color discrimination to test the limits of the deficient behavior. So we turned to brightness differences and to patterns of various sorts. Our findings showed that all manner of visual tasks are affected by this particular lesion (19, 21, 22).

Search and Sampling: All sorts of differences in the physical dimensions of the stimulus, e.g., size, are distinguished less after the lesion (20) (Fig 11). But there is more to the disability than this — as illustrated in the following story. One day, while testing monkeys with such lesions at the Yerkes Laboratories in Orange Park, Florida, I sat down to rest from the chore of carrying a monkey the considerable distance between home cage and laboratory. The monkeys, including this one, were failing miserably 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 as deficient in making visual *choices* as ever. But when no choice was involved the monkey's visually-guided behavior appeared to be intact. This gave rise to the following experiment (Fig. 12) which Ettlinger (11) accomplished. On the basis of this particular observation we made the hypothesis that choice was the crucial variable responsible for the deficient discrimination following inferotemporal lesions.

As long as a monkey doesn't have to make a choice, his visual performance should be found intact. To test this, monkeys were trained in a Gantzfeld made of a translucent light fixture large enough so the animal could be physically inserted into it. The animal could press a single 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 maximum during this "Bright" period. Under such conditions no differences in performance were obtained between inferotemporally-lesioned and control animals. The result tended to support the view that if an inferotemporally-lesioned monkey didn't have to make a choice he would show no deficit in behavior, since in another experiment (22) the monkeys failed to respond differentially to differences in brightness.

In another instance (Fig. 13) we (44) trained the monkeys on a very simple object discrimination test: an ashtray versus a tobacco tin. These animals had been trained for two or three years before they were operated on and were therefore sophisticated problem-solvers; this, plus ease of task, accounts for the minimal deficit in the simultaneous choice task. (There are two types of successive discrimination: In one the animal has either to go or not go and in the other he has to go left or go right.) When given the same cues successively the monkeys showed deficit when compared with their controls, despite this demonstrated ability to differentiate the cues in the simultaneous situation.

This result further supported the idea that the problem for the operated monkeys was not so much in "seeing" but in usefully differentiating what they saw. Not only the stimulus conditions *per se* but the contexts in which they appear determine the deficit. To test this idea in a quantitative fashion we next asked whether the deficit would vary as a function of the *number* of alternatives in the situation (30) (Fig. 14). This experiment has not as yet been replicated and so the results must be considered tentative, albeit persuasive. The hope was that an informational measure of the deficit could be obtained. Actually something very different appeared when the number of errors was plotted against the number of alternatives.

If one plots repetitive errors made before the subject finds a peanut — i.e., the number of times a monkey searches the same cue — versus 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 was a paradox. As the test continued, however, after the controls no longer made so many errors, the lesioned subjects began to accumulate an error hump even greater that that 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. 15): 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 in the visual field; however, the limitation is not in the visual-spatial field but in the information-processing field, i.e., in the number of alternatives they can sample or handle at any one time.

Most of the variance that produced the error humps is accounted for by the monkeys' reactions to the introduction of a novel cue. The inferotemporally-lesioned subjects (as well as the controls) made their runs of repetitive errors on these occasions. However, during the early parts of the procedure, when there were only four or five cues in the situation, the inferotemporally lesioned monkeys found the correct one more rapidly than did the controls, who sampled more of the previously reinforced cues before turning to the novel one. Frontally lesioned subjects invariably chose the novel cue immediately.

To summarize: The modality-specific defect that results from a posterior "association" system lesion appears to produce an information-processing defect best described as a restriction on the number of alternatives searched and sampled. In short, they fail to remember *prior* discriminations as well as do controls, and this failure alters the sampling of *current* cues. The process of selective attention is apparently impaired by the lesion. I will return to this notion of a memory-based *input* processing defect when I discuss the model. But first let me round out the present picture by presenting briefly some data on the frontolimbic systems.

The Consequences of Behavior: For purposes of comparison let me begin this time by summarizing these effects (Figs. 16 and 17) as well. Frontally (and also limbically) lesioned primates also fail to be influenced by their experience but in a very different way than are the posteriorly lesioned subjects. They appear to be impervious to the outcomes, the *consequences* of their behavior. Initially, this defect appeared most dramatically in situations demanding the avoidance of shock (27, 47) and those in which behavior is guided by errors.

Error sensitivity was tested in an operant conditioning situation (Fig. 18). After several years of training on mixed and multiple schedules, four hours of extinction were run, i.e. the reinforcement (peanuts) were no longer delivered, although everything else in the situation remained the same. Note that the frontally lesioned animals failed to extinguish in the four hour period, whereas the control monkeys did (33).

This failure in extinction accounts in part for poor performance in the alternation already described (Fig. 19): the frontally lesioned animals again make many more repetitive errors. Even though they don't find a peanut, they go right back and keep looking (30).

This result was confirmed and amplified in a study by Wilson (61). He analyzed the occasions for error — did errors follow alternation or nonreinforcement? To test this, he devised a situation in which both lids over the food well opened simultaneously but the monkey could obtain the peanut only if he had opened the baited well. Thus the monkey was given "complete" information in every trial and the usual correction technique

could be circumvented. With this apparatus he presented the procedure with four variations: correction-contingent, correction-noncontingent, noncorrection-contingent, and noncorrection-noncontingent. The contingency referred to is whether the position of the peanut depended on the prior correct or incorrect response of the monkey or whether this position was alternated independent of the monkey's behavior. Wilson then analyzed the relationship between an error and the trial preceding that error. Notice (Fig. 20) that, for the normal monkey the condition of reinforcement and non-reinforcement of the previous trial makes a difference, whereas for the frontally lesioned monkey this is not the case. Alternation affects both normal and frontal subjects about equally. In this situation, frontal subjects are simply uninfluenced by rewarding or nonrewarding the consequences of their behavior.

Now let me return to the multiple choice experiment we just discussed at such length (30). Here also this inefficacy of outcomes to influence behavior is demonstrated. This (Fig. 21) is what happens after the monkeys have found the peanut. The procedure calls for the strategy of return to the same object for five consecutive times, i.e., to criterion. The frontally lesioned animals are markedly deficient in doing this. Again we see that the conditions of reinforcement are relatively ineffective in shaping behavior once the frontal eugranular cortex has been removed, so that the monkeys' behavior is relatively random when compared to that of normal subjects (37). Behavior of the frontally lesioned monkeys thus appears to be minimally controlled by its (repeatedly experienced and therefore expected) consequences: the process of *intention* is impaired.

Should you object to descriptive labels taken from the subjective realm of discourse (on the basis that they must not be applied to animals) this figure (Fig. 22) shows that the results obtained with monkeys also hold for man. These experiments were performed with 20 lobotomized patients and their controls. The procedure was made as alike as possible to that used with the furry primates, and results were remarkably similar (24).

PHASE III. NEUROLOGICAL SIGNIFICANCE OF THE BRAIN-BEHAVIOR RELATONSHIPS

These data led me to define (31) the psychological processes impaired by "association" cortex lesions and to suggest the outlines of a model for these processes. To review the definition elaborated earlier: the posterior system apparently is concerned in the process of selective *attention* (i.e., search and sampling the environment) while the frontal cortex has to do with the process of *intention* (i.e., the guiding of behavior by its expected consequences).

And now, to turn to the model proper: the neurophysiology of selective attention and of intention. The model is, of course, far from being complete. It should, therefore, be accepted with caution and viewed as a progress report and projection of current endeavors.

Cortical Control Over Input: It is appropriate to begin with some

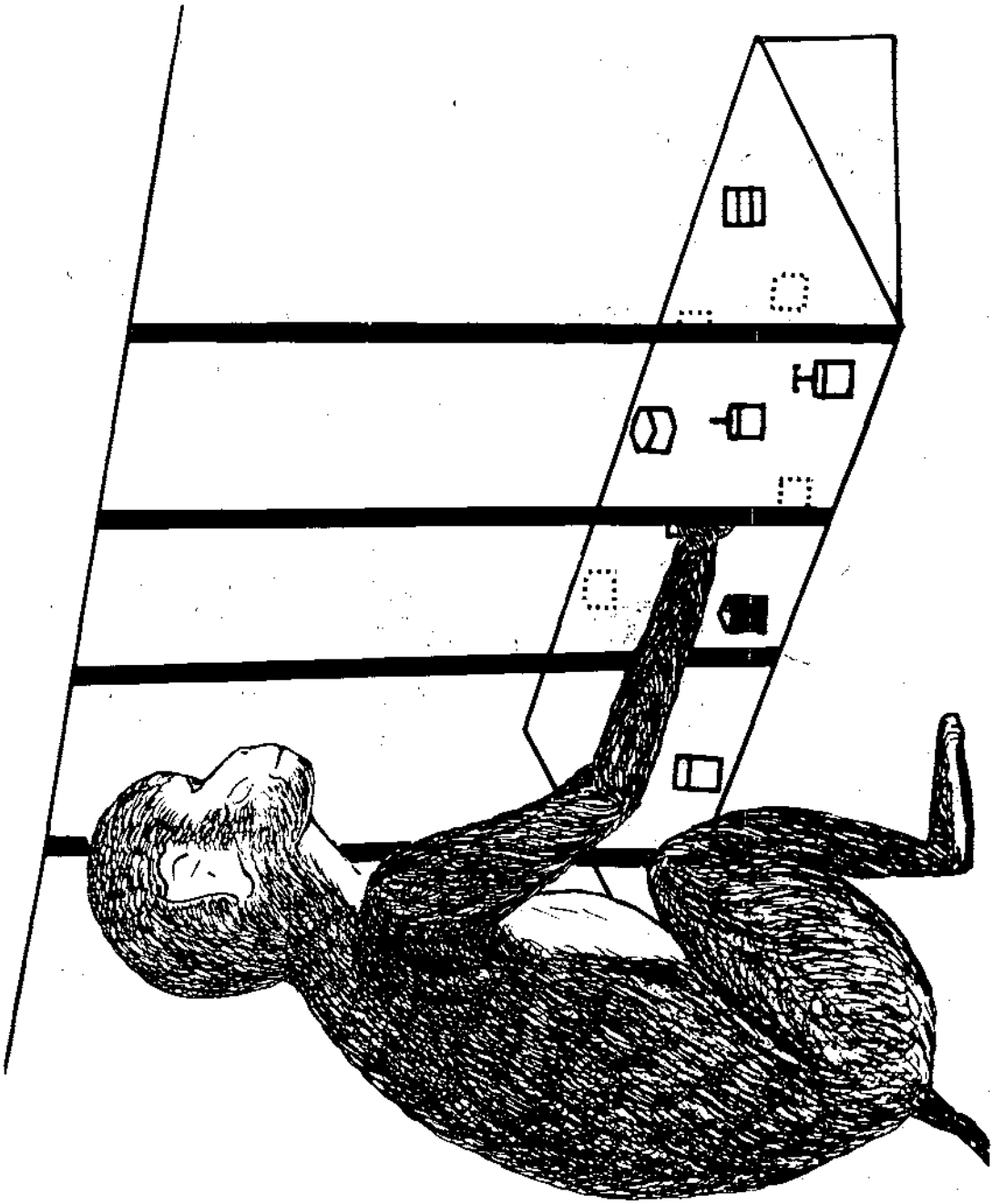

Fig. 1

Modification of Yerkes apparatus for multiple-choice testing.

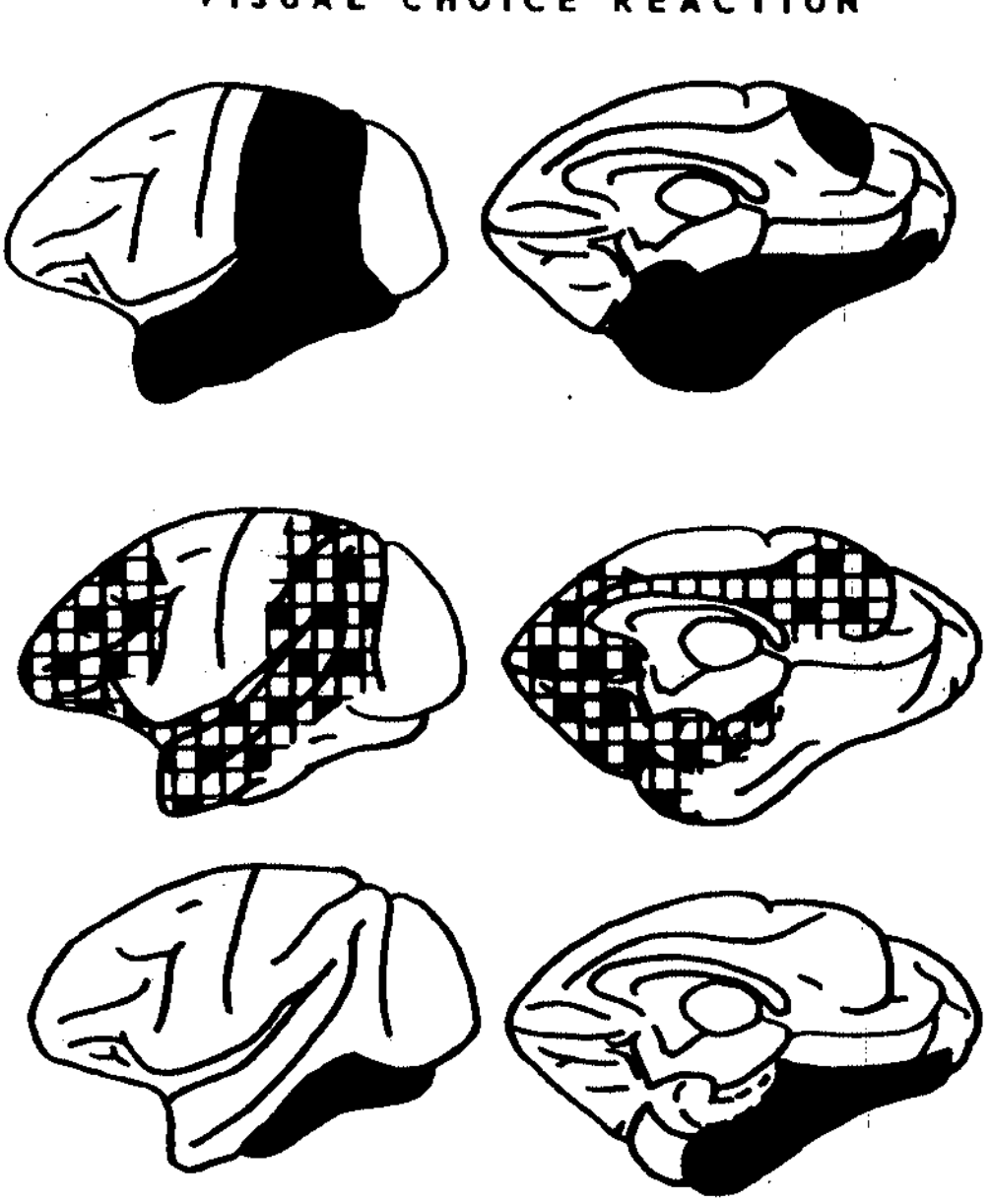

Fig. 2

The upper diagram A 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 C represents the intersect of the area shown in 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.

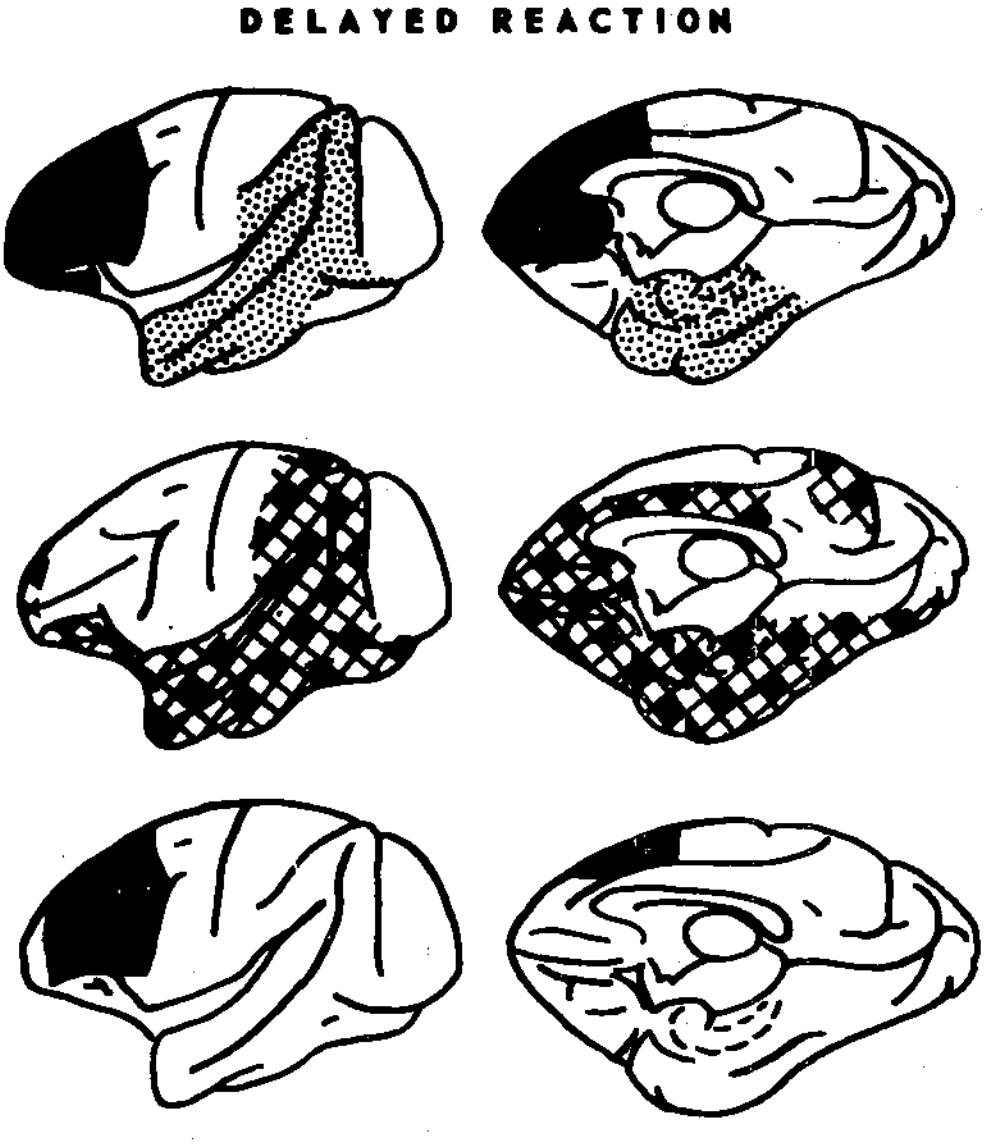

Fig. 3

The upper diagram A represents the sum of the areas of resection of all of the animals grouped as showing a deficit. The middle diagram B represents the sum of the areas of resection of all the animals grouped as showing no deficit. The lower diagram C represents the intersect of the area shown in the upper diagram and that *not* checkerboarded in the middle diagram. This intersect represents the area invariably implicated in delayed reaction performance in these experiments. (Note that resections within the area stippled in the upper diagram occasionally result in "deficit" as defined here. However, note also that a similar "deficit" appears in nonoperate controls. This finding resolves the discrepanices regarding occasional occurrence of deficit on delayed reaction following posterior cortical resections. For the purposes of "localization" procedure, the delayed alternation task appears to be more reliably retained. Nevertheless, as demonstrated here, the results of delayed reaction experiments may still be useful.)

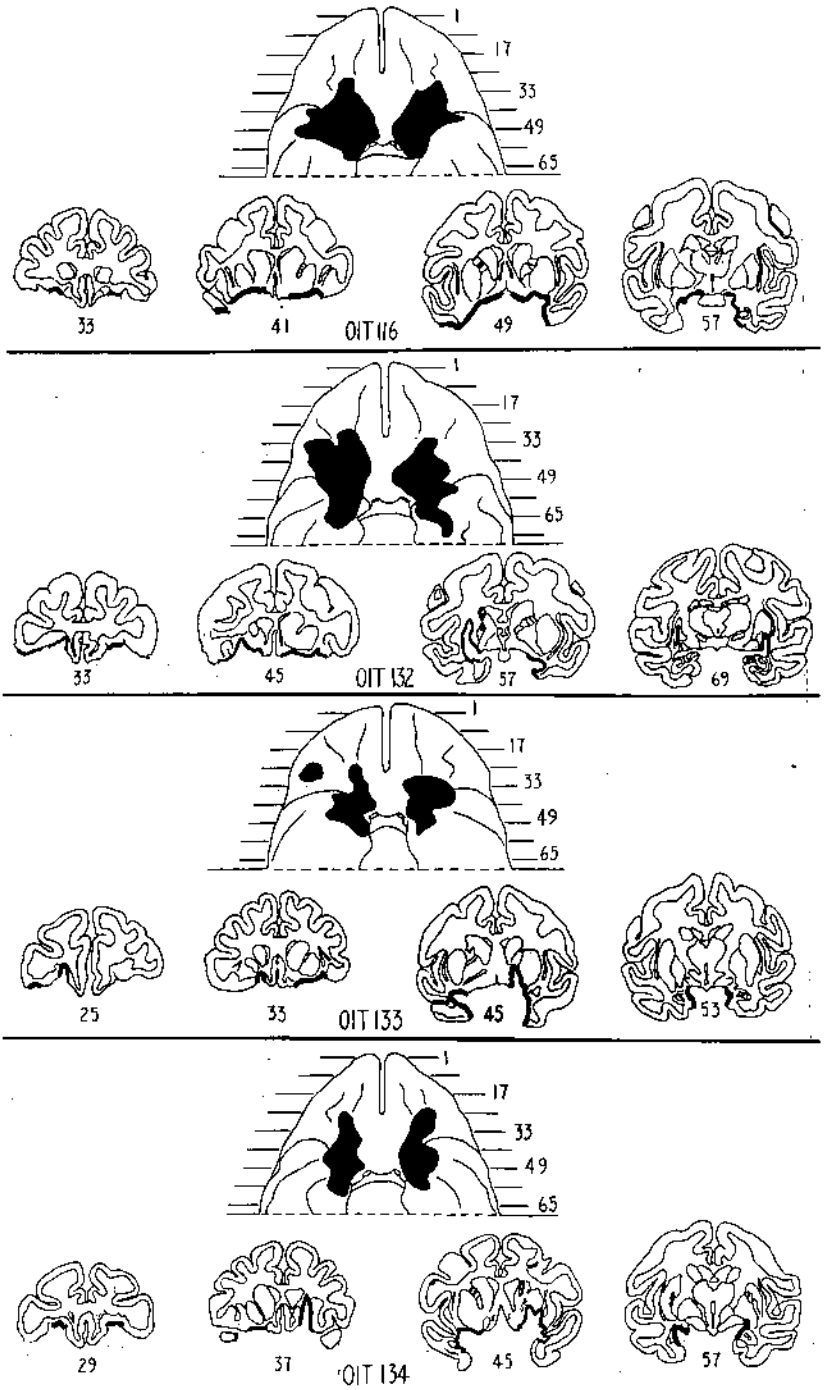

Fig. 4a

Reconstruction of ablation of the orbitoinsulotemporal (left) and medial frontal cingulate (right) cortex.

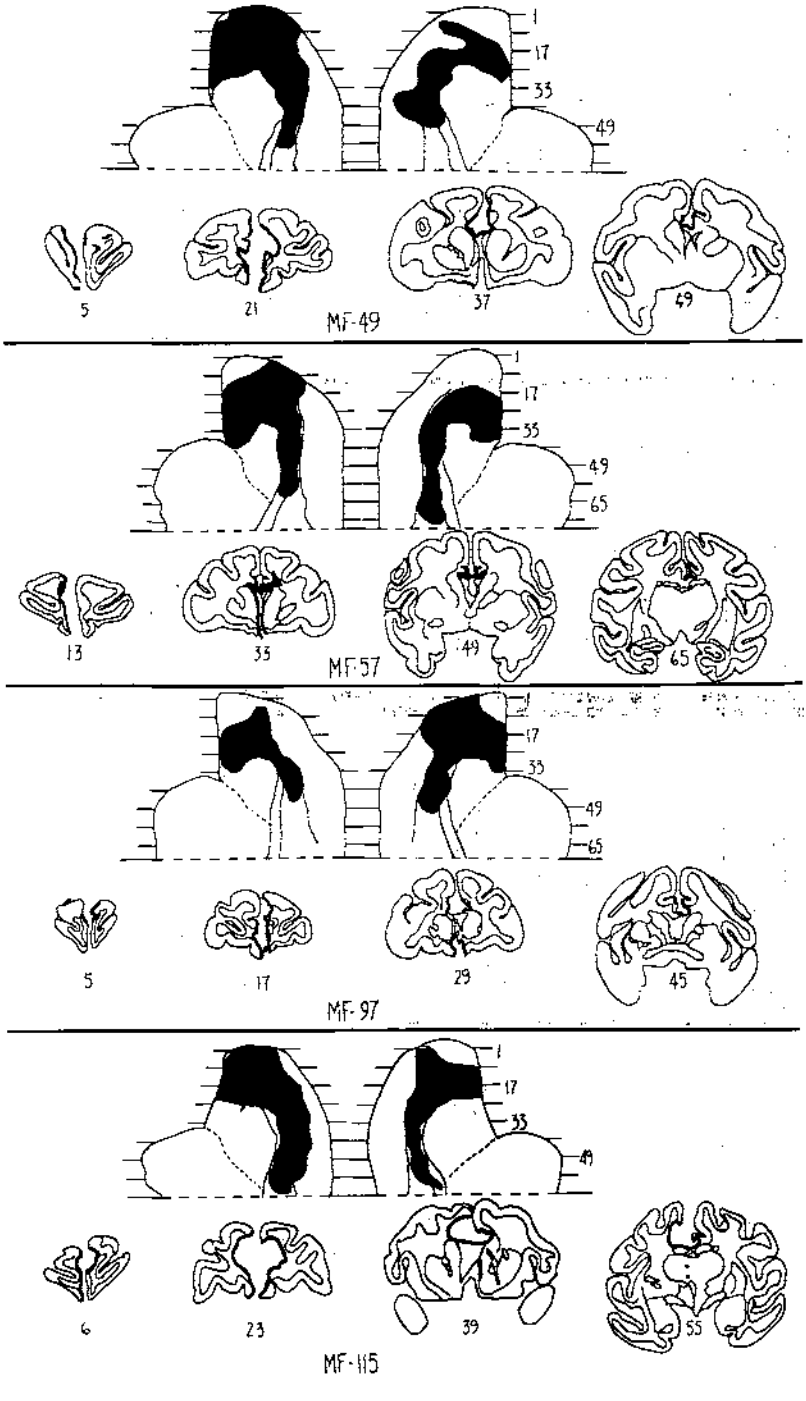
1
17
33
49
5
21
37
49
MF-49
1
17
33
49
65
13
33
49
65
MF-57
17
33
49
65
5
17
29
45
MF-97
1
17
33
49
6
23
39
55
MF-115

Fig. 4b

CLASSICAL ALTERNATION

Animal	Preoperative Learning To Criterion Days	(Errors)	Postoperative Relearning To Criterion Days	(Errors)	Preoperative Savings On Retention Days	(Errors)	Postoperative Savings On Relearning Days	(Errors)
MFC 49	16	(99)	8	(8)	14	(99)	8	(91)
MFC 57	27	(257)	2	(3)	21	(253)	25	(254)
MFC 97	5	(9)	4	(8)	3	(9)	+1	(1)
MFC 115	10	(24)	2	(19)	-2	(0)	8	(+5)
Avg.	14.5	(97)	4	(10)	9.0-62%	(90)-(93%)	10.5-72.4%	(88)-(90.7%)
OIT 116	16	(166)	2	(3)	14	(166)	14	(163)
OIT 132	23	(97)	22	(209)	17	(96)	+1	(-112)
OIT 133	13	(18)	4	(25)	11	(18)	9	(-7)
OIT 134	20	(130)	8	(50)	16	(128)	12	(80)
Avg.	18	(102)	9	(72)	14.5-80.6%	(102)-(100%)	9-50%	(31)-(30.4%)
Total Avg.	16.2	(100)					9.8-60.5%	(59)-(59%)

Fig. 5a

Comparison of the effects of limbic ablations [medial-frontal cingulate (MFC) and orbitoinsulate temporal (OIT)] on classical and go no-go alternation.

GO NO-GO ALTERNATION

Animal	Preoperative Learning To Criterion Days	(Errors)	Postoperative Relearning To Criterion Days	(Errors)	Preoperative Savings On Retention Days	(Errors)	Postoperative Savings On Relearning Days	(Errors)
MFC 49	13	(533)	4	(27)	2	(471)	9	(526)
MFC 57	20	(886)	14	(245)	16	(860)	6	(641)
MFC 97	23	(745)	12	(726)	21	(741)	11	(19)
MFC 115	14	(340)	4	(55)	12	(337)	10	(285)
Avg:	17.5	(631)	8.5	(263)	14.2-81.1%	(602)-(95.4%)	9-51.4%	(368)-(58.3%)
OIT 116	11	(496)	6	(192)	9	(491)	5	(304)
OIT 132	15	(816)	10	(380)	12	(813)	5	(436)
OIT 133	11	(713)	10	(1125)	7	(690)	1	(-412)
OIT 134	18	(618)	10	(800)	16	(615)	8	(-182)
Avg.	13.8	(661)	9	(624)	11-79.7%	(652)-(98.6%)	4.8-34.8%	(36)-(5.4%)
Total Avg.	15.6	(646)					6.9-44.2%	(202)-(31.3%)

Fig. 5b

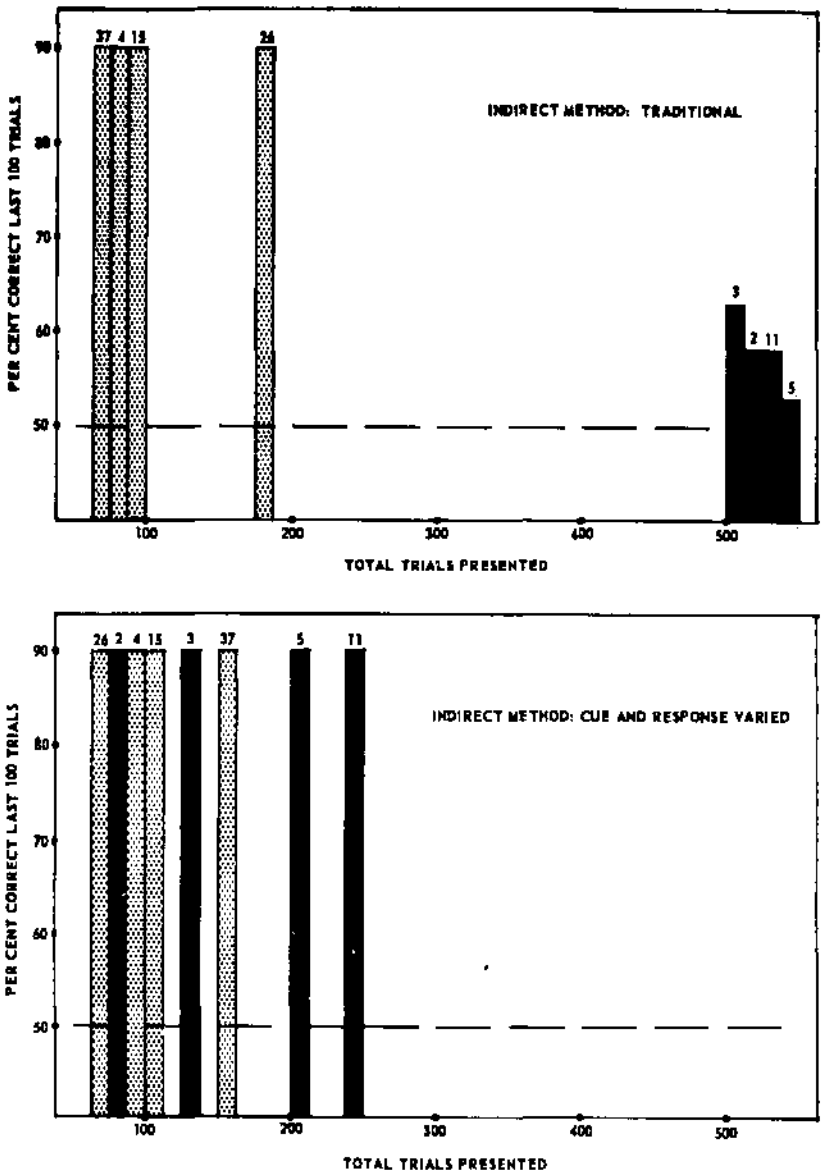

Fig. 6

Comparison of the effects of frontal ablations on classical (upper diagram) and go no-go (lower diagram) alternation.

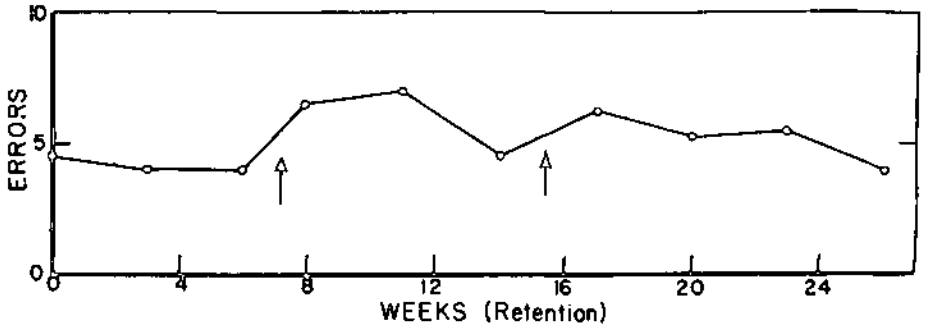

Fig. 7

Performance score on a visual discrimination problem before and after aluminum hydroxide implantation on the inferotemporal cortex. First arrow indicates implantation; second indicates the onset of electrical seizure patterns.

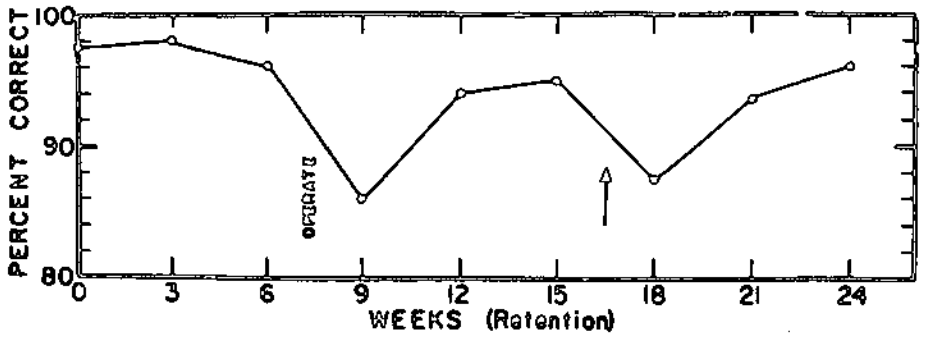

Fig. 8

Same as Fig. 7 except that this illustrates alternation performance and frontal lobe implantation.

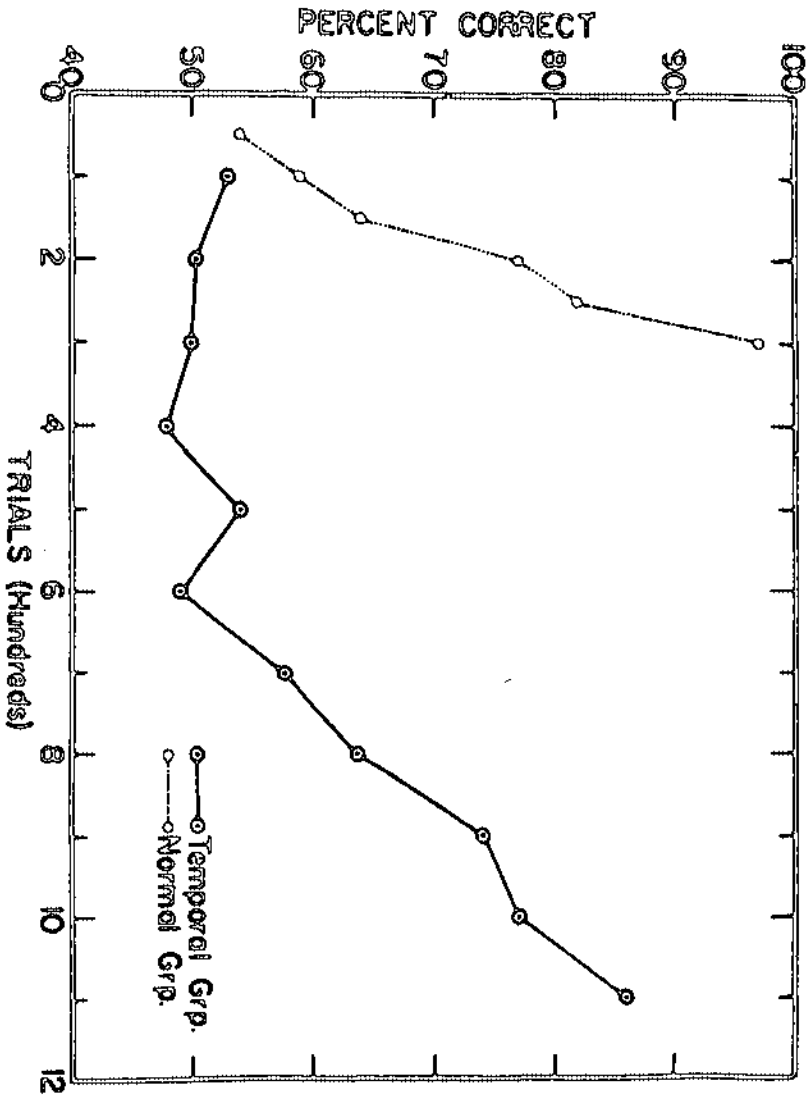

Fig. 9

Visual discrimination of a learning curve obtained from a group of monkeys with electrical seizures recorded from inferotemporal implantation sites.

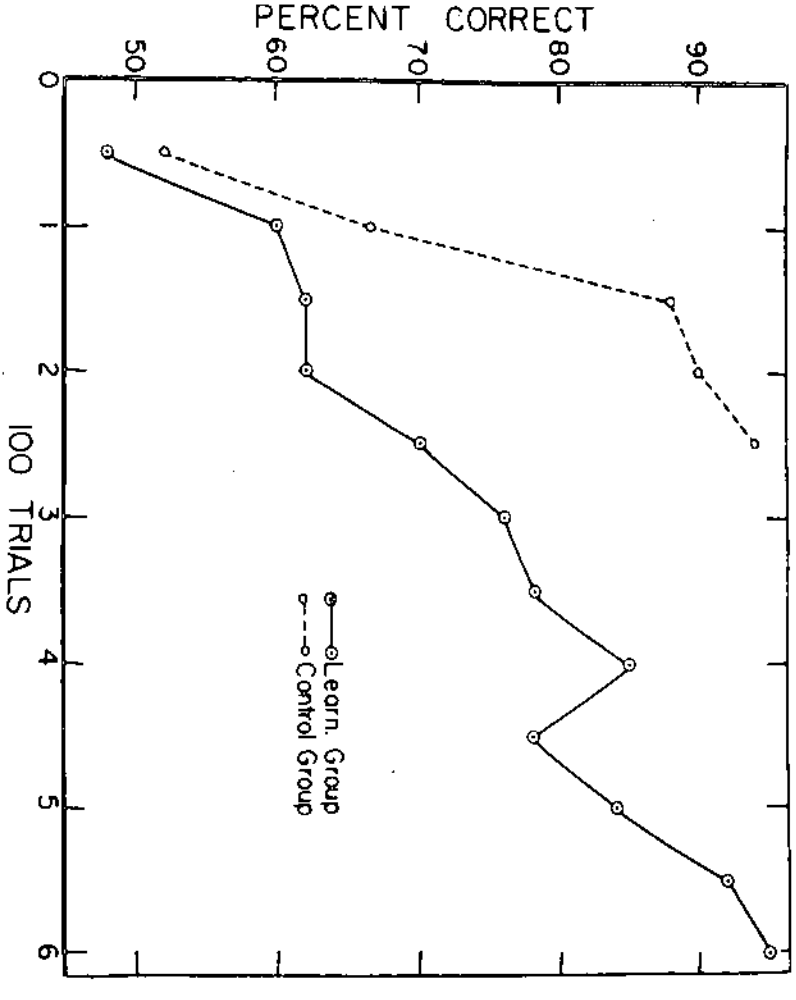

Fig. 10

Alternation learning curve recorded from a group of monkeys with frontal lobe electrical seizures.

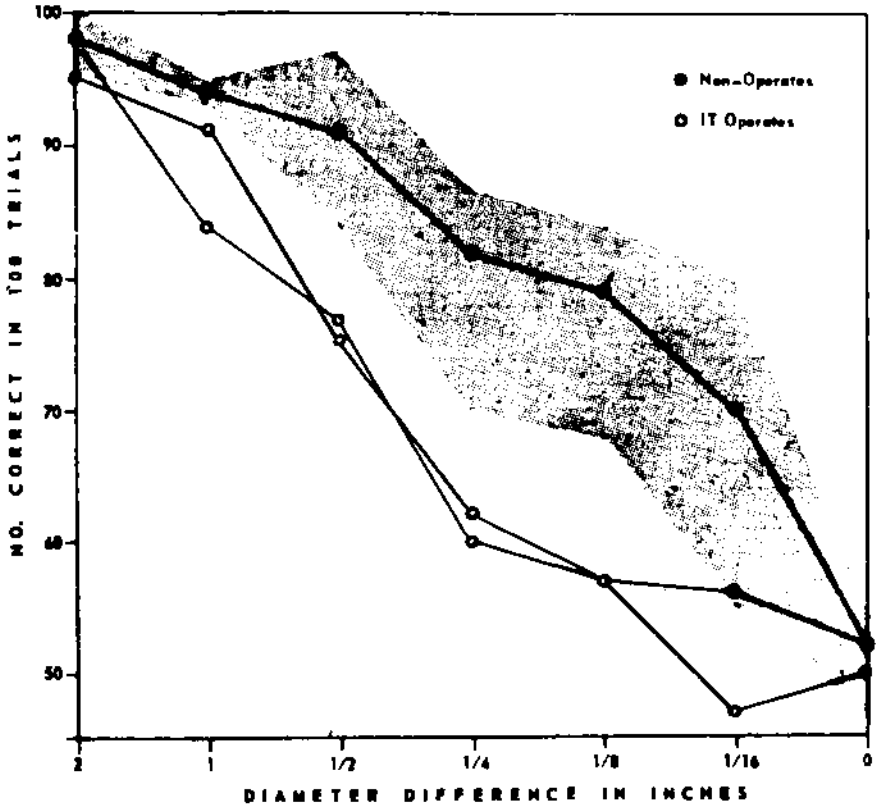

Fig. 11

Difference in performance of inferotemporal and control monkeys on a visual discrimination problem in which size discrimination was varied parametrically.

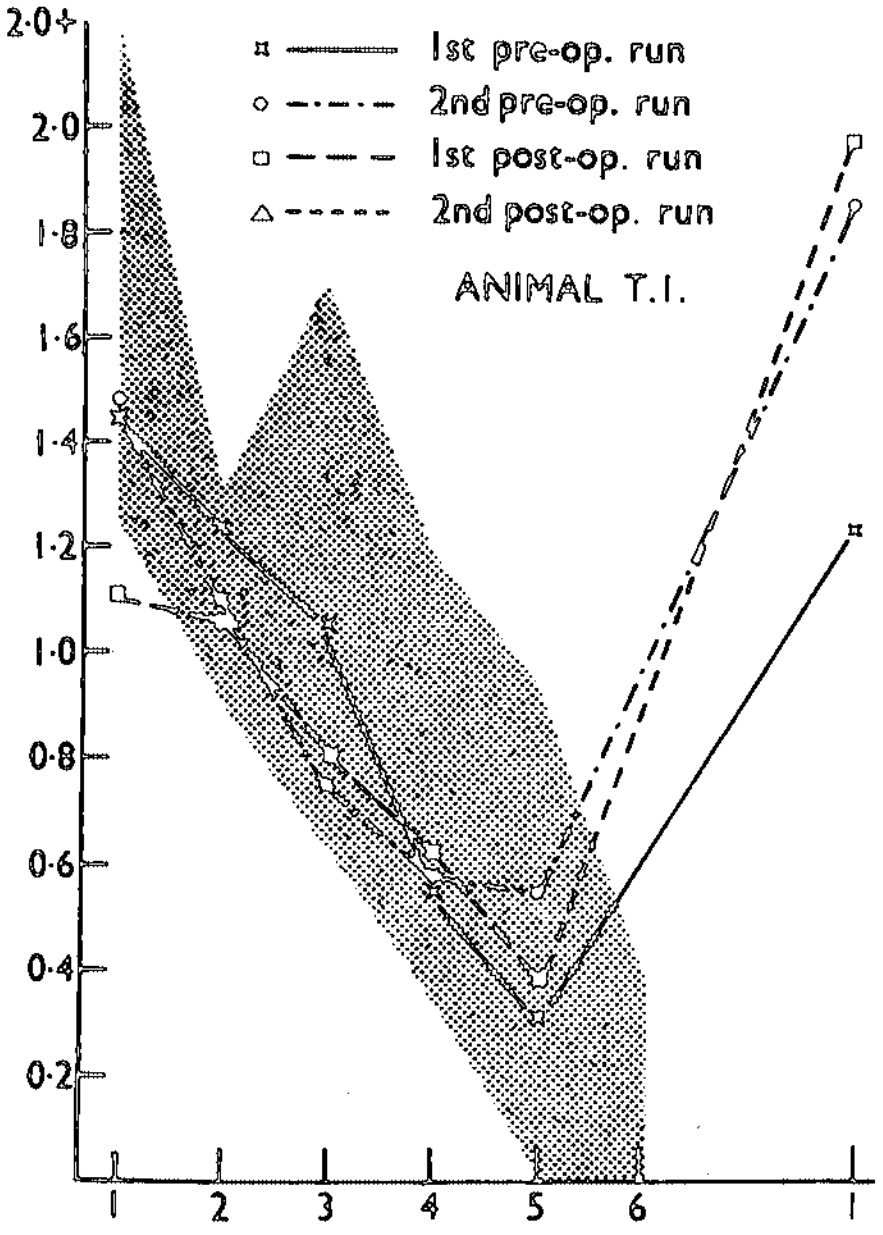

Fig. 12

Single manipulandum performance curves of a single animal in a varying brightness situation. Shaded area indicates variability among groups of four animals.

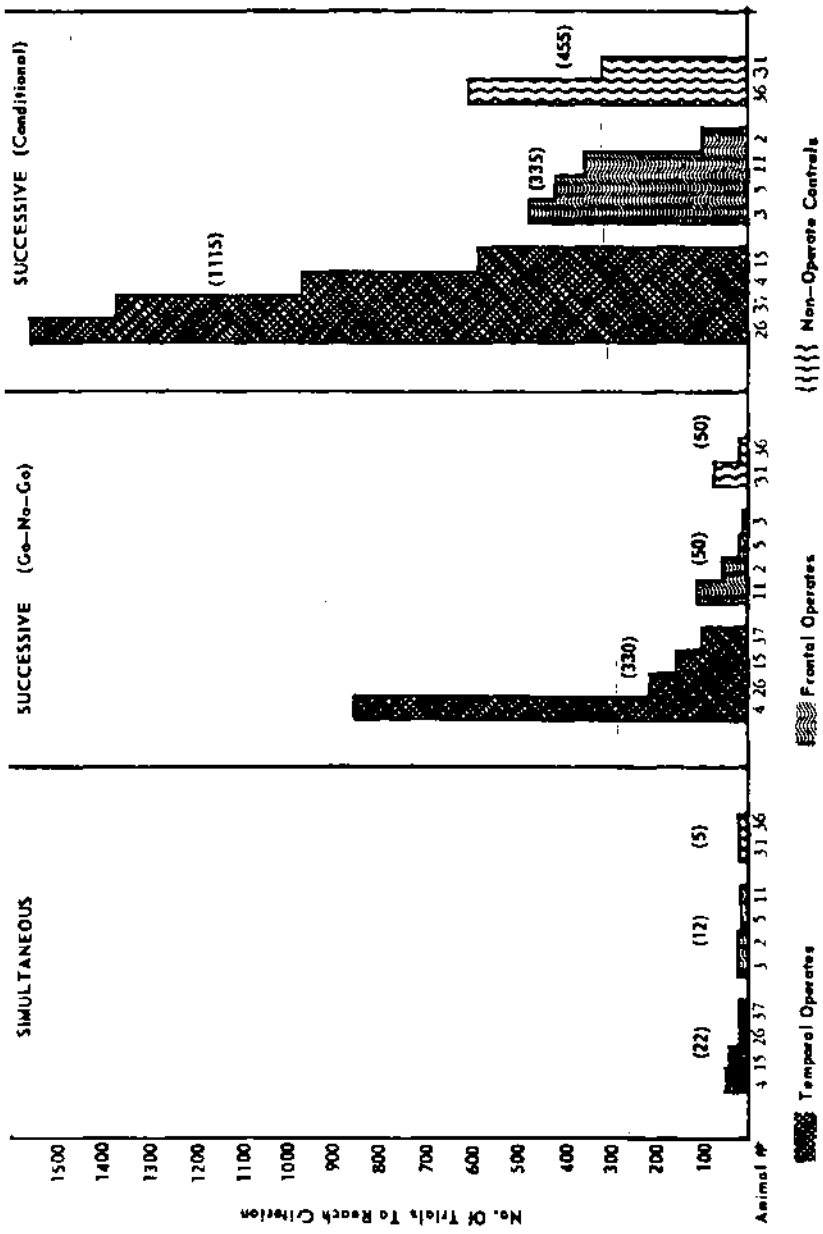

Fig. 13.

Comparison of learning scores on three types of object discrimination by three groups of monkeys. Note that though the cues remain the same, changing the response which was demanded increased the deficit of the inferotemporal groups.

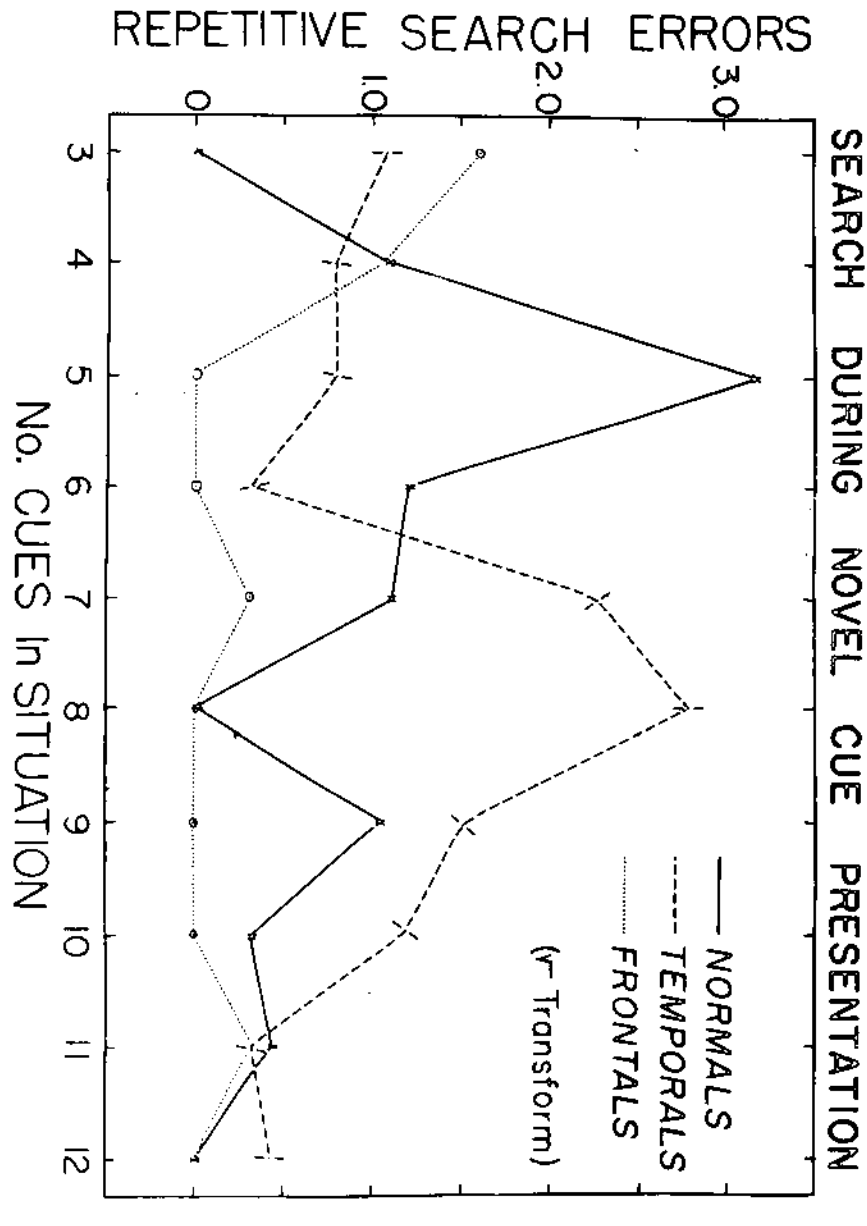

Fig. 14

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

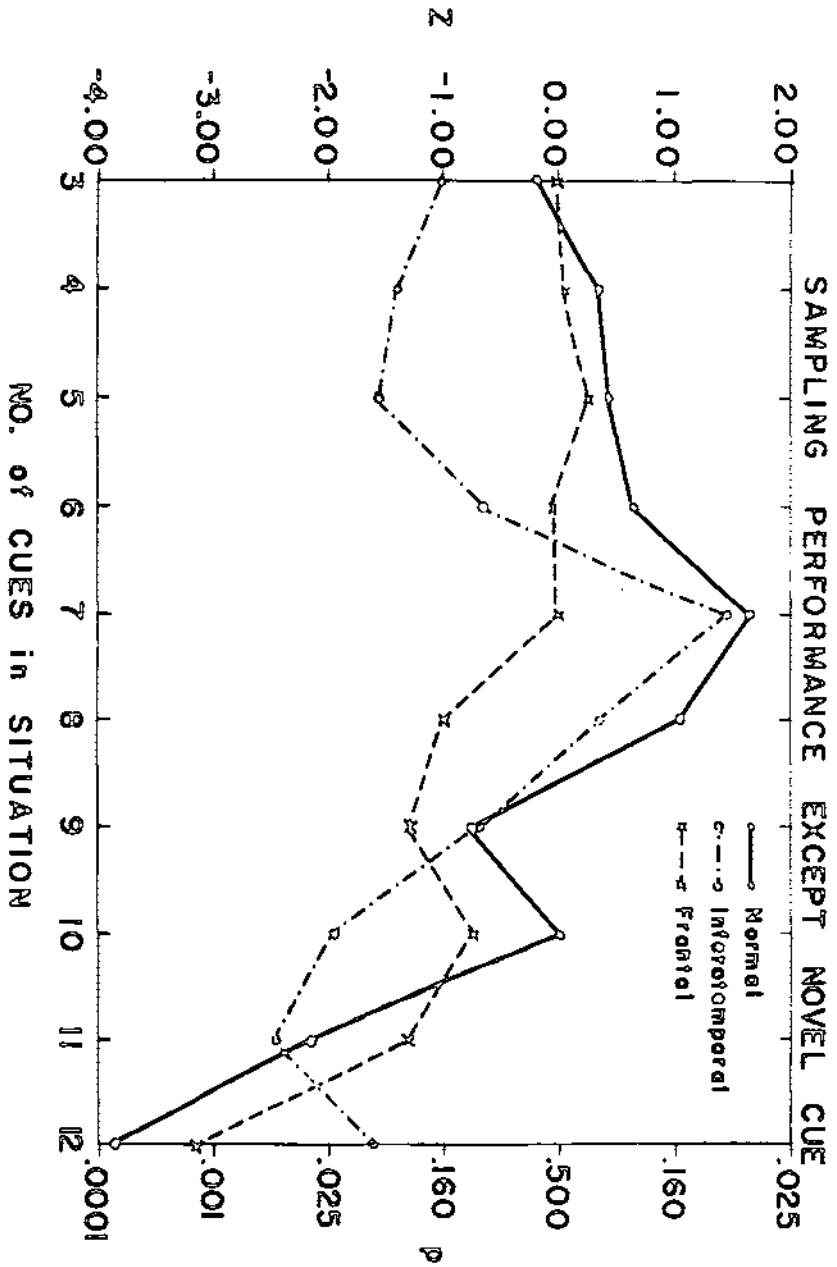

Fig. 15

Graph of the average of the per cent of the total number of objects (cues) that are sampled 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 non-parametric Mann-Whitney U procedure).

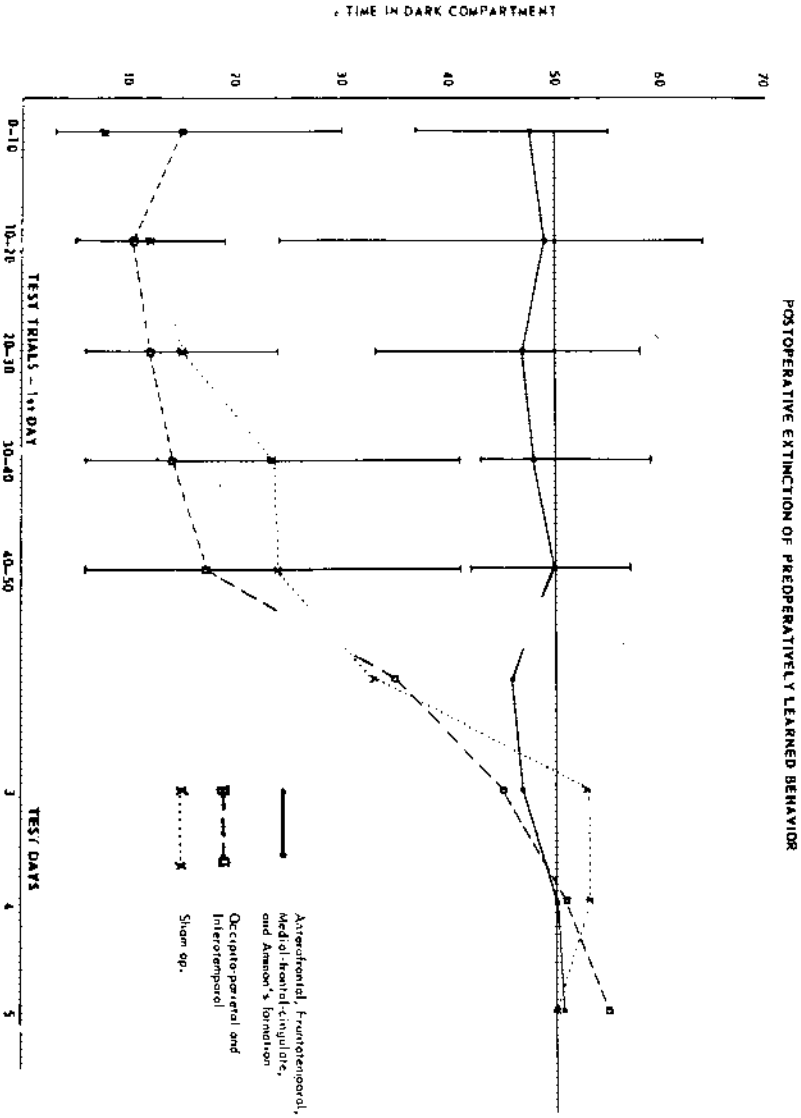

Fig. 16

Performance of limbic and nonlimbically ablated monkeys during postoperative extinction of a *pre*-operatively learned conditioned avoidance. Note that limbic and frontally-operated monkeys behave alike.

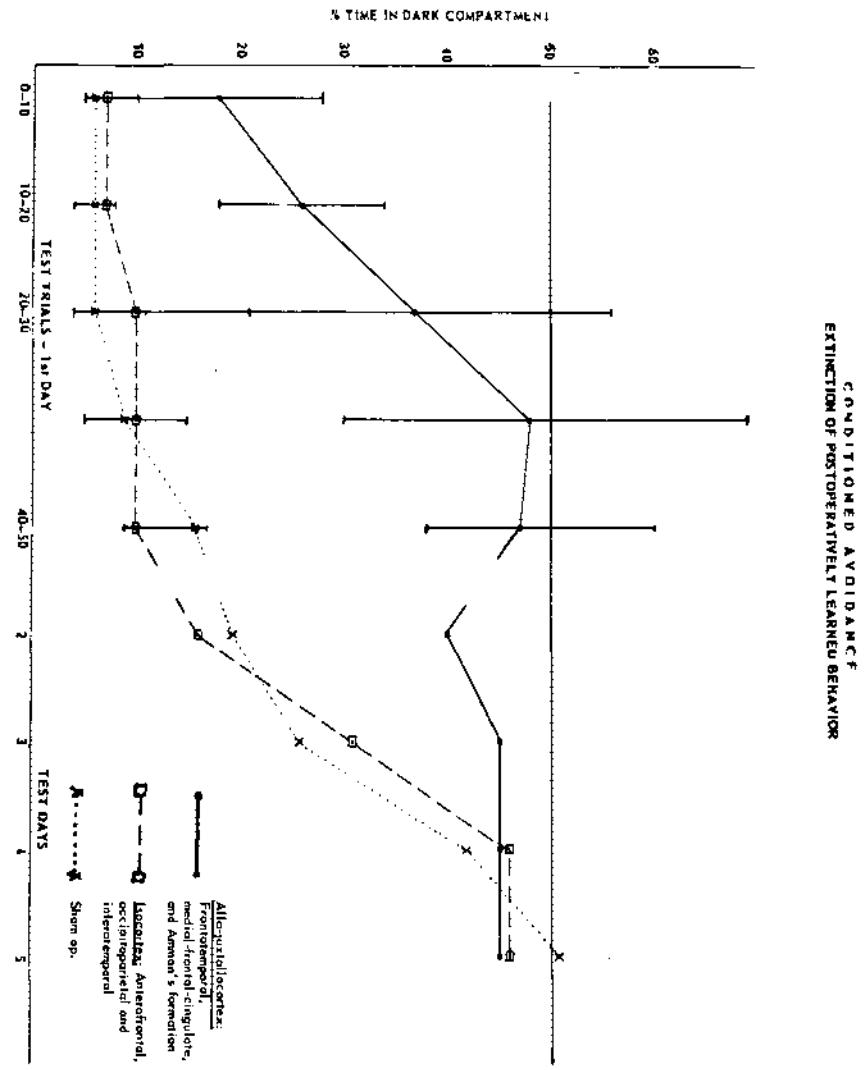

Fig. 17

Performance of limbic and nonlimbically ablated monkeys during post-operative extinction of a *post*-operatively learned conditioned avoidance. Note that the limbic groups are clearly separated out by this procedure.

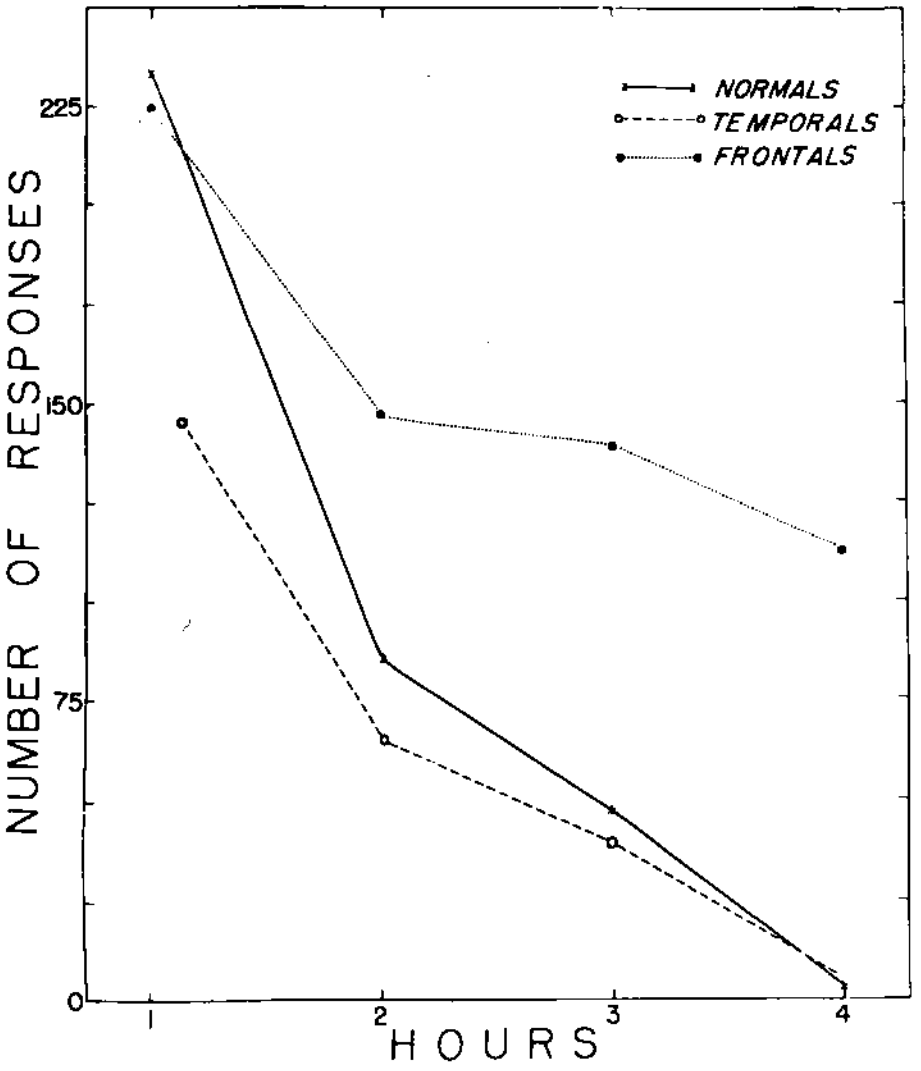

Fig. 18

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

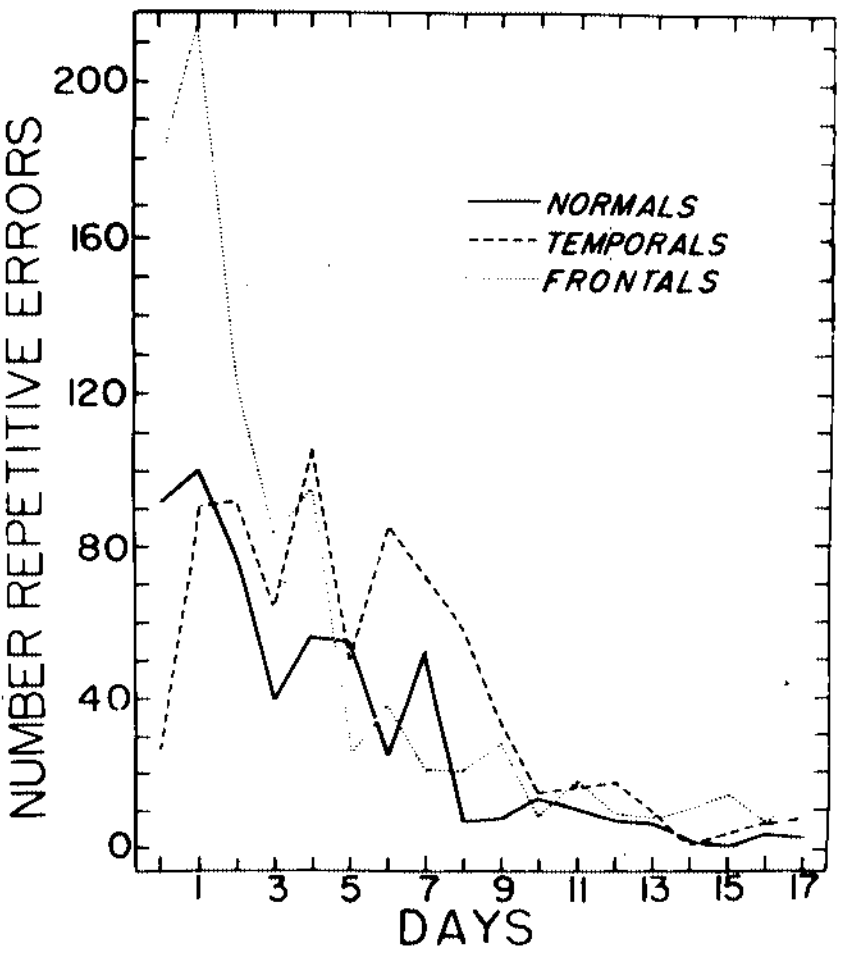

Fig. 19

Graph showing the differences in the number of repetitive errors made by groups of monkeys in a go no-go type of delayed reaction experiment. Especially during the initial trials, frontally operated animals repeatedly return to the food well after exposure to the "nonrewarded" predelay cue. Note, however, that this variation of the delay problem is mastered easily by the frontally operated group.

PERCENTAGE OF ALTERNATION AS A FUNCTION OF RESPONSE AND OUTCOME OF PRECEDING TRIAL

S	Preceding Trial[a]			
	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] A, alternated; NA, did not alternate; R, was rewarded; NR, was not rewarded.

Fig. 20

Comparison of the performance of frontally ablated and normal monkeys on alternations made subsequent to reinforced (R) and non-reinforced (NR), and an alternated (A) and non-alternated (NA) response.

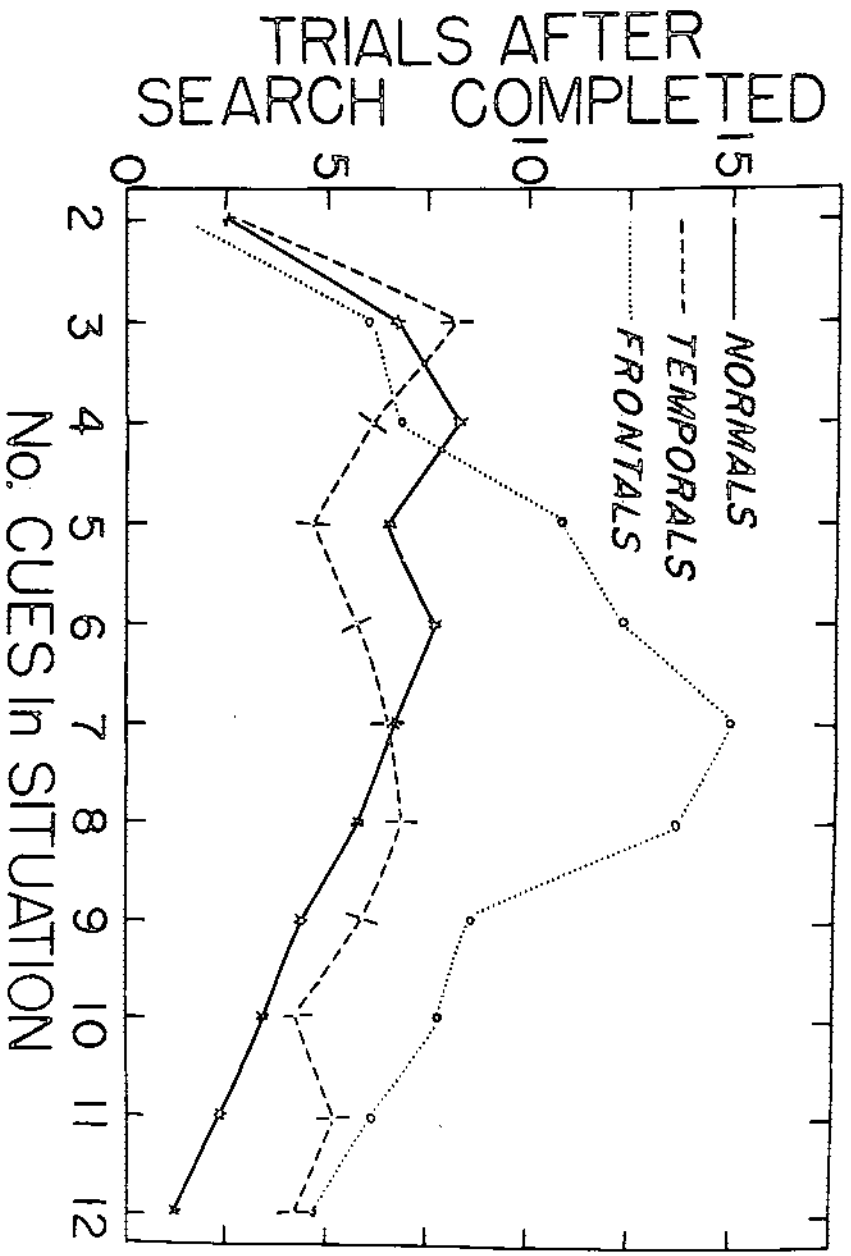

Fig. 21

Graph of the average of the number of trials to criterion taken in the multiple object experiment by each of the groups in each of the situations after search was completed, i.e., after the first correct response. Note the difference between the curves for the controls and for the frontally operated group, a difference which 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.

Performance of Lobotomized and Control Ss Who Completed The Tast

	L+	C+
Mean Total Responses	625.9	359.2
Mean Average Search Responses Per Program on Non-New-Cue Programs	8.8	5.9
Mean Average Search Responses Per Program on New-Cue Programs	6.1	4.2
Mean Average Post-Search Responses per Program on Non-New-Cue Programs	24.7	12.9
Mean Average Post-Search Responses per Program on New-Cue Programs	3.7	5.9

Fig. 22

Comparison of performance of lobotomized and normal humans in a multiple-choice experiment. L+ indicates those lobotomized subjects who completed the test; C+ the controls who did so.

	Animal	3 vs. 8	R vs. G	3 vs. 8
Crosshatch	158	380	82	0
	159	180	100	0
	161	580	50	0
	166	130	0	0
Undercut	163	[1014]	100	300
	164	[1030]	200	[500]
	167	704	50	0
	168	[1030]	150	[500]
Normal	160	280	100	0
	162	180	100	0
	165	280	100	0
	170	350	100	0

Fig. 23

Comparison of the effects of undercutting and crosshatching inferotemporal cortex of monkeys on their performance in several discriminations.

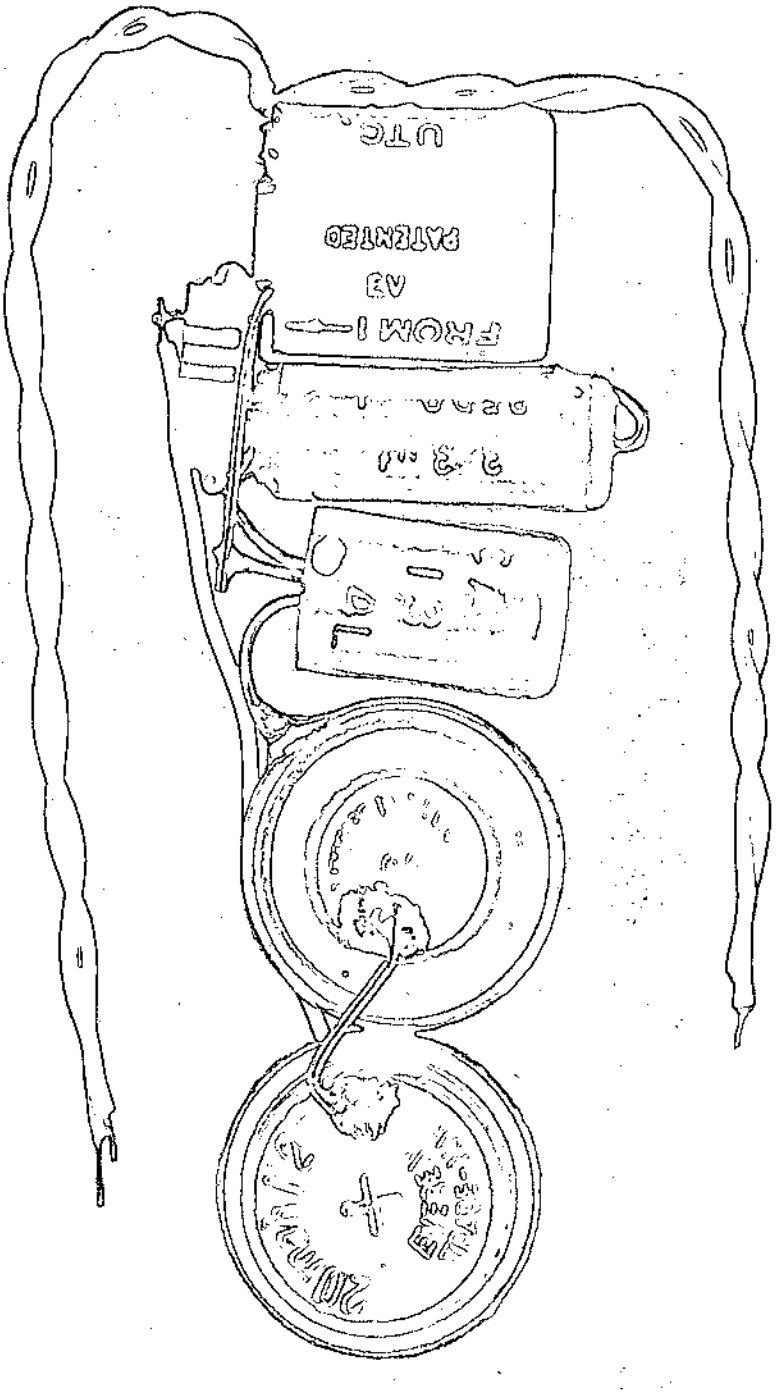

Fig. 24

Stimulator and batteries for chronic brain stimulation. Batteries are rechargable nickel-cadmium and are available in different sizes from the manufacturer.

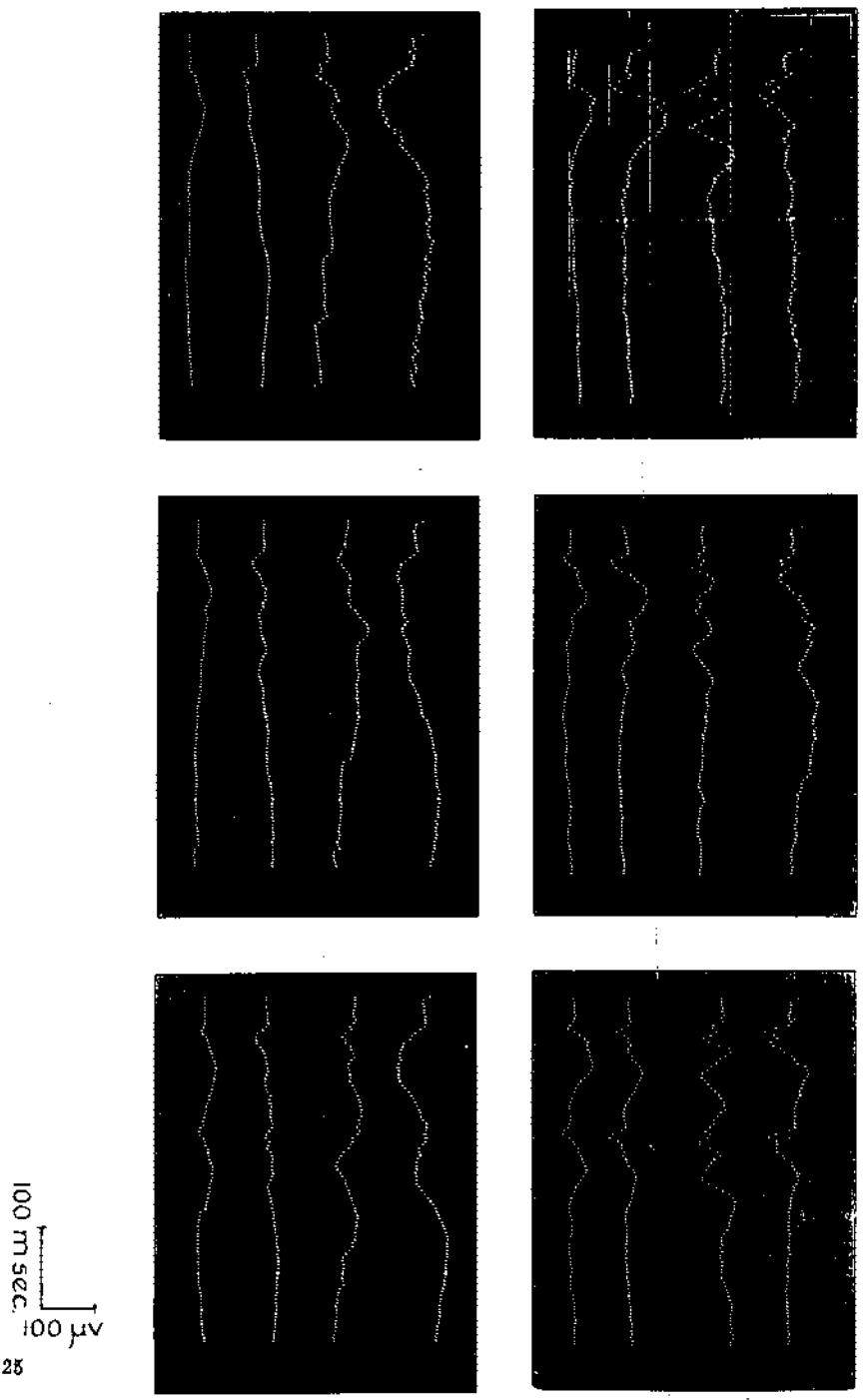

Fig. 25

A representative record of the change produced in visual evoked responses by chronic stimulation of the inferotemporal cortex. Upper set of records was taken before stimulation; lower set, during stimulation. All traces were recorded from the visual cortex; the first set in response to a single flash, the second to flashes separated by 75 msec. and the third to flashes separated by 150 msec. Actually this was the first of our series of experiments which called our attention to the changed recovery phenomenon. Note here also the change in wave form of the response even when a single flash was presented. However, this change did not appear in all of our subjects.

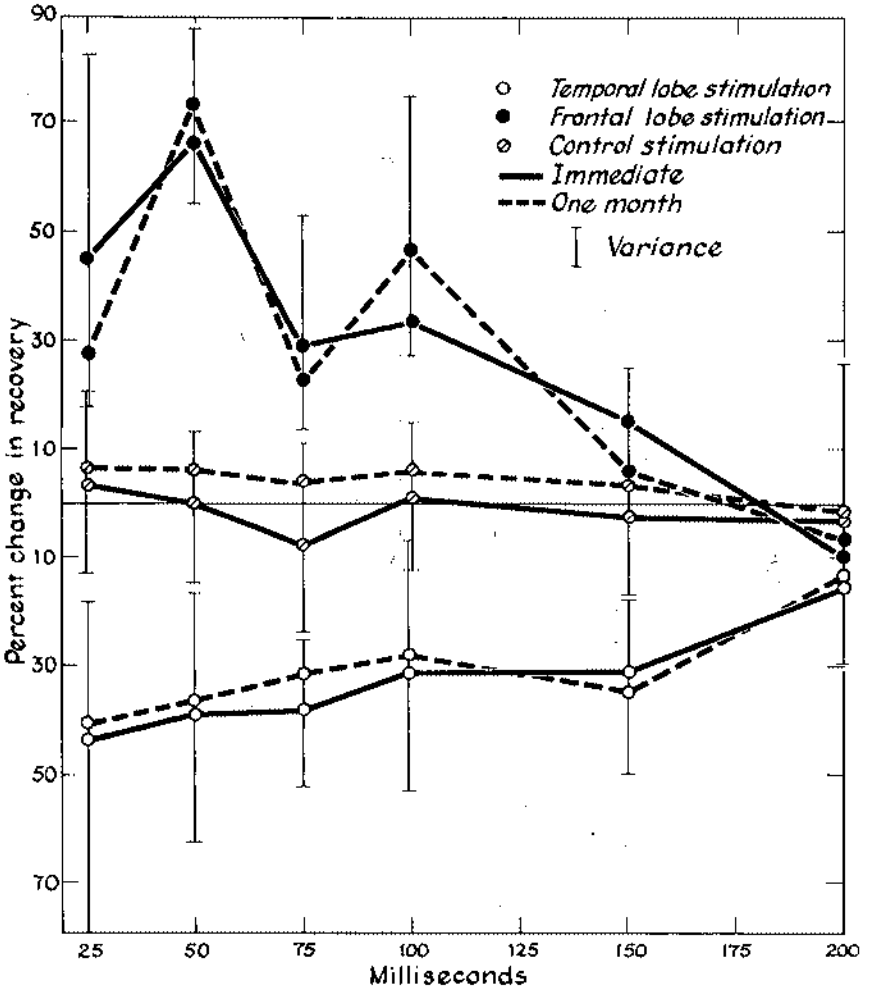

Fig. 26

A plot of the recovery functions obtained in 12 monkeys before and during cortical stimulation.

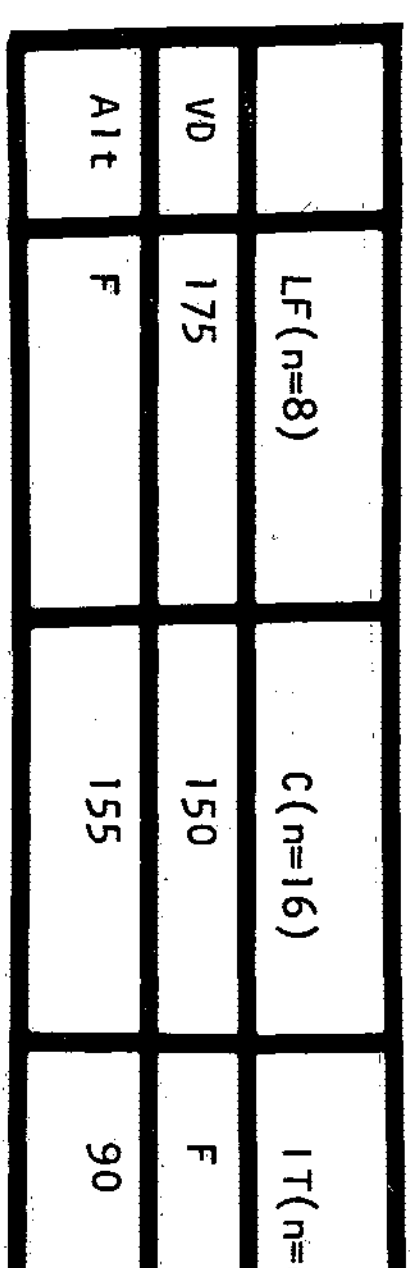

Fig. 27

Comparison of the effects of "lateral frontal" (LF) and "inferotemporal" (IT) and control subjects (C) on learning of visual discrimination (VD) and alternation (ALT). Scores are average number of errors made; F means failure in 1000 trials; n = number of subjects in group.

facts — or rather lack of facts — about the neuroanatomical relationships of the inferotemporal cortex. There is a dearth of neurological evidence linking this cortex to the known visual system, the geniculostriate system. There are no definitive anatomical inputs specific to the inrerotemporal cortex from the visual cortex or the geniculate nucleus. Of course, connections can be traced via fibers that synapse twice in the preoccipital region; but connections also exist which connect the visual cortex to the parietal lobe, whose excision results in no change in visual behavior (as you have seen). In addition, circumsection of the striate cortex does not impair visual discrimination (5). Further evidence that these "corticocortical" connections are *not* the important ones can be seen from the following experiment. I performed (Fig. 23) a crosshatch of the inferotemporal cortex much as Sperry had done earlier for the striate cortex (50) and found no deficit either in visual learning or in performance. On the other hand, undercutting the inferotemporal cortex makes a vast difference: it precludes both learning and performance of visual tasks. This suggests that the relationships of this cortex essential to visual behavior must come from somewhere below — though large, deep-dipping U fibers are not yet ruled out.

However, another proposal can be tested, viz., that the essential relations of the posterior association cortex are centrifugal, efferent (29). And there is anatomical evidence to suggest and support such a notion. Some time ago, two brains with inferotemporal resections were studied by Dr. Walle Nauta in his laboratory. These showed an efferent tract leading to the region of the superior colliculus ending either within its substance or in the surrounding reticular formation (23). No such fibers could be traced to the lateral geniculate nucleus. In support of this finding is a report by Kuypers who has also traced temporo-collicular fibers in monkey (14). The idea of an efferent mechanism "gating," or otherwise "partitioning," the input to the geniculostriate system has some backing as an explanation for the process of selective attention. How would an efferent mechanism of this sort work? To find out we performed the following experiment:

Instead of making ablations or implanting an epileptogenic lesion, we now chronically and continuously stimulate the brain. These experiments are still in progress and are being accomplished in collaboration with Dr. D. N. Spinelli, a physiologist who designed the stimulator (Fig. 24) and the recording equipment we are using (51). The stimulator is sufficiently small so that it can be implanted under the scalp. It puts out a square-wave bidirectional pulse 1 msec. in duration, about 3 V in amplitude. The frequency of stimulation is approximately 8-10/sec. The batteries that drive the stimulator are rechargeable.

Records were made in the awake monkey (Fig. 25). Paired flashes are presented and recordings are made from electrodes implanted in the occipital cortex. The response to 50 such paired flashes are accumulated on a Computer for Average Transients. The flash-flash interval is varied from 25 to 200 msec. All are records 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 has begun. Note that with concurrent cortical stimulation the recovery function is depressed — i.e., recovery is delayed.

Figure 26 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 was done in collaboration with Dr. James Dewson (9). 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 had shown that its removal impairs complex auditory discrimination (speech sounds), leaving simple auditory discriminations (pitch, loudness) intact (8). Removal, in addition, alters paired click recovery cycles recorded as far peripherally as the cochlear nucleus. Bilateral ablation shortens the recovery cycle markedly. And, 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.

The Model: These results allow us to specify a model. On the basis of the neurobehavioral and neuroanatomical data I had earlier suggested (31) that the posterior "association" cortex, by way of efferent tracts leading to the brain stem (most likely to the colliculi or surrounding reticular formation (29)), partitions the events that occur in the sensory specific system and classifies these events according to one or another scheme. During the course of our joint work, Dr. Spinelli would repeatedly ask: "What do you mean by 'partitioning'? What is 'partitioning' in neuro-logical terms?" Until we had accomplished these electrophysiological experiments, I really had no idea just how to answer. But once we saw the results of these experiments, the neurophysiological explanation became evident: 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 the concurrent input. Each of a successive series of inputs will thus 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 (1). Ordinarily a particular input excites a great number of fibers in the channel, insuring replication of information transmission. Just as lateral inhibition in the retina has the effect of reducing redundancy (3), so the operation of the "association" cortex enhances the density of information within the input channel.

Implications of The Model: This model has several important implications. *First*, the non-recovered cells, the ones that are still occupied by excitation initiated by prior inputs, will act as context- or short-term memory against which the current input is matched. A match-mismatch operation of operation of this sort is demanded by models of the process of recognition and selective attention spelled out on other occasions by Craik (7), Sokolov (49), Bruner (4), MacKay (15) and myself (32, 34,

35). These "occupied" cells thus form the matrix of "uncertainty" that shapes the pattern of potential information, i.e., the "expectancy" which determines the selection of input signals which might or might not occur.

Second, in a system of fixed size, redundancy reduction increases the amount of correlation possible with the set of external inputs to the system (13) — i.e., the number of alternatives, the complexity of items, to which an organism can attend is enhanced. This internal alteration in the functional structure of the classical sensory projection system thus allows attention to vary as a function of the spatial resolution which excitations can achieve, with the result that events of greater complexity can be attended to. The more the resolution, the sharper the "uncertainty" and, thus, the more likely that any set of inputs will be sampled for information. In the extremes, this sharpening of the appetite for information becomes what the clinical neurologist calls stimulus-binding. Its opposite is agnosia — the blurring of uncertainty due to the simplification of the structure of the channel after damage to the "association" area which leads to an organism's inability to seek information.

Third, this corticofugal model of the functions of the so-called association systems relieves us of the problem of 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. The problem of the homunculus is, of course, an extremely interesting one. Former ways of looking at the input-output relationships of the brain have come up against the problem of an infinite regression (implicit or explicit) of "little men"-inside-"little men": "homunculi" associating sensations, abstracting from these associations and passing these abstractions on to the motor systems for action. Somewhere along the line of regress awareness comes in, perhaps in yet another anatomically separable system. And then, of course, there is "awareness of awareness...."

According to the model presented here, there is no need for such 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 sensory-specific associated systems and the frontolimbic systems exert their effects by altering the functional organization of the primary systems. Thus these *associated* systems are not "association" systems; they simply alter the configuration of input-output relationships processed by the projection systems. In computer language the associated systems function by supplying *subroutines* in a hierarchy of programs, subroutines contained within and not superimposed above the more fundamental processes. In this fashion the infinite abstractive regress is avoided. One could argue that in its place a downward regress of sub- and sub-subroutines is substituted. I would answer that this type of regress, through progressive differentiation, is the more understandable and manipulable of the two.

Concretely, the posterior association cortex is conceived to program, to structure an input channel, perhaps through action on inhibitory collaterals within the channel. The effect of such action is to alter the

speed of recovery of neurons in the channel once they are excited by inputs. And by means of the operation of such a simple device, information processing, sampling of the environment, selective attention "automatically" follows.

Another 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 — which is altered. Thus, the same signal carries more or less information, depending on the "width" of the channel. And I am here tempted to extrapolate, and say that the signal carries different meanings depending on the particular structure or organization of the redundancy of the channel.

The Neurophysiology of Reinforcement: I wish I could, at this time, present an equally rigorous neurophysiological model for the process of intention. But here we are a considerably greater distance from a precisely-stated model. True, the process of reinforcement enhances redundancy (12). And in part, the operations of the frontolimbic systems and that of the sensory-specific systems tend to balance one another. Chronic concurrent stimulation of frontal and some limbic structures does enhance redundancy in the visual channel (51a).

Also, monkeys with inferotemporal ablations tend to perform better on the alternation tasks which are so disturbed when frontolimbic lesions are made.

But the converse does not hold (Fig. 27), and this suggests that the change resulting from frontal ablation is in some respects different from that produced by inferotemporal stimulation. Perhaps this difference lies in the fact that the amount of redundancy *per se* is an insufficient measure of its efficacy (e.g., in minimizing error). The form of pattern of the redundancy is crucial. Mere repetition is an ineffective form; redundancy is not a measure of simplicity. Rather, when properly used, redundancy is not strictly opposed to information (or uncertainty) but becomes an additional dimension of complexity (13).

Clearly, then, the structure of redundancy, its temporal pattern, is the key to the neurophysiological model of intentional behavior. As such it will most likely deal with temporal resolution of events, the temporal structure of behavior. Outlines of this structure have been formulated but experiments have not as yet been accomplished to detail it sufficiently to permit the model to become actualized in strict neurological terms (17, 32, 34).

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