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BRAIN FUNCTION AND LEARNING

EDITORS

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NEUROPHYSIOLOGY AND LEARNING

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II. A "MODEL NEURAL SYSTEM" APPROACH TO THE NEURAL BASIS OF BEHAVIORAL CHANGE

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I. MEMORY AND THE ORGANIZATION OF ATTENTION

Pribram: The full title of my talk should be "Remembering and the Organization of Attention and Intention: The Case History of a Model." There are, of course, other models—other ways of handling the data I shall present —but right now I feel my model to be the best available. To show why, I would like to present a great deal of the data on which this model is built, for many of you have never had the opportunity to see as a whole the material gathered by my colleagues and myself. I shall organize my argument into three questions. Most of the work pertaining to the first question was done some years ago; most of the work related to the last question is now in progress.

First, 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.

Second, what is the psychological meaning of the brain-behavior relationships uncovered?

Third, what is the neurophysiological meaning of these brain-behavior relationships? By this I mean, what is a plausible model that would account for them? This last aspect is, of course, the most interesting, but in order to develop it properly I have to answer the other two questions first.

THE BRAIN-BEHAVIOR RELATIONSHIP

At the time I began this work, around 1946, there were two vast expanses of the brain cortex which were essentially silent to experimental manipula-

tion: the posterior "association" cortex and the frontolimbic systems. We knew of no physiological function to assign to them, and we did not know, though conjectures had been abundant on the basis of clinical and anatomical relationships, what their function in behavior might be. Therefore the first subquestion was, how could we best proceed to desilence these brain areas?

The primary, though not the sole, physiological-anatomical technique used in this early phase of the work was the ablation method, checked histologically. As shown in Figure 18, serial reconstructions of the lesion were always made after sacrifice of the animal, and the depth and relation of thalamus or other structures were outlined whenever possible. Figure 19 shows some examples of reconstructions: a lesion of the hippocampus, showing the sparing (what was not removed at surgery); also shown is the extent of surface lesion.

The two areas I will be most concerned with, the inferotemporal region and the dorsolateral frontal region, are illustrated in Figure 20. With few exceptions, the subjects of the experiment are primates, mostly *Macacus rhesus*; experiments using man will be mentioned as well, but here reconstruction of lesions is of course not feasible.

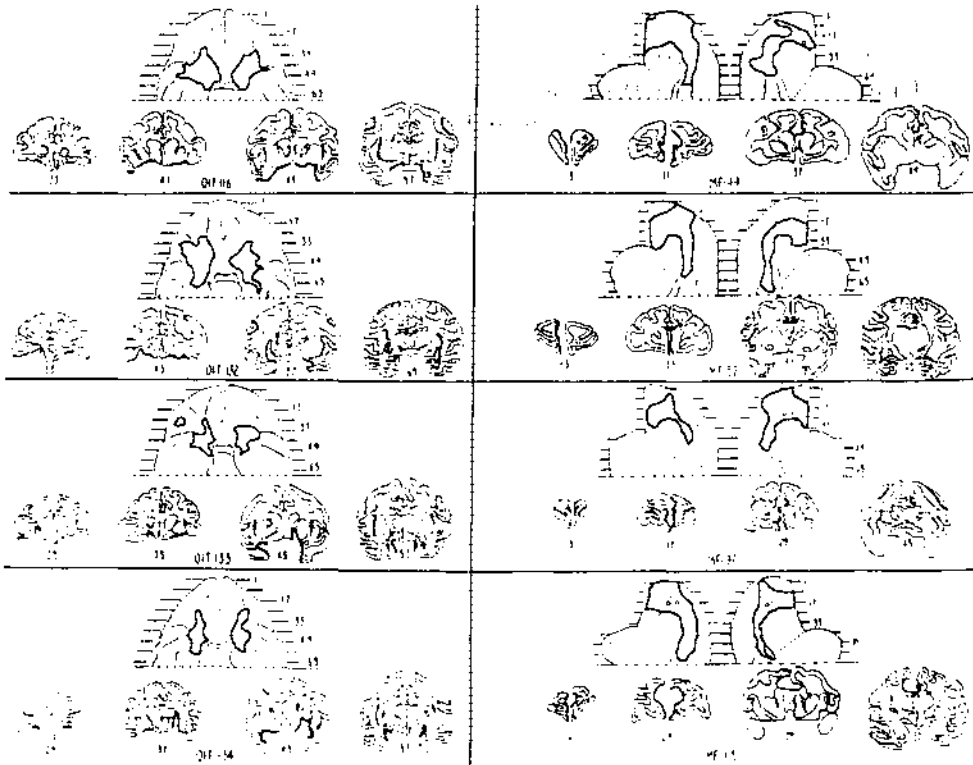Combined with cortical removals was an extensive behavioral survey of

Figure 18. Reconstruction of ablation of the orbitoinsulotemporal *(left)* and medial frontal cingulate *(right)* cortex. (From Pribram, Lim et al., 60.)
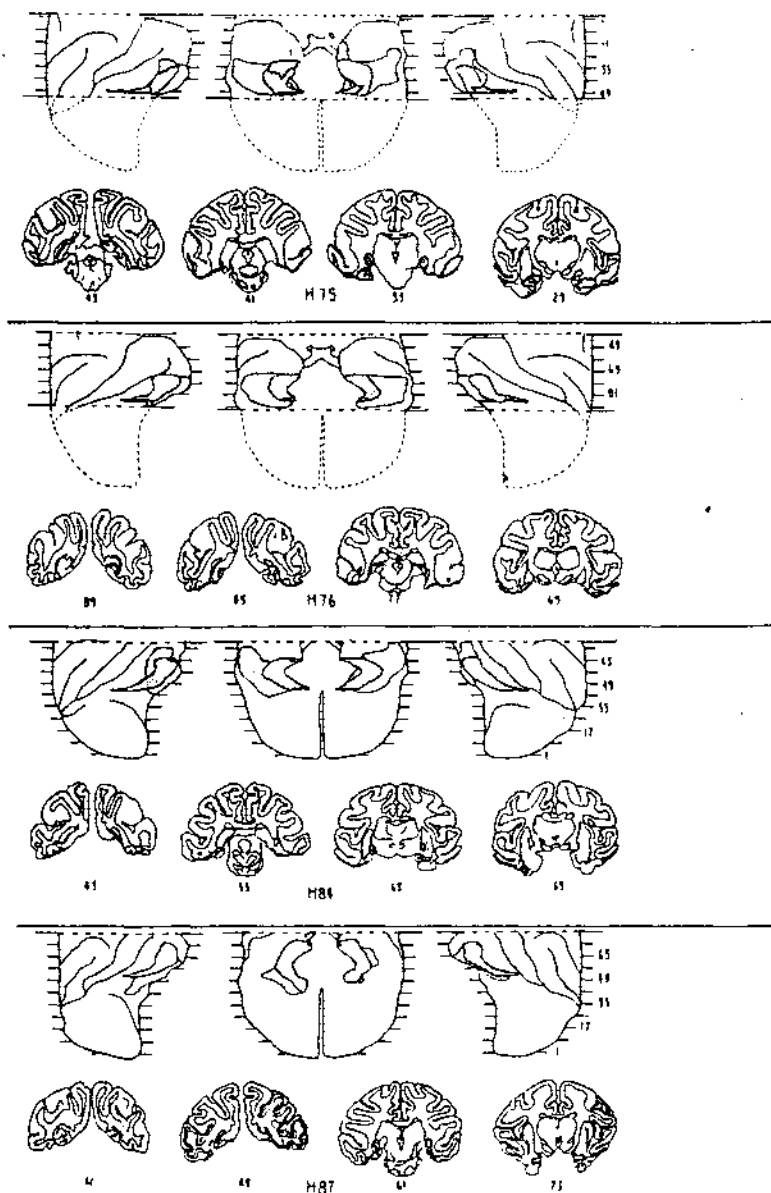
Figure 19. Reconstruction of hippocampal ablation. (From Bagshaw, Kimble & Pribram, 2.)

the subject, both pre- and postoperatively. A variety of behavioral techniques was used. Figure 21 shows a shuttle box in which conditioned avoidance behavior was studied. In Figure 22 we see an operant conditioning situation in which the monkey is taught to lever-press; its pressing rate can be controlled by simple cues and by programming the reinforcement. Figure 23 is an example of the Yerkes box in which monkeys can be taught to make visual choices between two alternatives. Figure 24 demonstrates a multiple-choice procedure which I devised. It is a modification of the Yerkes box; a
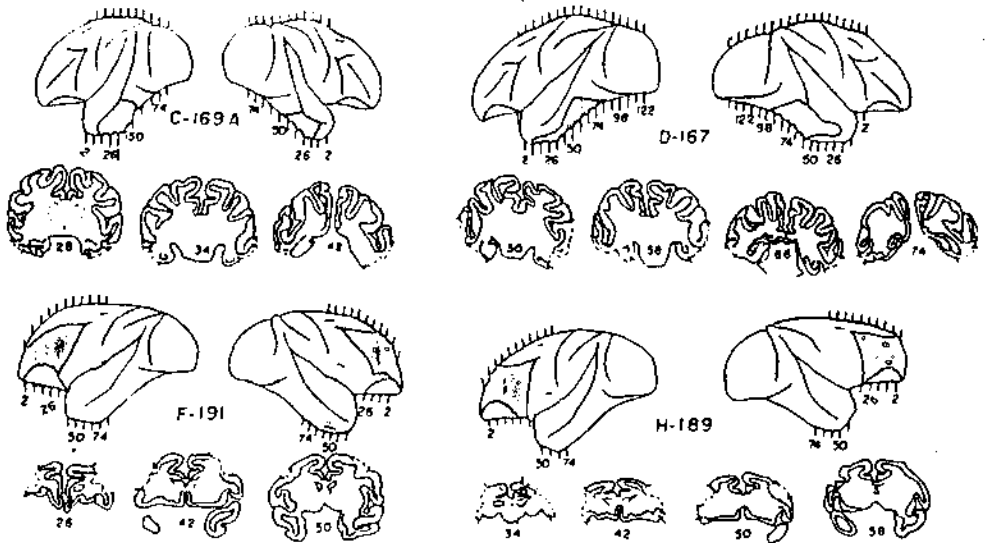
Figure 20. Reconstructions of ablations of the inferotemporal (*above*) and dorsolateral frontal (*below*) cortex. (From Pribram, 50.)

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.

A further modification (Figure 25) shows our present setup. The multiple choice procedure has been automated and is programmed by a special purpose computer called the DADTA (Discrimination Apparatus for Discrete Trial Analysis) machine (56, 57). This device allows us to perform a great variety of behavioral tasks and saves much effort. 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 involved in the situation, so there is reliability comparable to that obtained with operant equipment. Furthermore, 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. We can, for instance, program a sequence so the subjects must respond to 1, 3, 5, 7 in that order, before they receive a reward. There are a variety of problems that can be presented (57), and I shall describe some of these.

Figure 21. Shuttle box used for conditioned avoidance experiments. Upon shock from floor or other noxious stimulus, monkey moves from left to right box, eventually learns to respond to CS preceding shock and thus avoid unpleasant stimulus.
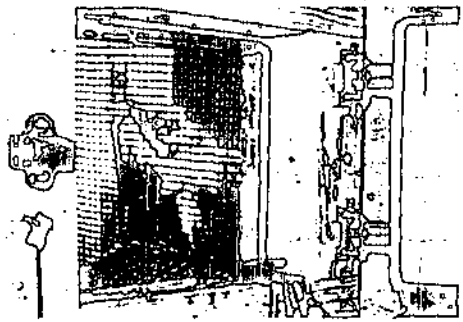
Figure 22. An operant conditioning apparatus (Skinner box). Press-lever or other manipulanda together with stimulus cues and reward source shown at back of box.

Just to give an example of the power of this instrument, in the old hand-operated Yerkes box, when a sophisticated animal—one having been trained for several years—is asked to discriminate between the numeral 3 and the numeral 8, it will probably fail to master this in 1000 trials. With the DADTA, a completely naive animal, directly out of the jungle, takes an average of 250 trials, which is five days of training. This is an unexpected dividend.

Grant: Besides Yerkes, Klüver (24) should be given credit for the apparatus.

Pribram: All right, Yerkes, Klüver, then Harlow. There is a current tendency among scientists to attribute a test or device to the latest of its inventors rather than to the earliest. We usually call the crude hand-operated device a "Wisconsin" apparatus.

Grant: I think there is a dramatic difference between Klüver's way of doing it and Yerkes', and it is a very important one. Animals can be readily trained with the Klüver version and not with Yerkes', for example.

Pribram: That is an important technical point. I would also like to point out that the DADTA machine is even better. Let us skip for the moment whose apparatus it is and ask what makes it better. There may be two reasons. One is that it is much more fun to work, with all the clicks and clatter. 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,

Figure 23. Yerkes (Wisconsin General) testing apparatus used to evaluate discrimination and alternation learning and performance. Monkey in cage faces apparatus but is separated from it by opaque screen; on each trial stimulus objects and manipulanda are changed and moved to within its reach on a sliding track and the screen is lifted: animal responds and a new trial begins. Note stimulus objects and manipulanda in lower left and right corners.
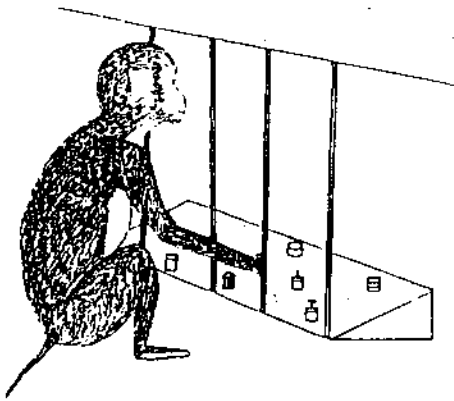
Figure 24. Modification of Yerkes apparatus for multiple-choice testing. (From Pribram, 47.)

therefore, no position habits to confound 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 they may be irrelevant. By initially changing position from trial to trial, we immediately alert the subject to the fact that position is irrelevant. That is why the DADTA machine is a more effective tool for teaching discriminations. Another factor may be that in the hand-operated apparatus there is a screen interposed between subject and cues. This screen goes up and down; it is distracting and may interfere with testing. The automated apparatus has no screen; the lights that illuminate the cues just go out. We are designing a new version which will be programmed by a small general purpose computer (PDP-8) which will both operate the display and record the behavior. The technology has advanced sufficiently so that within a year we should be able to type in the particular task that we wish to display to the animal for solution, instead of having to dial it in, as we do now. This will give us still more flexibility in the choice

Figure 25. The automated form of the multiple choice apparatus (Discrimination Apparatus for Discrete Trial Analysis, DADTA). (From Pribram, Gardner et al., 56.)

of tasks. And, of course, with the general purpose computer on line, we will hasten our initial data processing as well.

The experiments to be reported 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 mention that these experiments stand alone, and have not as yet been checked either by someone working with me at some time or another, or in other laboratories such as Harlow's.

The first question, then, is how to establish and characterize the brain-behavior relationships. To do this, I devised a technique which is called the method of "the intersect of sums" (44). What I did was to take the first 40 animals and list separately those that had a postoperative deficit on a particular problem (such as a visual choice reaction), and those without such deficit. By a deficit is meant either failure to perform the task at criterion in 1000 trials, or to learn it in the number of trials taken to learn the task preoperatively (in other words, no savings). My criterion, arbitrarily chosen, is 90 correct out of 100 consecutive responses; all I have to say in its defense is that it worked. There are of course other criteria—for retention, for instance—and these serve other purposes.

The work summarized in Figure 26 included initially some hippocampal lesions; I felt at the time that I had invaded neighboring structures, and subsequently more precisely placed lesions showed the hippocampus uninvolved in the retention of simple visual discrimination performances. The method of intersect of sums was then applied in this fashion: plots were made of all the lesions that produced deficit (Figure 26A); of all the lesions with no deficit (Figure 26B); the two were superimposed (Figure 26C). The remaining cortex, the inferotemporal region, is the crucial cortical area concerned in visual choice behavior. No other portion of the "silent" cortex is involved. As I mentioned, there were 40 animals in this initial phase of the study. Since then this finding has been replicated many more times. We have now probably close to 1500 monkeys in the total series, a goodly number of which have had the inferotemporal lesion.

Using this method to pinpoint an effect, we next asked the question, what characterizes the brain-behavior relationship? One way of stating the problem is to ask what it is that is localized. Is there a "center" for "biological intelligence"? Is there one for "visual-somatic" space? Is there one for "sensory associations", and so on? We did not ask the question in that form at all. We asked, more simply, what we would find if we extended the "intersect of sums" technique to include other tasks. We discovered that the posterior portion of the silent areas was divisible into regions, each of which served one or another modality. There is modality specificity within this posterior "association" cortex. An example is seen in Table 3. One group of animals was given a parietal lesion; another an inferotemporal lesion. They were tested either for original postoperative learning (Group A) or for postoperative retention of a preoperatively learned task (Group B). The two
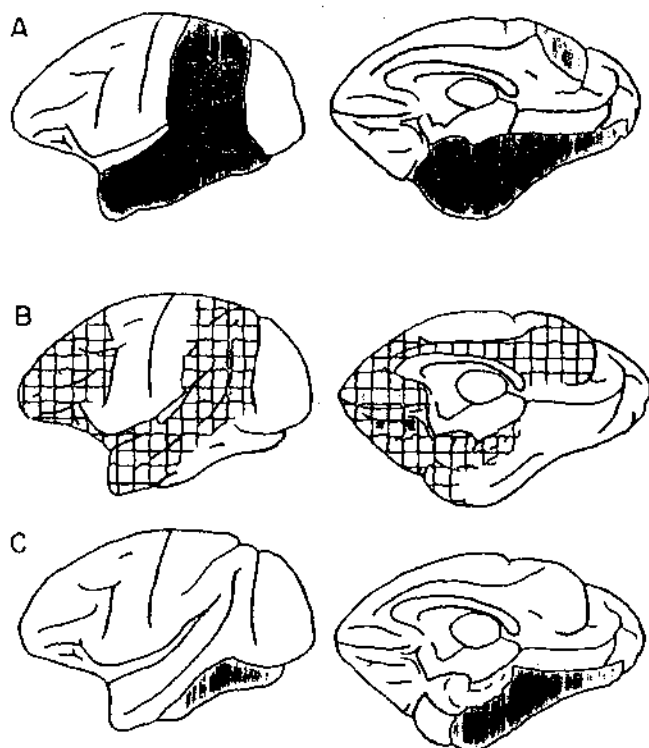
Figure 26. Diagrams of visual choice reaction. A: Sum of the areas of resection of all of the animals grouped as showing deficit; B: sum of the areas of resection of all of the animals grouped as showing no deficit; C: intersect of the area shown in black in A and *not* checkerboarded in B. This intersect represents the area invariably implicated in visual choice behavior in these experiments. (From Pribram, 44.)

TABLE 3

Effects of Inferotemporal (IT) and Parietal (P) Ablations on
Visual and Somesthetic Learning and Performance*

		Preop. Learning		Preop. Retention		Postop. Retention		Postop. Learning	
		Discrimination		Discrimination		Discrimination		Discrimination	
		Visual	Somesth.	Visual	Somesth.	Visual	Somesth.	Visual	Somesth.
P	Group A	—	408	—	5	—	637	331	—
	Group B	320	—	0	—	194	—	—	821
IT	Group A	—	410	—	54	—	19	1000f	—
	Group B	518	—	0	—	1000f	—	—	439

* Data modified from Wilson (81).

TABLE 4

Effects of Inferotemporal (IT) and Posterotemporal (PT)
Lesions on Auditory Discrimination*

Animal	Learning and Performance Scores	
	Preop. trials	Postop. trials
IT-249	280	80
IT-282	280	0
IT-288	560	120
PT-248	200	600
PT-278	400	160
PT-289	380	440

* Data from Weiskrantz & Mishkin (80).

groups were trained on both a visual and a somesthetic discrimination task.
As can be seen, after the parietal operation there was a difficulty in original
learning and retention of the somesthetic discrimination (81). The apparatus
used was an infrared device (9) by which the monkeys' performance was
observed and televised, converted into visible light for display: the animals
were working in darkness, but we could watch their performance on the tele-
vision screen. Visual discrimination was intact, i.e., the savings criterion
was met and original learning fell within the scores of the controls. Con-
versely, the inferotemporal group performed the somesthetic problem with-
in normal limits, both on learning and retention, but showed complete fail-
ure in learning and retaining the visual discrimination (see also Pribram &
Barry, 42).

In the auditory mode, the data (80) are not as clearcut. These data have
not as yet been replicated, but we are now in the process of doing this ex-
periment again. However, Table 4 shows that inferotemporal lesions, which
resulted in a visual discrimination deficit, left auditory discrimination
unaffected. Conversely, a posterior temporal lesion, which left visual dis-
crimination intact, produced some deficiency in auditory choices; definitely
in one subject, not so clearly in two others. Current evidence places these
lesions somewhat too far posteriorly to obtain the main effect. For taste, an
anterior temporal locus has been isolated (3, 54) by the similar use of the
intersect of sums technique (44).

The question remains as to whether there are any "supramodality" re-
gions in this posterior cortical region. This problem has been worked on a
great deal, but as yet most of the results are unpublished. So far there has
been no evidence in the monkey that there is a supramodality organization
in the posterior "association" cortex (cf. Evarts [16] and Wegener*). In
man, the data from Milner's group (35) in Montreal and Teuber's group
(78) at MIT suggest that there might be such a thing as a locus for visual-

* Unpublished observations.

somatic spatial organization or for the organization of verbal behavior, irrespective of mode, but these data are also subject to other interpretations. This is an open subject at the moment which needs much more investigation at both the human and subhuman levels. To summarize, all the evidence points to modality specificity in the posterior "association" cortex.

Now let us turn to the frontolimbic sector and examine the evidence. Figure 27 illustrates the method of the intersect of sums applied to the delayed reaction experiment. The stippled portions in section A represent experiments from the literature (28), including one of my own (5), which suggest that there may be a deficit obtained from lesions in these locations; but this turns out to be an artifact of the particular task since there are control animals that have never been operated on at all—four such animals in my experience—which also show a deficit on this task (it is a most boring task, both to administer and to perform). This finding resolves the discrepancies regarding occasional occurrence of deficit on delayed reaction following posterior cortical resections.
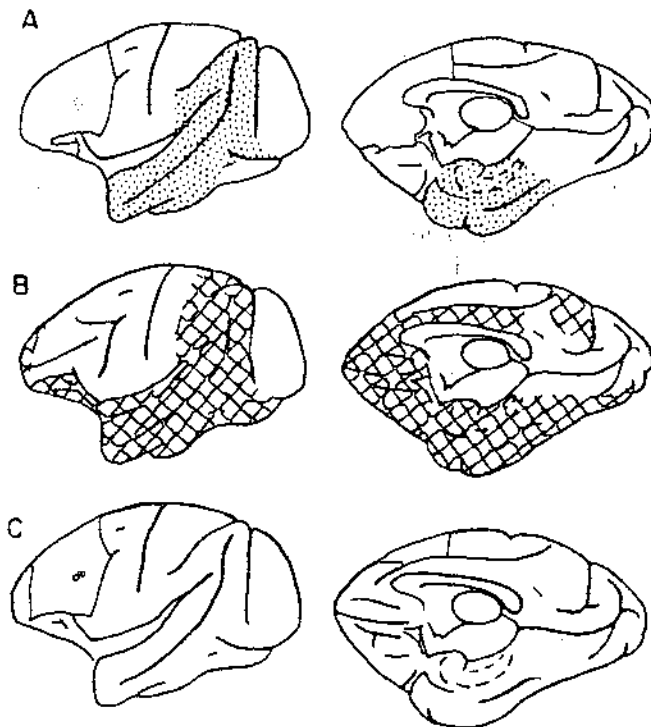

Figure 27. Delayed reaction performance. A: Sum of the areas of resection of all of the animals grouped as showing a deficit; B: sum of the areas of resection of all of the animals grouped as showing no deficit; C: intersect of the area shown in A and that not checkerboarded in B. This intersect represents the area invariably implicated in delayed reaction performance in these experiments. Resections within the area stippled in A occasionally result in "deficit" as defined here; however, a similar "deficit" appears in nonoperated controls. (From Pribram, 44.)

Another task, delayed alternation, is closely related but not identical to the delayed reaction. Performance of this task is impaired whenever a lesion invades the 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. For 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.

Grant: May I raise a question relative to information theory and the variability of the stimulus, particularly with respect to delayed reaction? I know what you mean by its being a rather boring experiment to run, but I assume you were running it with a fixed delay or long delay, or at least in blocks of constant delays. If you vary the delays from trial to trial, I suspect that you might get an entirely different kind of phenomenon, and possibly even a different part of the nervous system would be involved.

Pribram: There are variations of the delayed reaction problem that do make a difference (38, 39, 63), but varying the delay period is not one of them (64). We used a five-second delay, standard correction technique for both tasks. For the delayed response problem we showed a peanut to the animal over one of the food wells, brought down the screen, hid the peanut in the well, and then the screen went up.

Figure 18 illustrates some of the limbic lesions that produce changes in alternation behavior; an orbito-insulo-temporal (OIT) resection is shown. The OIT region includes the amygdala, the anterior portion of the insula, and the posterior orbital portion of the frontal lobe. It receives its projection from the midline, medial macrocellular mediodorsal and medial intralaminar nuclei (32). It can be differentiated as a unit by the method of strychnine neuronography (59, 61). Another such unit is the cingulate, 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 (55). These 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 in our repertoire. Finally, lesions of the hippocampal cortex also lead to difficulty with the delayed alternation problem.

When the method of presenting the delayed response and delayed alternation problems is varied, there can be further differentiation between lesion effects (Table 5). Frontal and limbic (OIT), cingulate and hippocampal lesions have different effects on the performance of different variations of the task (60). The effective variation is a change from a left-right to a go-no go procedure. In the alternation situation the animal is reinforced every alternate time and is expected to stay away from the well on the other times: on one trial a peanut is placed in the well, the screen goes up and the animal responds. On the following trial there is no peanut in the well; the animal has to learn to withhold its response. If it does not, the non-reinforced trial is repeated until it does withhold. On the next trial, the peanut is again

TABLE 5

EFFECTS OF LIMBIC ABLATIONS[*] ON CLASSICAL AND GO-NO GO ALTERNATION

Animal	Preop. Learning to Criterion		Postop. Relearning to Criterion		Preop. Savings on Retention		Postop. Savings on Relearning	
	Days	(Errors)	Days	(Errors)	Days	(Errors)	Days	(Errors)
Classical Alternation								
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)
Average	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)
Average	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%)
Go-No Go Alternation								
MFC 49	13	(533)	4	(27)	2	(471)	9	(526)
MFC 57	20	(886)	14	(215)	18	(860)	6	(641)
MFC 97	23	(745)	12	(726)	21	(741)	11	(19)
MFC 115	14	(340)	4	(55)	12	(337)	10	(285)
Average	17.5	(631)	8.5	(263)	11.2–81.1%	(602)–(95.4%)	9–51.4%	(368)–(58.3%)
OIT 116	11	(498)	6	(192)	9	(491)	5	(304)
OIT 132	15	(916)	10	(380)	12	(813)	5	(136)
OIT 133	11	(713)	10	(1125)	7	(690)	1	(−412)
OIT 134	18	(618)	10	(800)	16	(615)	8	(−182)
Average	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%)

MFC: medial-frontal cingulate; OIT: orbitoinferotemporal.
[*] Data from Pribram, Lim et al. (60).

in the well. This go-no go form of alternation is more severely impaired than the right-left variation of the task when the lesions are limbic (especially OIT). On the other hand, Figure 28 shows that, when the lesions are of frontal cortex, the go-no go variation of the procedure turns out to be much easier for the monkey than the right-left variation (39).

In summary, frontal and limbic lesions produce effects different from those produced by lesions in the posterior cortex. I have not reviewed here the evidence that the frontolimbic defect is not modality specific, but such reviews are in the literature (50, 67). The frontolimbic 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 suffers most from limbic lesions.

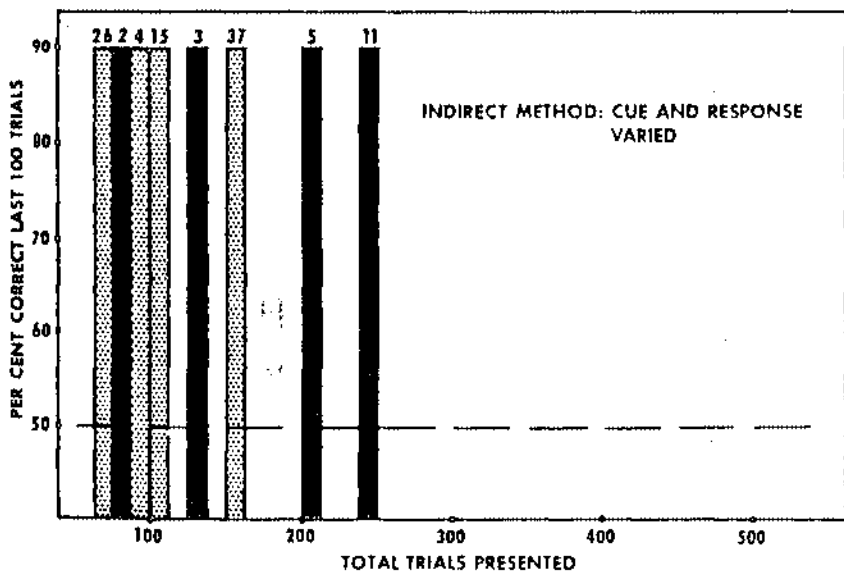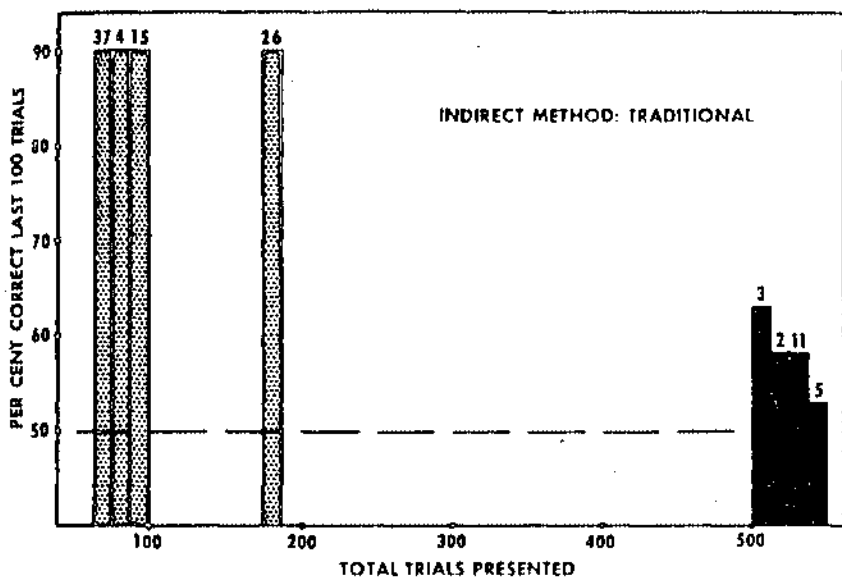Removal of cerebral tissue has not been the only tool in our armamen-

Figure 28. Comparison of the effects of inferotemporal frontal ablations on classical *(above)* and go-no go *(below)* alternation. Each bar represents the performance of one animal (designated by numbers atop bars). Black: frontal ablations; stippled: controls. (From Pribram, 44.)

tarium. Simultaneously, experiments have been carried out in which we placed aluminum hydroxide cream on the cortex or injected it into selected cortical areas (43, 45, 77). 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 we train the animal before the abnormal electrical activity de-

velops (e.g., spikes or spike and slow wave complexes), we find no impair-
ment of visual discrimination behavior as a result of inferotemporal implant-
ation. As seen in Figure 29A, the monkey runs along smoothly at crite-
rion, 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 we have been discussing. Performance was recorded for
many weeks (74, 75, 76). In Figure 29B we have another example—alterna-
tion performance after frontal implantation.

On the other hand, if the animals are trained only after the abnormal
electrical activity has appeared, a marked change in behavior can be dem-
onstrated (Figure 30): original learning of a particular task is impaired
when the electrical activity of the appropriate cortex becomes abnormal.
The figure depicts again the visual choice reaction, visual discrimination
following EEG abnormality in the inferotemporal cortex. Learning is de-
layed approximately fivefold. Note that the slope of the curve is not drasti-
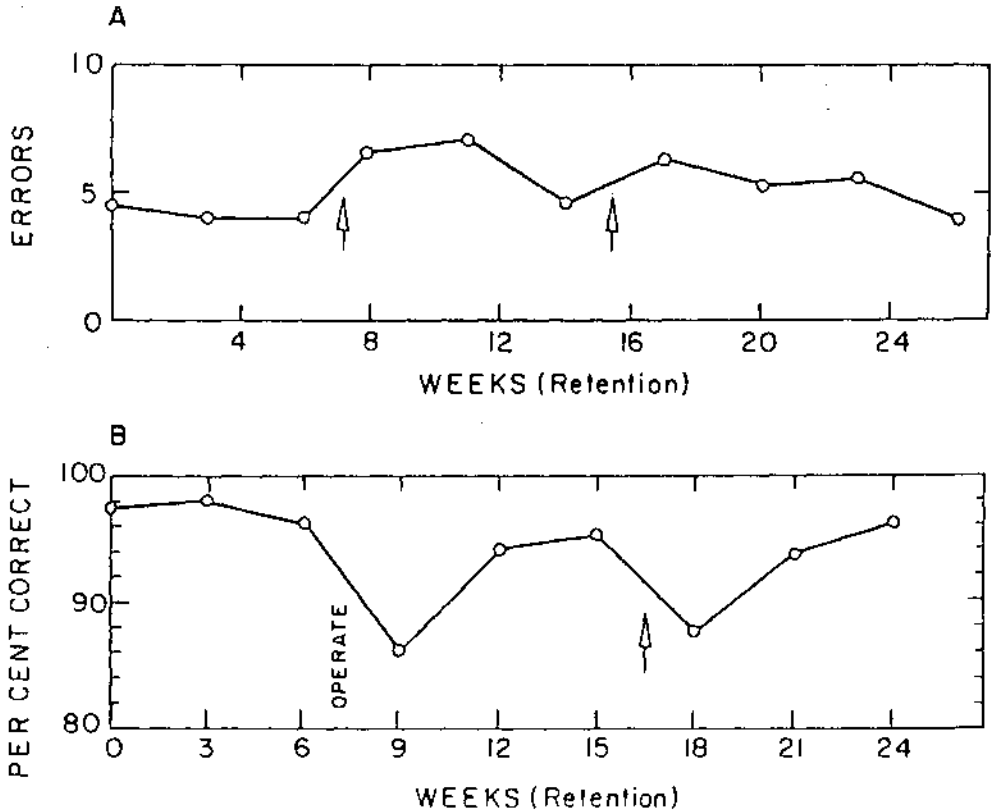

Figure 29. Performance scores before and after aluminum hydroxide implantation. A:
Visual discrimination problem and implantation (left arrow) on the inferotemporal cortex;
right arrow indicates the onset of electrical seizure patterns. (From Stamm & Pribram, 75.)
B. Alternation performance and frontal lobe implantation; arrow shows onset of electrical
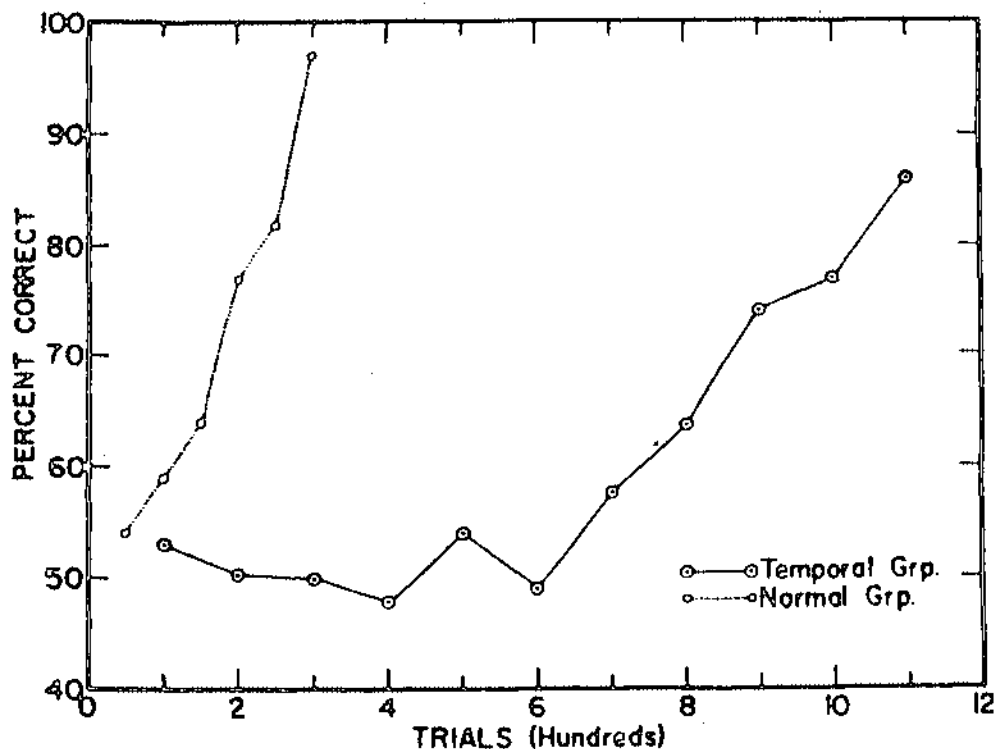seizure patterns. (From Stamm & Pribram, 76.)

Figure 30. Visual discrimination of a learning curve obtained from a group of monkeys with electrical seizures recorded from inferotemporal implantation sites. (From Stamm & Pribram, 76.)

cally changed; rather, the onset of learning is retarded. This finding may be important to uncovering the mechanism which underlies the disturbance.

Impaired alternation learning is represented in Figure 31. There is no long delay before an inflection point, but alternation learning ordinarily shows no such "single element" attributes even when normal subjects are used, as can be seen from this control sample.

Before going on to even more interesting data, I would like to point out that these last experiments bear directly on something Dr. Galambos mentioned earlier, the problem of distinguishing between performance and the acquisition of that performance. The acquisition of behavior appears to be highly correlated with what we obtain electrically from the brain, but we have not been able to find any such correlation between electrical changes and performance *per se*.

To make the story complete I should mention that a converse experimental result has also been obtained. Using the ablation technique, Lawrence Weiskrantz (79) of Cambridge University followed this paradigm: train the animals on a particular day to criterion on a particular discrimination, let us say A *versus* B. On the following day, test for the retention of A *versus* B,
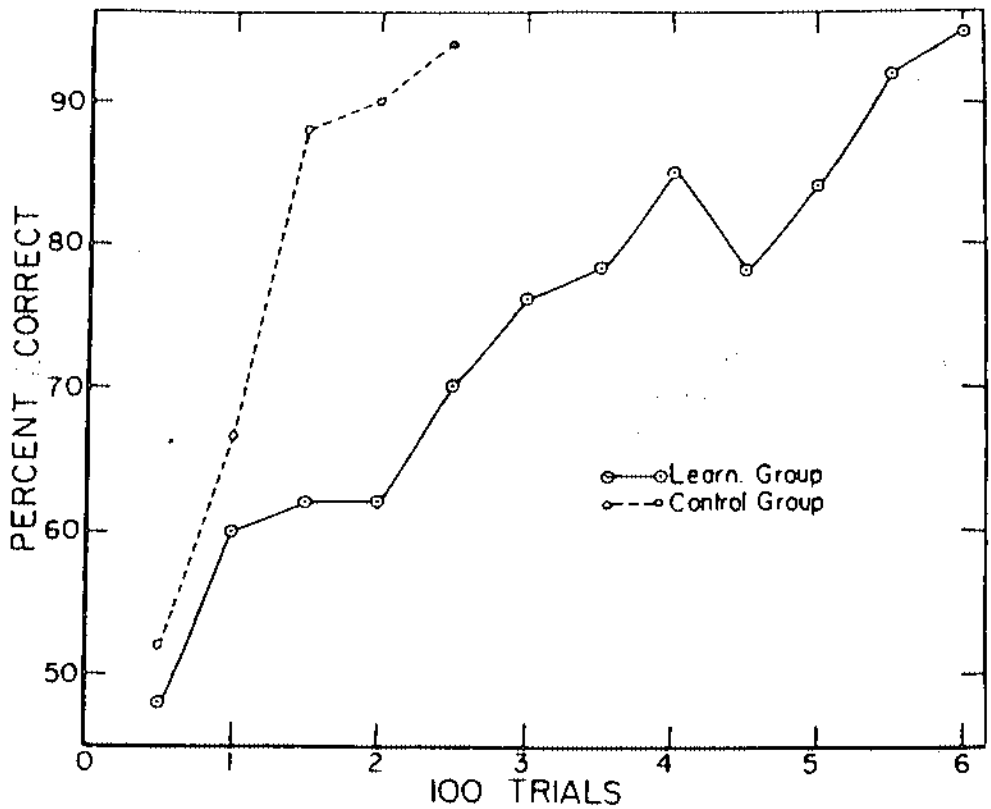
Figure 31. Alternations learning curve recorded from a group of monkeys with frontal lobe electrical seizures. (From Stamm & Pribram, 75.)

and teach a new discrimination, C *versus* D. On day three 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 a variety of small 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.

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 as opposed to "dismembering", in the sense that these animals must put together again, or retrieve, elements used to solve problems.

Birch: How do you interpret the long period of no change? Is there something happening that is irrelevant to learning?

Pribram: If you can tell me what process is going on to generate the part of a backward learning curve prior to the inflection when learning presumably takes place, I will be glad to tell you what may be going on in these experiments. At the moment, I do not know. (Incidentally, we have plotted many of our visual discrimination results as backwards learning curves and usually obtained a nice sharp rise—though we have not tested for stationarity in most of the experiments.) Figure 31 shows that this same abrupt rise does not appear when alternation behavior is examined.

Brazier: How many days postoperative is zero?

Pribram: Zero is usually at least a month postoperative in these experiments, because the seizure pattern usually does not develop until three weeks to a month after implantation. We wait at least until we have seen the abnormal electrical pattern on two occasions and we record once a week.

Miller: Do such patterns continue once they are established? Is there no change in the electrical activity associated with the beginning of learning?

Pribram: We have tested for only three to six months, and during that time the abnormalities are maintained. I would not say there is no change, but we still see the abnormal activity.

O'Connor: Because of the behavioral manifestation in the operated group, is there any indication of head or eye movements during the delayed augmentation?

Pribram: Not from the lesions I am reporting here. If we make the irritative focus in the motor region, we see Jacksonian motor seizures, and the animals also show tremors both at rest and during intentional movement. But that is another story (58).

THE PSYCHOLOGICAL SIGNIFICANCE OF THE BRAIN-BEHAVIOR RELATIONSHIPS

The psychological significance of the findings described can be concretized somewhat like this: if a deficit in color discrimination is obtained, does it mean that the animal is color blind? Part of the cortex is removed, and the animal now fails a color discrimination: does that in itself mean the animal is color blind? Obviously not. We need other kinds of tasks besides color discrimination to test the limits of the deficient behavior. We turned to brightness differences and to patterns of various sorts, and found all manner of visual tasks to be affected by this particular lesion (36, 38, 39). Figure 32 shows that differences in the physical dimensions of the stimulus, in this case a size, are distinguished less after the lesion (37), but this is not the whole 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 the monkeys the goodly distance between home cage and laboratory. The monkeys were failing miserably the visual discrimination tasks. 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: I did not catch it. The monkey reached out again, and again it caught a gnat and put it in its mouth. I reached out—missed! Finally, the paradox of the situation forced itself on me. I took the beast back to the testing room, but it was as deficient as ever in making visual choices.

This observation gave rise to the following experiment, which Ettlinger (15) accomplished, with the results shown in Figure 33. We hypothesized that choice was the crucial variable: as long as a monkey does not have to make a choice, its visual performance should be found intact. Monkeys were trained in a Ganzfeld made of a translucent light fixture large enough so that an animal could be physically inserted into it. The monkey 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
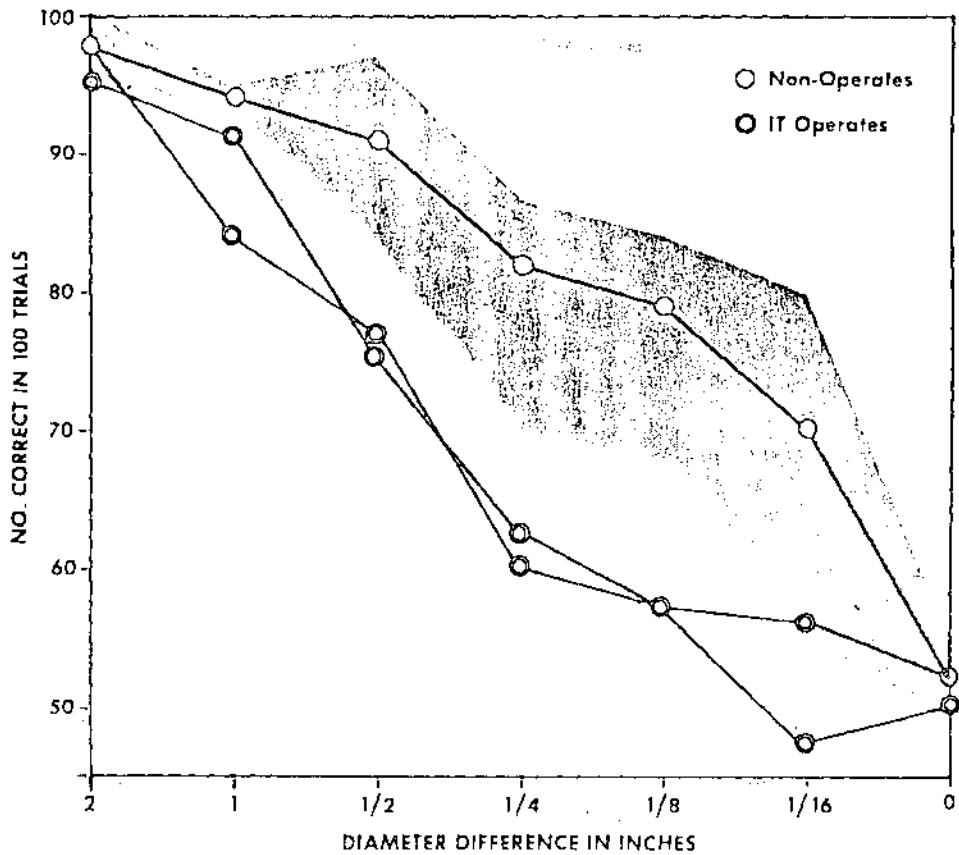

Figure 32. Difference in performance of inferotemporal and control monkeys on a visual discrimination problem in which size discrimination was varied parametrically. Shaded area: variability. (From Mishkin & Hall, 37.)
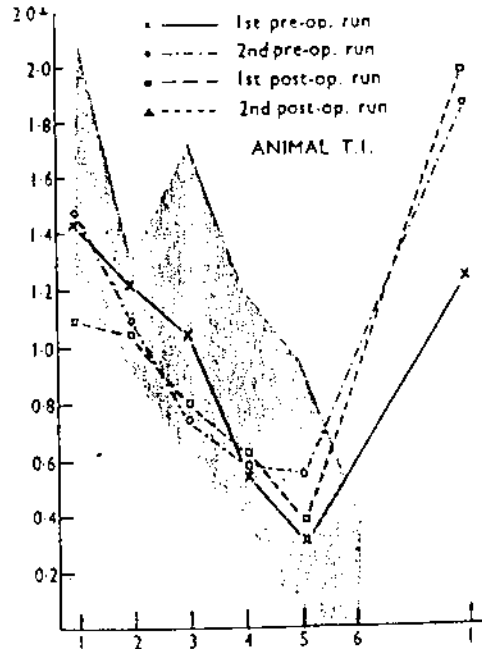
Figure 33. Single manipulandum performance curves of a single animal in a varying brightness situation. *Abscissa:* Geometrically decreasing differences in luminance between positive and negative cues; *ordinate:* log of ratio of response rate to positive and negative cues. Shaded area indicates variability among groups of 4 animals. (From Ettlinger, 15.)

tended to support our feeling that, if an inferotemporally lesioned monkey did not have to make a choice, he would show no deficit in behavior. The animal is not punished for error because that would entail a choice. The lack of punishment is important in making this experiment closer to an "existential" discrimination, which would be ideal. Error is not involved. The monkey can press any time, but has been reinforced only when the "brighter" condition is in effect, and the difference in reward between the conditions is further minimized by the fact that the brighter condition is rewarded on a modified fixed ratio schedule, i.e., not every lever press is rewarded.

Miller: If you reinforced the animal 100 per cent for responding to the correct light and zero for the incorrect response, would it then have a deficit in that same Ganzfeld?

Pribram: In a simultaneous choice experiment, inferotemporally lesioned monkeys fail to respond differentially to differences in brightness (39). Another difference in discrimination is that illumination is general in one case and specific to the object in the other; one is of "ground", the other of "figure". There are also differences in the reinforcement schedule. The following experiment may clarify the problem.

We (62) trained the monkeys on a very simple object discrimination, an ash tray *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. When, however, we take the same cues and present them successively (there are two types of successive discriminations: in one the animal has to go either left or right), the monkeys show a deficit

when compared with their controls (Figure 34). We know they can
differentiate the cues from their performance in the simultaneous situation;
yet when a more difficult response is required they have difficulties.

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. It is not only the stimulus condition *per se* but the contexts in
which it appears that determine the deficit. Another, more precise, way of
stating this is that the deficit ought to vary as a function of the number of
alternatives in the situation. This hypothesis was therefore tested directly in
another experiment (47). It has not as yet been replicated and so the results
must be considered tentative, albeit persuasive. The hope was that an infor-
mational measure of the deficit could be obtained. Figure 35 shows that ac-
tually something very different appeared when the number of errors was
plotted against the number of alternatives.

The square root transformation is of the raw data. Since analysis of vari-
ance was used to establish significance, the data had to be normalized first.
I want to point out some other complications in the experiment. First, there
is the confounding of the number of alternatives in the situation and the
order in which they were presented; this is therefore not really a good test
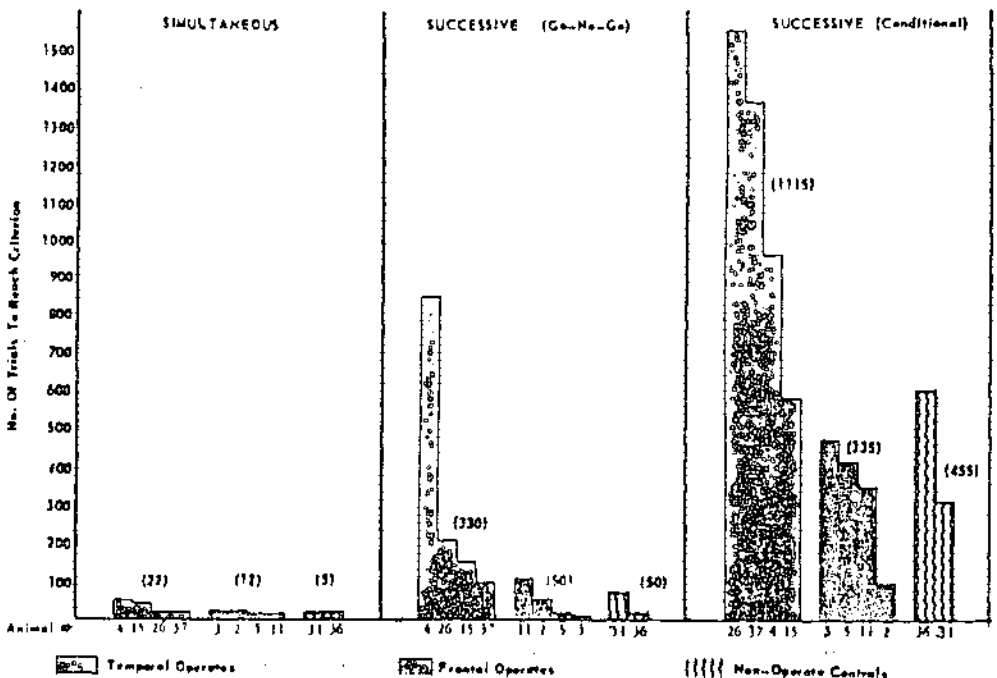of the information measurement model that I had in mind. The experiment

Figure 34. 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
demanded response increases the deficit of the inferotemporal groups. (From Pribram
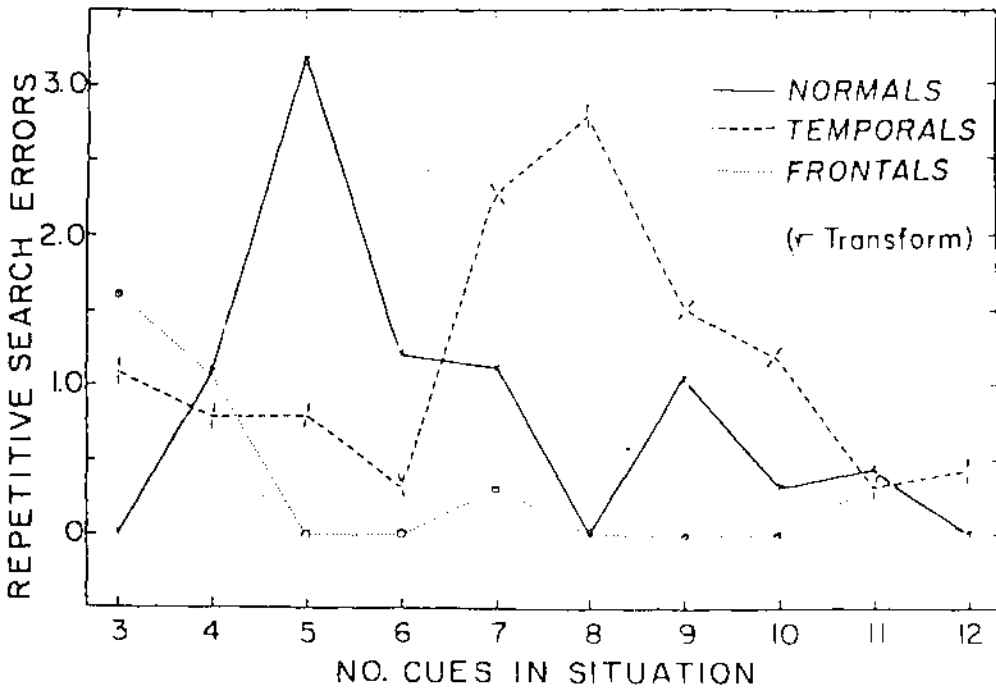& Mishkin, 62.)

Figure 35. Average number of repetitive errors made in the multiple object experiment during those search trials in each situation when the additional (novel) cue is first added. Four animals per group. (From Pribram, 47.)

could not be done as originally planned because, up to that time, no one had ever tested monkeys on more than three cues at a time. I had to start with two cues and work up. Also, there was no way of matching preferences for cues and so the same cues were used throughout the experiment, balanced in order of presentation among subjects but given in a standard order for each subject. Despite these limitations, a thought-provoking result emerged from the experiment.

If repetitive errors are plotted—i.e., the number of times a monkey searches the same cue, against the number of alternatives in the situation—a hump is found in the curve, a stage of many repetitive errors through which normal animals go; they then recover adequate performance 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 procedure continued, however, and after the controls no longer made so many errors, the inferotemporals began to accumulate an error hump even greater than that shown earlier by the controls. The analysis of variance shows these two curves to be significantly different despite their overlap in the latter half of the graph.

Before I had such an explanation I presented the results of this experiment informally to a group, and Edward Green, a mathematical psychologist, suggested that the position of this hump varied with the number of alternatives sampled by the subject, and that the inferotemporally lesioned

monkeys who showed the delayed hump had sampled fewer cues in the early stages of the experiment. Since these cues had to be uncovered, I had a record of actual "sampling" when a particular cue was turned over. It was only necessary to go back through the data to see whether differences in sampling between groups was obtained.

The differences did occur, as can be seen in Figure 36. The monkeys with inferotemporal lesions showed a lowered sampling ratio: they sampled fewer cues during the first half of the experiment. We might characterize their performance as a restriction in their visual field; however, the limitation is not in the visual-spatial field but in the information-processing field, i.e., the number of alternatives they can sample or handle at any one time. This curve shows that most of the variance that accounts for the error humps was obtained when a novel cue was introduced into the situation. The inferotemporally lesioned subjects (as well as the controls) made their runs of repetitive errors on these occasions. (Frontally lesioned subjects invariably chose the novel cue immediately.) During a trial, the monkeys had just one chance to sample a cue; the screen came down between trials. When there were only four or five cues in the situation, the inferotemporally lesioned monkeys found the correct one more rapidly than did the con-
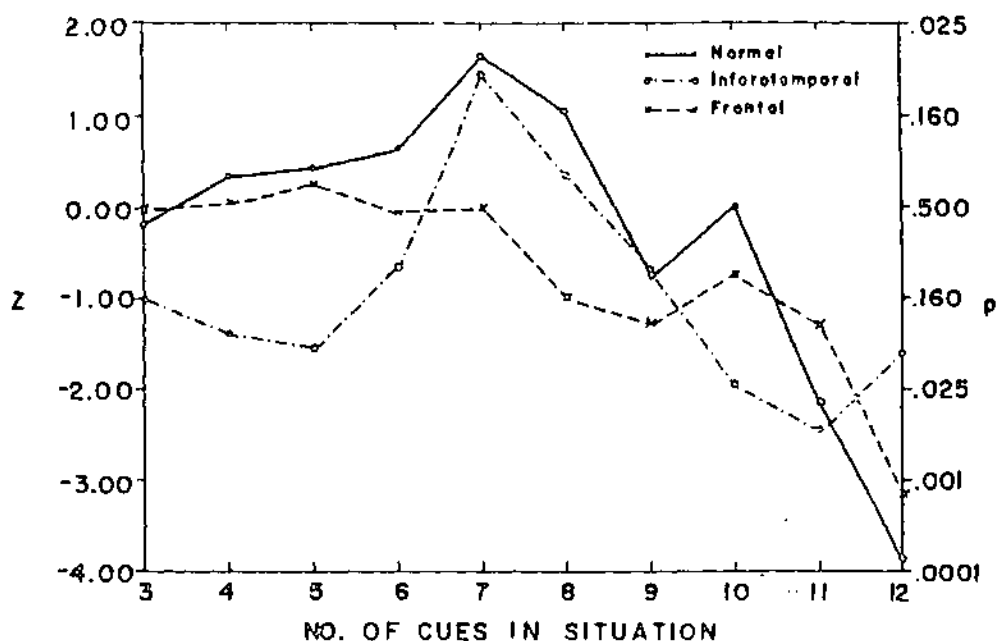
Figure 36. Sampling performance except novel cue. Average percentage of the total number of objects (cues) that are sampled by each of the groups in each of the situations. To sample, a monkey had to move an object until the content or lack of content of the food well was clearly visible to the experimenter. As was predicted (47), 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; cf. Mann & Whitney, 31.)

trols, who sampled more of the previously reinforced cues before turning over the novel cue. The correct cue object is always the same until a criterion is met; then a "discrimination reversal" type of procedure takes place until all of the cues have been rewarded. All cues that had previously been reinforced are still present; only the currently correct cue changes. For example, the monkeys go through a whole series of problems for which red is correct. When they reach criterion on red, then reversal is instituted and green becomes correct. After criterion is reached on green, blue is added as the correct cue. To reach criterion they must choose the correct cue on five successive trials.

In summary, 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—a reversion to chance behavior when compared with a control group whose sampling is guided by the history of prior differential discriminations. In short, the lesioned monkeys fail to remember prior discriminations as well as the controls, and this failure alters the sampling of current cues, i.e., the process of selective attention. I will return to this notion of a memory-based information processing defect when I discuss the model. But first let me present briefly some data on the frontolimbic systems.

For purposes of comparison, Figure 37, A and B, demonstrates that frontally (and limbically) lesioned primates also fail to be influenced by their experience, but in a very different way from the posteriorly lesioned subjects. They appear to be impervious to the consequences of their behavior. Initially, this defect appeared most dramatically as imperviousness to error, i.e., in avoiding shocks (44, 65) and non-reinforcements.

In another experiment (50), the animals were trained in an operant conditioning situation. After several years of training on mixed and multiple schedules, four hours of extinction were run, i.e., the reinforcements (peanuts) were no longer delivered, although everything else in the situation remained the same. Figure 38 shows the results; note that the frontally lesioned animals failed to extinguish in the four-hour period, whereas the control monkeys did. This failure in extinction accounts for poor performance in another task. Figure 39 shows what happens to the number of repetitive errors made in a go-no go alternation: the frontally lesioned animals do make many more repetitive errors. Even though they do not find a peanut, they go right back and keep looking (47).

This result was confirmed and amplified in a study by Wilson (82), who analyzed the occasions for error—did errors follow alternation or nonreinforcement? He devised a situation in which both lids over the food well opened simultaneously, but the monkey could obtain the peanut only if it 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 the procedure was presented with four variations:
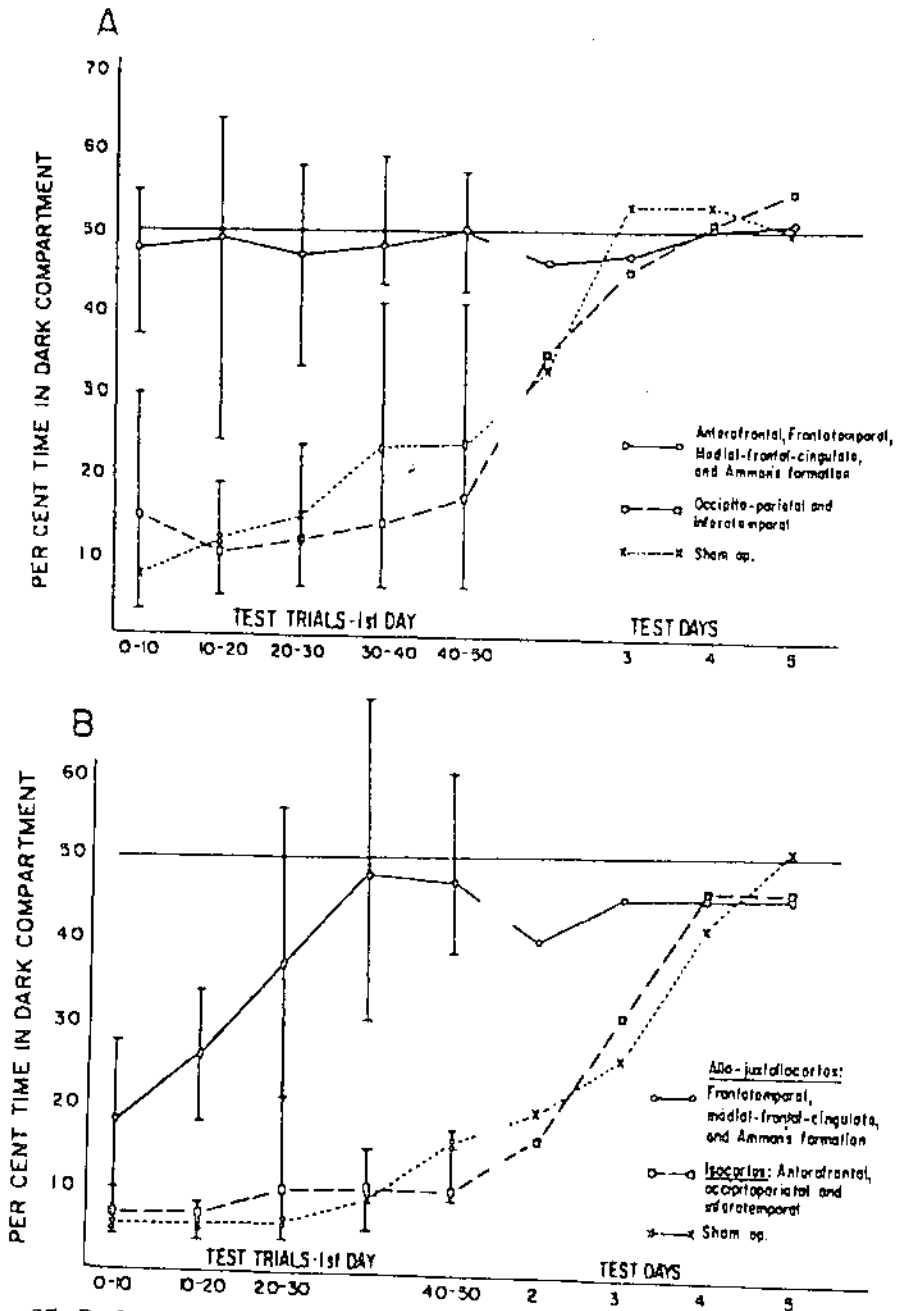
Figure 37. Performance of limbically and nonlimbically ablated monkeys during postoperative extinction of a learned conditioned avoidance response. A: *Preoperatively* learned avoidance; note that limbically and frontally operated monkeys behave alike. B: *Postoperatively* learned avoidance; the limbic groups are clearly separated out by this procedure. (From Pribram & Weiskrantz, 65.)
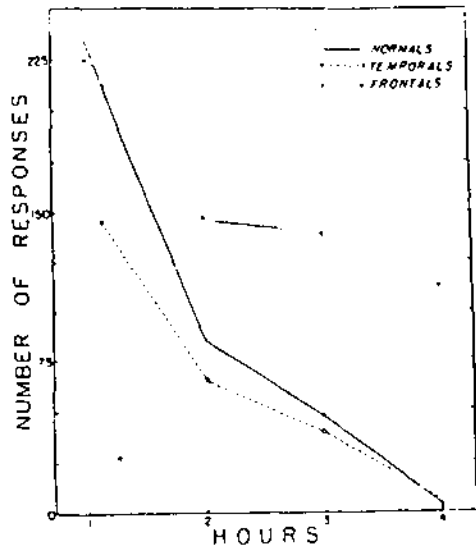
Figure 38. Graph of performance of three groups of monkeys under conditions of extinction in a mixed-schedule operant conditioning situation. Note the slower extinctions of the frontally lesioned monkeys. (From Pribram, 50.)

correction-contingent, correction-noncontingent, noncorrection-contingent, and noncorrection-noncontingent (Table 6). The contingency was whether the position of the peanut depended on the prior correct or incorrect response of the monkey, or whether this position was alternated independently of the monkey's behavior. Wilson then analyzed the relationship between an error and the trial preceding that error. For the normal monkey, the condition of reinforcement and nonreinforcement of the previous trial makes a difference, whereas for the frontally lesioned monkey it does not; alternation affects both normal and frontal subjects about equally; frontal subjects are simply not influenced by rewarding or nonrewarding consequences of their behavior.

This inefficacy of consequences to influence behavior is also demonstrated in the multiple choice experiment just discussed (47). Figure 40

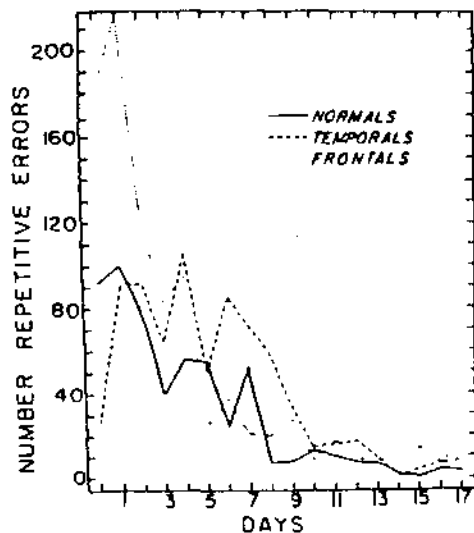Figure 39. Graph showing the differences in the number of repetitive errors made by groups of monkeys in a go-no go type of delayed reaction experiment. Especially during the initial trials, frontally operated animals repeatedly return to the food well after exposure to the "nonrewarded" predelay cue. Note, however, that this variation of the delay problem is mastered easily by the frontally operated group. (From Pribram, 47.)

TABLE 6

PERFORMANCE OF FRONTALLY ABLATED AND NORMAL MONKEYS[*]

Percentage of Alternation as a Function of Response and Outcome of Preceding Trial

Subject	Preceding trial			
	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: alternated; NA: did not alternate; R: was rewarded; NR: was not rewarded.
[*] Data from Wilson (82).
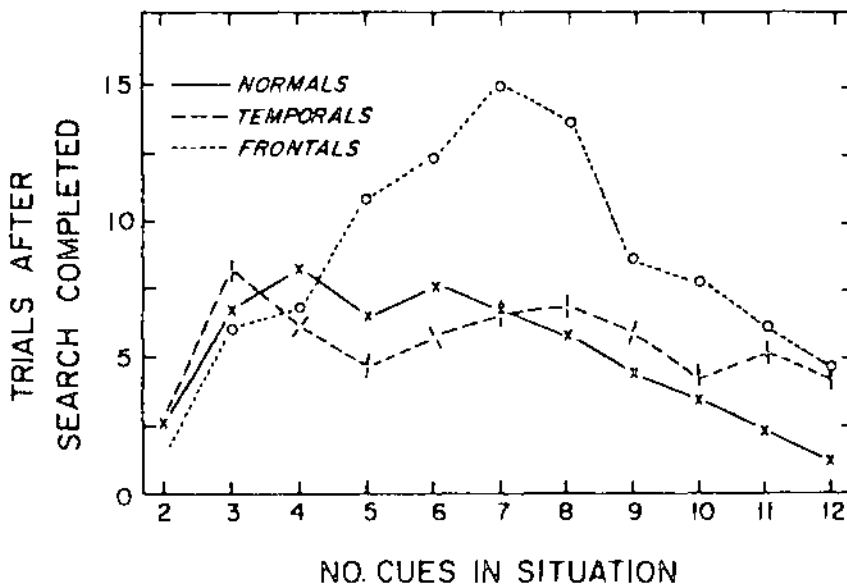
Figure 40. Graph of the average number of trials to criterion taken in the multiple object experiment by each of the groups in each of the situations after search was completed, i.e., after the first correct response. Note the difference between the curves for the controls and for the frontally operated group, a difference which is significant at the 0.05 level by an analysis of variance (F = 8.19 for 2 and 6 df) according to McNemar's (33) procedure performed on normalized (by square root transformation) raw scores. (From Pribram, 47.)

TABLE 7

PERFORMANCE OF LOBOTOMIZED AND CONTROL HUMAN SUBJECTS
WHO COMPLETED THE MULTIPLE CHOICE TEST*

	Lobotomized	Controls
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

* Data from Poppen, Pribram & Robinson (41).

shows what happens after the monkeys have found the peanut. The procedure calls for the strategy of return to the same object five consecutive times, i.e., to criterion. The frontally lesioned animals are markedly deficient in accomplishing this task. Again we see that the conditions of reinforcement are relatively ineffective in shaping behavior once the frontal engranular cortex has been removed, so that the monkeys' behavior is relatively random when compared to that of normal subjects (53). 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.

In case you should object to descriptive labels taken from the subjective realm of discourse (on the basis that they must not be applied to animals), Table 7 shows that the results obtained with monkeys hold for man, in experiments (41) performed with 20 lobotomized patients and their controls. The procedure was made as similar as possible to that used with the primates. And results were remarkably similar.

THE MODEL

These data led me to define (48) the psychological processes impaired by "association" cortex lesions and to suggest the outlines of a model for these processes. To review the definition, the posterior system apparently is involved 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).

Now, at last, the model: the neurophysiology of selective attention and intention. The model is far from being complete or even buttressed by data. Rather, it should be viewed as a progress report and a projection of our current endeavors. Therefore let us first consider some facts, or rather some lack of facts, about the neuroanatomical relationships of the inferotemporal cortex. There is a dearth of neurological evidence to link this cortex to the

known visual system, the geniculostriate system. There are no definitive anatomical inputs specific to the inferotemporal cortex from the visual cortex or the lateral geniculate nucleus. Of course, connections can be traced via fibers that synapse twice in the preoccipital region, but such connections also exist to link the visual cortex to the parietal lobe, the excision of which does not change visual behavior, as we have seen. In addition, circumsection of the striate cortex does not impair visual discrimination (8).

Further evidence that these "corticocortical" connections are not the important ones can be seen from the results of the experiment in which I performed a cross-hatch of the inferotemporal cortex, much as Sperry (71) had done, and found no deficit either in visual learning or in performance (Table 8). On the other hand, undercutting the inferotemporal cortex makes a vast difference: both learning and performance of visual tasks become precluded. This suggests that the connections to this cortex essential to visual behavior must come from somewhere below, although large U fibers, dipping deeply into white matter, are not yet ruled out. However, another possibility can be tested, namely that the essential relations of the posterior association cortex are centrifugal, i.e., efferent (46). There is some anatomical evidence to suggest such a notion: some time ago, I prepared two brains with inferotemporal resections. These were stained in Dr. Walle Nauta's laboratory by his technique, and showed an efferent tract going down to the region of the superior colliculus, ending either within its substance or in the surrounding reticular formation. No such fibers could be traced to the lateral geniculate nucleus. In support of this finding there is a report by Kuypers (27), who has also traced temporocollicular fibers in monkey.

The idea of an efferent mechanism "gating" or otherwise "partitioning" the input to the geniculostriate system has a good deal of appeal as an explanation for the process of selective attention. To determine how an effer-

TABLE 8

Effects of Undercutting and Crosshatching Inferotemporal Cortex of Monkeys on Their Performance in Several Discriminations

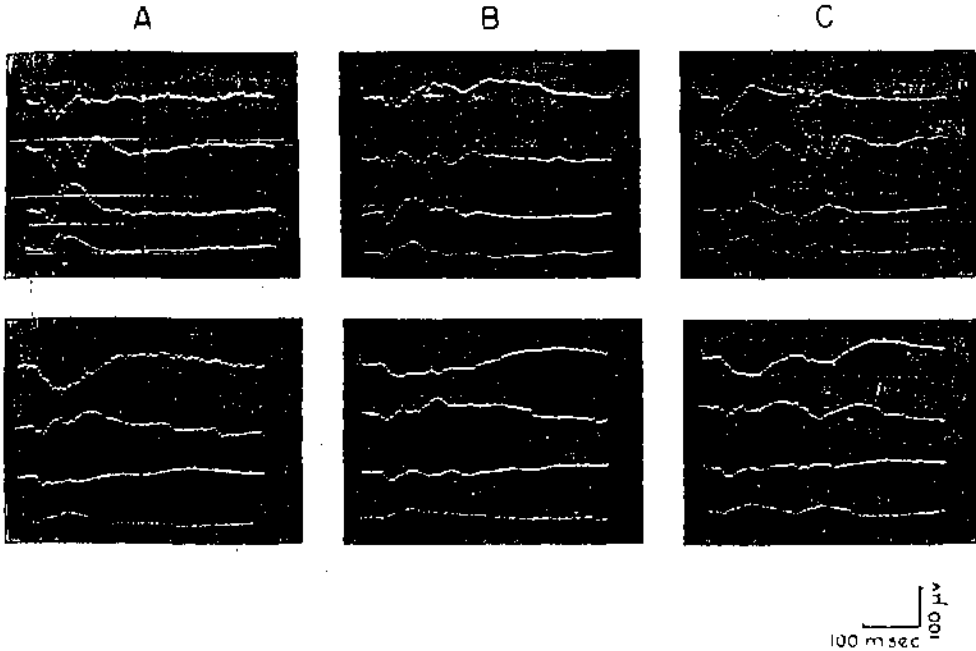|	Animal	3 vs. 8	R vs. G	3 vs. 8
Crosshatch	158	380	82	0
	159	180	100	0
	161	580	50	0
	166	130	0	0
Undercut	163	(1014)	100	300
	164	(1030)	200	(500)
	167	701	50	0
	168	(1030)	150	(500)
Normal	160	280	100	0
	162	180	100	0
	165	280	100	0
	170	350	100	0

Figure 41. A representative record of the change produced in visual evoked responses by chronic stimulation of the inferotemporal cortex. *Above:* Records taken before stimulation; *below:* records during stimulation. All traces were recorded from the striate (visual) cortex; the top two in each set from posterior, the lower two from anterior striate cortex. The first sets (A) were recorded in response to a single flash, the second (B) to flashes separated by 75 msec., and the third (C) to flashes separated by 150 msec. In addition to the changed recovery, note also the change in wave form of the response upon presentation of even a single flash; this latter change, however, did not appear in all of our subjects. (From Spinelli & Pribram, 72.)

ent mechanism of this sort would work, we did experiments in which, instead of making ablations or implanting an epileptogenic lesion, we chronically and continuously stimulated 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 and the recording equipment we are using (72). 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, of about 3 V. The frequency of stimulation is approximately 8-10/sec. The batteries that drive the stimulator are rechargeable.

The records in Figure 41 were made in the awake monkey. Paired flashes were presented and recordings made from electrodes implanted in the occipital cortex. The responses to 50 such paired flashes were accumulated on the Computer for Average Transients. The flash-flash interval is varied from 25-200 msec. The top traces were recorded prior to the onset of stimulation; the lower ones were made after chronic stimulation had been started. Actually, this was the first of our series of experiments to call our attention to the changed recovery phenomenon. Note that there is a general flattening (a finding idiosyncratic to this particular monkey) of the record made with

the stimulator on, and that the recovery function is depressed, i.e., recovery is delayed. Figure 42 shows such effects summed across five subjects. I think it reasonable to conclude that chronic stimulation of the inferotemporal cortex produces a very marked delay in recovery of the cells in the visual system to visual stimuli.

A parallel experiment (12) in the auditory system was done in collaboration with Dr. James Dewson. In this study, made in cats, removals of the auditory homologue of the inferotemporal cortex were performed. This homologue is the insular-temporal region in the cat. Dewson has shown that its removal impairs complex auditory discrimination (speech sounds), leaving simple auditory discrimination (pitch, loudness) intact. Removal also alters paired click recovery cycles recorded as far peripherally as the cochlear nucleus. Bilateral ablation markedly shortens the recovery cycle. 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.

These results allow us to specify the model. On the basis of the neurobehavioral and neuroanatomical data, I had previously suggested (48) that the posterior "association" cortex, by way of efferent tracts leading to the brain stem—most likely to the colliculi or surrounding reticular formation (46)—partitions the events that occur in the associated sensory specific system, classifying these events according to one or another scheme. During the
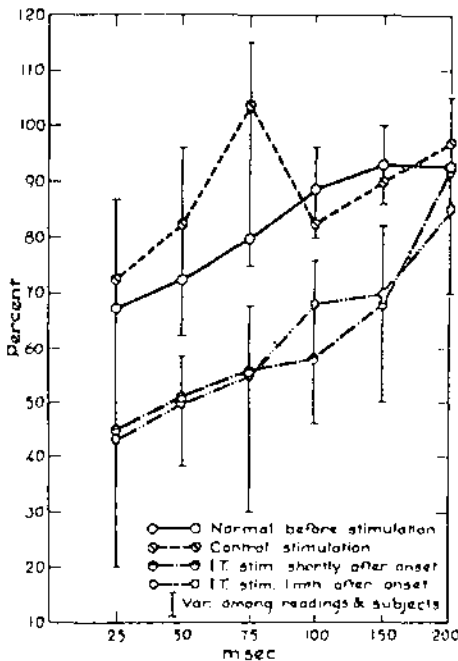

Figure 42. Plot of the recovery functions obtained in five monkeys before and during stimulation of the inferotemporal cortex. (From Spinelli & Pribram, 72.)

course of our joint work, Dr. Spinelli would ask me again and again, "How do you define "partitioning" in neurological terms?" Until we had accomplished these electrophysiological experiments, I had no idea. But once we saw the results, the neurophysiological explanation became evident: partitioning must work somewhat like a multiplexing circuit in electronics. In neurophysiological terms, when recovery time of neurons in the sensory projection system is increased by stimulation of the posterior "association" cortex, fewer cells are available at any given moment to the concurrent input. Each of a 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, i.e., 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 (or surround) inhibition in the retina has the effect of reducing redundancy (4), so the operation of the "association" cortex enhances the density of information which the channel conveys.

This model has several important implications. First, the nonrecovered 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 this sort is demanded by models of the process of recognition and selective attention spelled out on other occasions by Craik (10), Sokolov (69), Bruner (6), MacKay (30), and myself (49, 51, 52). 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 (20)—that is, 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; i.e., attention can be given to events of greater complexity. The greater the resolution, the sharper the "uncertainty" and, thus, the more likely that any set of inputs will be sampled for information. In the extreme, 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.

I wish I could, at this time, present an equally rigorous neurophysiological model for the process of intention. But in this area we are a considerably greater distance from a precisely statable model. It is true that the process of reinforcement enhances redundancy (17). And, in part, the operation of the frontolimbic systems tends to balance that of the sensory specific systems. Monkeys with inferotemporal ablations tend to perform better on the alternation tasks which are so disturbed when frontolimbic lesions are made.

The reverse, however, does not hold. The data in Table 9 suggest 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 or pattern of the redundancy is crucial; mere repetition is an ineffective form. Redundancy is thus not a measure of simplicity. Rather, when properly used, redundancy is not solely opposed to information (or uncertainty) but becomes an additional measure of complexity (20). The structure of redundancy, its temporal pattern, is therefore the key to the neurophysiological model of intentional behavior. Its keystone 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 allow the model to become actualized in neurological terms (34, 49, 52).

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

TABLE 9

Average Number of Errors Made on Learning of Visual Discrimination and Alternation

	Laterofrontal $n = 8$	Control $n = 16$	Inferotemporal $n = 8$
Visual Discrimination	175	150	F
Alternation	F	155	90

n: Number of subjects in group.
F: Failure in 1000 trials.

there is awareness of awareness. According to the model presented here, there is no need for such infinite regress. Important functions such as perception and decision 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 systems are *not* "association" systems; they simply alter the configurations of input-output relationships processed by the classical 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 it is replaced by a downward regress of sub- and subsubroutines; to me this type of regress is the more understandable and manipulatable. The posterior association cortex is conceived simply to program, to structure, an input channel, perhaps through action on recurrent 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, and selective attention "automatically" follow.

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 channel itself—the channel 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. I am tempted to extrapolate and say that the signal carries different meanings depending on the particular structure or organization of the redundancy of the channel.

Discussion

Magoun: Dr. Pribram's proposal that the association areas of the cortex exert their functions in discriminatory behavior by varying the information-conveying properties of input signals to primary cortical areas, through cortico-reticulo-cortical loops with the central brain stem, seems to me to have a great potential for elucidating the role played by these so-called silent areas of our hemispheres. I thought he built this up in a splendidly sequential fashion. He started with some of the basic features of neuronal physiology, through identification of frequencies of firing as the method by which neurons signal the intensity of excitation, and made reference to the action of facilitation in abbreviating the recovery time of discharging neurons and consequently modifying their interspike intervals, hence their firing frequencies. He went on to relate this to information theory (which explores how neural activity conveys information) in terms of the relations of interspike intervals and the probabilistic aspects of firing frequencies and

timing to the features of novelty, monotony, redundancy, association, habituation, or extinction. He then applied these basic concepts to a functional model of higher neural activity, to account for the impairment of perceptual discrimination and acquired performance following lesions in these silent, lateral, frontal, limbic or infratemporal areas, which have never been found to influence significantly the activity of primary sensory or motor cortical systems by way of direct connections.

He next identified the corticifugal projections from these regions to the nonspecific facilitatory or inhibitory corticipetal systems in the central cephalic brain stem, and proposed that, by this route, the silent cortex acquired the capacity for modifying the function of the primary sensory and motor cortical regions serving perceptual and motor skills.

It seems to me that we can find support for this concept from a number of current findings. The collective magnitude of corticifugal projections to the central brain stem appears second only to that from peripheral receptors, and their capacity to modify the discharge properties of subjacent nonspecific systems can be inferred from changes in cortical EEG patterns, as well as by direct observation of changes induced in reticulo-reticular conduction by cortical stimulation.

In addition, as has recently been demonstrated, nonspecific corticipetal influences from the brain stem can either reduce or prolong corticoneuronal recovery time, thus controlling interspike intervals and, hence, the information-conveying properties of these discharging cortical areas. On the input side, for example, Fuster (18), Lindsley (28a, 73), Davis (11), and Dr. Hernández-Peón, as he showed earlier, have all demonstrated improved cortical reception of paired visual, auditory, or tactile signals during attention in human subjects, as well as during behavioral alertness and EEG arousal induced by direct electrical stimulation of the central brain stem in animals.

On the motor side, in both higher animals and man, the initiation of so-called skilled performance or voluntary movement has been found to be associated with the appearance of an arousal pattern of the EEG in the cortical motor area. Moreover, the threshold for evocation of movement by direct stimulation of this cortical area is much lower during alert wakefulness than during drowsiness or sleep. These data seem to fit well with the model presented by Dr. Pribram.

Its elaboration seems to me to provide some of the most insightful and potentially fruitful hypotheses yet to have been proposed concerning the mode of action of these most recently acquired and highly evolved areas of the cerebral cortex, which reach their greatest development in the brain of man. Its formulation seems a brilliant development on the part of a person who has devoted so much of his research career to the study of these cortical regions. I think all of us have been privileged, indeed, to have been able to hear its exposition at this conference.

II. A "MODEL NEURAL SYSTEM" APPROACH TO THE NEURAL BASIS OF BEHAVIORAL CHANGE[*]

Thompson:[†] Both Dr. Sperry and Dr. Calvin emphasized earlier the importance of analyzing behavioral change at the level of interactions among nerve cells. The research strategies discussed so far in the Conference have generally involved somewhat different levels of analysis. Normal animals are trained, and chemicals are subsequently extracted and analyzed (Dr. Gaito's review), electrical activity is concurrently recorded (Dr. Galambos' review) or, as in Dr. Pribram's extensive and elegant research, lesions are made and the subsequent deficits in performance are measured. All of these approaches are faced with the enormous complexity of organization of the nervous system.

I would like to talk briefly about a somewhat different strategy which is more specifically oriented toward the synaptic interactions among neurons that form the basis of changes in behavior. This approach might be called the analysis of "model neural systems". Instead of dealing with the complexities of the intact nervous system, we first eliminate much of the system and then study the neural processes underlying behavioral changes in the simplified remainder (e.g., spinal cord). The use of neural models is, of course, not without hazards. It is always possible that neither the synaptic mechanisms nor the behavioral changes of the simplified model can be generalized to the intact organism. At the very least the behavioral characteristics of the phenomenon under study ought to be parallel for the model system and the intact animal.

The type of model system analysis I am referring to is well represented by the recent work of Eccles and coworkers on long-term plastic changes in monosynaptic reflexes of the neurally isolated spinal cord (13). As an example, I would like to cite an ingenious study (14), in which nerves for all but a few ankle or toe synergists were cut on one hind limb in chronic spinal cats. The animals were given forced exercise, and the monosynaptic reflex responses were tested for the reduced groups *versus* control side and for an intact group on the operated side *versus* control side. Just as predicted, there was a large increase in monosynaptic responses from stimulation of the reduced muscle nerve relative to the control side, but no asymmetry for synergist groups not operated. Unfortunately, control animals in which the residual muscle groups were carefully protected from all exercise or mechanical stress showed just the same asymmetry of monosynaptic reflexes. The increase in reflex response was at least in part the result of severed nerves, not just of exercise.

In general, such studies have shown a variety of changes in monosynaptic

reflexes, but only subsequent to surgical intervention. Normal use alone apparently is not sufficient to produce long-term plastic changes in these reflexes. It would seem that the neuronal system most amenable to synaptic analysis, the spinal monosynaptic reflex, is peculiarly resistant to normal plastic changes.

In searching for model systems useful in the study of learning, it is well to remember Dr. Konorski's suggested term "plasticity", which labels many types of behavioral change resulting from experience (25). While the transected spinal cord may not exhibit classical conditioning or other specific types of learning as they are usually defined, it does show a variety of plastic changes.

I would like to describe briefly a project in which Dr. W. A. Spencer and I have been engaged on the synaptic basis of flexion reflex habituation in the acute spinal cat (cf., 70). Our results to date illustrate some of the advantages and some of the limitations of the "model neural system" approach. Habituation, incidentally, is perhaps the simplest kind of plastic behavioral change. Its general importance has been emphasized in recent studies and writings by Hernández-Peón (22), Pribram (52), Galambos et al. (19) and others, as well as in extensive behavioral literature extending back to the 19th century (cf., 21). While habituation may not be conditioning *per se*, it is certainly a change in behavior as a result of training, and thus by definition an aspect of learning. Sherrington (68), using the acute spinal dog, was perhaps the first to study spinal flexion reflex habituation as such. Prosser & Hunter (66) demonstrated in a very careful study both habituation and dishabituation of flexion reflexes in the chronic spinal rat. In recent times, spinal response habituation has been studied by Hernández-Peón & Brust-Carmona (23), Nesmeianova (40), Kozak et al. (26), and Buchwald et al. (7). In our own experiments we used the unanesthetized decerebrate cat with low thoracic cord section.

The basic experimental design is extremely simple. Amplitude of the response of a flexor muscle to weak skin shocks delivered every few seconds is measured. "Dishabituation" is accomplished by a strong extra stimulus to the leg. During habituation training the muscle response amplitude decreases over a period of minutes to a stable habituation level. If the stimulus is withheld or given only once per minute, the response recovers gradually to control amplitude. A strong dishabituating stimulus given when the response has been habituated produces an immediate increase in response amplitude.

It would seem mandatory for those who study model neural systems to show that the behavioral phenomena of the model resemble those of the intact animal. In searching the behavioral habituation literature, we were able to identify some nine parametric characteristics relating habituation to stimulus and training variables for a wide range of responses and species.

For example, degree of habituation is directly related to frequency of test stimulus, inversely related to intensity of test stimulus, and so on. We were able to show that spinal flexion reflex habituation exhibits the same nine parametric features characteristic of behavioral response habituation, and may thus be considered a legitimate example of habituation. Times for development of spinal reflex habituation and spontaneous recovery range from minutes to an hour or more, depending upon conditions, thus placing them in the normal behavioral time domain.

The roles of several possible mechanisms were tested by the following simple experiments: (a) Electrical stimulation of afferent nerves with monitored neurograms ruled out changes in receptor function and nerve excitability, both for habituation and dishabituation. (b) Ventral root or efferent nerve recordings exhibited the same habituation and dishabituation as did muscle responses, ruling out muscle fatigue and neuromuscular changes. (c) Crucial participation of the gamma loop system was ruled out by section of all ventral roots and/or all dorsal roots, and by administration of Flaxedil: both habituation and dishabituation could still be obtained. (d) Stimulus generalization of habituation occurred to completely separate input nerves, ruling out changes in excitability of input afferent terminals. These experiments show that the essential mechanisms for habituation and dishabituation lie within the spinal cord.

Perhaps our most interesting data came from intracellular recordings of motor neurons participating in the flexion reflex being habituated. These data allowed us to test several hypotheses regarding the central mechanisms underlying habituation and dishabituation. Recordings were obtained using micropipettes filled with potassium chloride, citrate, or sulphate, from flexor spinal motor neurons identified by antidromic activation of muscle nerves.

The basic findings of the microelectrode studies are illustrated in Figure 43. Each response represents a series of approximately ten superimposed tracings recorded from a peroneal motor neuron. The upper lines show polysynaptic PSP's to cutaneous nerve stimulation (superficial peroneal N.) for control tests given once per minute (A), following habituation to a one per 3 sec. stimulus (B), and following recovery at 1/min. stimulation (C). There is a marked and significant decrease of PSP amplitude during habituation, followed by recovery to the control level. Note that not only do the EPSP's habituate, but that the IPSP components also decrease during habituation.

The lower line of tracings (D, E, F) shows interpolated monosynaptic test volleys (stimulation of the deep peroneal N.) given during each of the periods described above. That is, in the control and recovery series (A, and CF) each type of activation (polysynaptic and monosynaptic) was given once per minute, alternating every 30 seconds between the two types. In the habituated series (BF) the monosynaptic test activation was interpolated once a
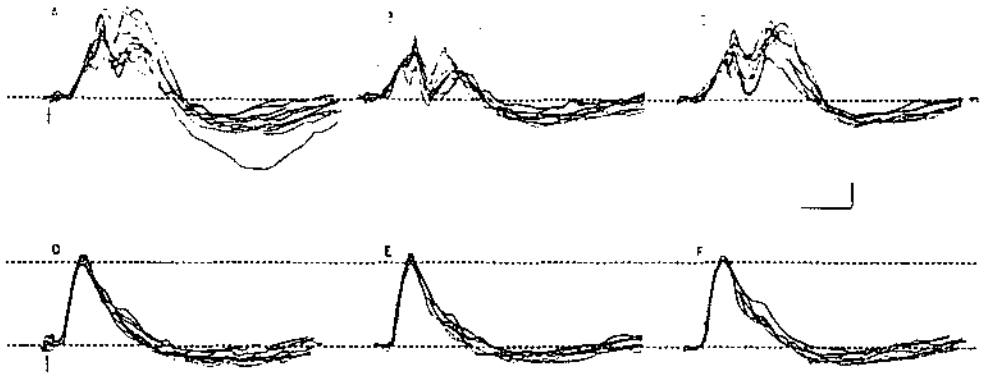
Figure 43. Intracellular responses (K citrate microelectrode) from identified peroneal motor neuron to polysynaptic (A, B, C; superficial peroneal N.) and monosynaptic (D, E, F; deep peroneal N.) activation. A: Control series, stimuli, 1/min.; B: during habituation training, stimuli 1/3 sec.; C: after recovery, stimuli 1/min.; D: monosynaptic tests interpolated 1/min. during control period (same time period as A); E: monosynaptic tests interpolated 1 min. during habituation training (obtained while the polysynaptic response was habituated to the level shown in B); F: monosynaptic tests interpolated 1/min. following recovery (same time period as C). Note decrease of both EPSP and IPSP components of the polysynaptic response (shown in B) after habituation training, but complete absence of any changes in the monosynaptic response. Calibration: 1 mV and 10 msec. (W. A. Spencer and R. F. Thompson, unpublished data.)

minute. There was no change in the monosynaptic EPSP as the polysynaptic PSP decreased. Consequently, there would seem to be no tonic change in the excitability of the motor neuron during habituation.

Interestingly enough, dishabituation by a strong stimulus (we used strong electric shocks or strong pinching of the skin) generally causes a significant increase in the monosynaptic test EPSP, as well as in polysynaptic PSP. There does seem to be a tonic increase in excitability during dishabituation. The influence of the gamma system had been ruled out in the situation, but an interesting point was raised: with the gamma system intact, the duration of the dishabituation effect is greater. The gamma system does seem to play a role in the time course of dishabituation.

Several lines of evidence tend to rule out phasic polysynaptic inhibition as a likely mechanism for habituation. Note in Figure 43 that the IPSP's of the polysynaptic responses decrease rather than increase during habituation. This suggests (but does not prove) that the amount of postsynaptic inhibition on the motor neuron is also "habituating" (i.e., decreasing). Since the postsynaptic responses are polysynaptic, there could be hidden IPSP's in the EPSP portions which might increase during habituation, thus leading to a net decrease in the size of the polysynaptic EPSP. To test this possibility, we obtained polysynaptic PSP's that were predominantly hyperpolarizing (i.e., mostly IPSP's) and reversed the polarity either by injecting chloride ions electrophoretically, or by electrically hyperpolarizing the cell. In both cases the inverted IPSP's *decreased* during habituation. Incidentally,

IPSP's from cells that showed purely hyperpolarizing responses also decreased during habituation. Thus it appears that phasic postsynaptic inhibition on the motor neuron is not the mechanism for habituation.

Finally, the possibility that pre- and postsynaptic inhibitory processes are occurring elsewhere in the system (i.e., in interneurons between input and motor neurons) can be tested with drugs. Strychnine abolishes several known instances of postsynaptic inhibition, and picrotoxin markedly reduces presynaptic inhibition (13). Administration of these drugs, given separately and in combination in doses sufficient to reduce or abolish both pre- and postsynaptic inhibition, has no significant effect on habituation or dishabituation. Consequently, we would suggest that pre- and postsynaptic inhibitory processes are not the neural basis of habituation.

As far as habituation is concerned, all of these data indicate that the decrease in response is the result of reduced input to motor neurons. The decrement must therefore occur in interneurons between input and output. Furthermore, results of the drug studies suggest that pre- and postsynaptic inhibitions are not involved. Our guess, and it is little more than a guess at the moment, is that the neuronal mechanism for habituation may be a polysynaptic analogue of the process of monosynaptic "low frequency depression". The latter appears to be a pre- or subsynaptic process (29), and bears some resemblance to the phenomena of polysynaptic response habituation. Dishabituation appears to be a separate superimposed sensitization process, possibly related to afterdischarge.

I have presented this material in order to illustrate both the advantages and limitations of the "model neural system" approach to the analysis of the synaptic basis of changes in behavior. We have been a great deal more successful in showing what the neural basis of flexion reflex habituation (and, hopefully, behavioral response habituation as well) is not, rather than what it is. Using some of the analytic tools now available from synaptic physiology, we were able to eliminate a number of possible hypotheses with some degree of confidence. However, we still cannot say what mechanisms do form the neural basis of response habituation.

Successful application of the "model neural system" approach is dependent both upon an understanding of synaptic processes in simplified systems and upon the choice of simplified neural systems that appear to show meaningful behavioral changes. Assuming that the latter requirement can be met, the rapid current progress in the field of synaptic physiology would seem to offer increasing possibilities for the "model neural system" approach to the analysis of neural mechanisms underlying behavior.

Galambos: Do you have measurements of any currents that might be flowing as a result of standing D.C. potentials in the spinal cord? Can the change in amplitude you see actually merely reflect a change with time in the standing potential of the spinal cord?

Thompson: I cannot give you a direct answer, since we did not measure

gross D.C. levels of the spinal cord. Our intracellular recordings were D.C. and no slow shifts were seen. The fact that the interpolated monosynaptic EPSP's in Figure 43 did not change would seem to rule out the possibility that shifts in the "standing potential" of the spinal cord are involved.

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