T-47

THE PRIMATE BRAIN AND HUMAN LEARNING

Karl H. Pribram

Stanford University

1969

Introduction

The opening of this conference on human learning has been entrusted to two presentations, one from a philosopher and the other from a neuroscientist whose experimental work has devolved mainly on non-human primates. The aim of these presentations must be to develop the problem which is primarily one in the behavioral sciences, by displaying its epistemological extent and by delving into its biological base. I take it as my task therefore to examine brain as the instrument of learning and to address specifically the question of what makes man's brain human.

Learning is a change in performance which comes about with experience. Experimental psychology has attempted to discover the laws of learning on the assumption that learning is all of a piece, that a paradigm such as classical or instrumental conditioning can be used to chart the route to discovery. At one level--I am tempted to say the rat level and will not resist the temptation--this assumption may prove valid. But at the rational, human level and even in non-human primates, studies of brain function in learning have shown the assumption to be of little use.

The primate brain is a complex organ composed of many systems and subsystems. Damage to one system influences some learning but not all; damage to another system will affect learning processes considerably different from those influenced

by injury to the first. In my laboratory we have therefore distinguished a variety of types of learning: some basic such as configural learning, discriminative learning, and learning to transfer experience gained in one situation to another; and some of a higher order such as the development of learning skills, and of linguistic learning--in other words, thinking. The laws of learning that apply to each type are considerably different as are the parts of the brain involved.

Configural Learning

Configural learning is difficult to separate out from discrimination learning. Yet some characteristics have made it fruitful to distinguish the two. The impetus for me to do so came from some work by Patrick Bateson, an ethologist at Cambridge University who came to my laboratories to become acquainted with more formal problem solving techniques. Bateson (1964) had done his thesis on the topic of imprinting and had shown that imprinting is a special case of perceptual learning. What he had done was to raise newborn chicks in an environment of either horizontal or vertical stripes and he showed that this early experience dramatically influenced subsequent imprinting. Thus it appeared that the development of an expectancy or neuronal model was as important to imprinting as to later perceptual performances (as shown by Sokolov, 1960). Once the model was established, orienting, imprinting, imitation, i.e. configural learning took place within one or at most a very few trials. Bateson then showed, at Stanford, that a similar type of non-problem oriented (latent) learning occurred in young monkeys. A pattern was placed in the animals' home cage

for three months. Then a discrimination task was given using this pattern in connection with a novel one and learning was compared to that obtained in a task where the novel one was matched to one which had previously been used in a problem solving situation. The "latently" learned cue proved easily as influential in determining behavior as did the "problem" learned one.

Direct evidence from brain recordings also confirms the independence of configural learning from problem guided learning. Records of the electrical activity evoked in the occipital (striate) cortex of monkeys show a differentiation of wave forms even when the animal is simply exposed to two different patterns (Spinelli, 1967), and before discrimination learning

[Figures 1 and 2]

has taken place (Pribram, Spinelli & Kamback, 1967). Because of the rapidity of configural learning we have not as yet completely followed this differentiation but have enough evidence to show that considerable sharpening of the difference in wave forms occurs over the course of repeated exposure to the patterns. We do have good evidence, however, (Grandstaff, & Gerbrandt, submitted) that the cortical electrical responses show habituation. Interestingly, both the differentiation and the habituation are highly individualistic. Although any specific electrode placement gives consistent and reliable recordings from day to day and week to week, different placements show markedly different electrical response patterns.

We have concluded therefore that at the cortex a configuration develops during perceptual learning and that perception is a function of this configuration.

Considerations which I have reviewed elsewhere (Pribram, 1966; 1969, in press) have led me to propose that this configuration resembles a holographic pattern. The critical evidence is the fact that extensive destructions of primary cortex do not interfere with pattern recognition except for the production of scotomata. The "memory" upon which recognition is based must therefore be distributed over the primary cortex and the perceptual recognition must therefore be constructed or composed from the distributed store. Direct neuroelectric evidence for such distribution comes from the experiments just cited. The concept of a neural hologram, that is, of sets of interfering wave forms constituted of postsynaptic potentials, provides a reasonable model that handles many hitherto unexplainable neurobehavioral data such as the lack of effect of epileptogenic lesions and cortical cross hatchings on perceptual performances and provides a solid base for the associative properties of recognition (Kraft, Obrist & Pribram, 1960; Stamm & Pribram, 1960; Stamm & Pribram, 1961; Stamm & Knight, 1963; Stamm, Pribram & Obrist, 1958; Stamm & Warren, 1961; Pribram, Blehert & Spinelli, 1966; Sperry, Miner & Meyers, 1955). The reconstructive process is, however, more complicated and for evidence on this we turn to discrimination learning.

Discriminative Learning

Discriminative or differentiative learning is distinguished from configural learning by its long time course. This distinction may be partially spurious, however. When discrimination learning curves are plotted in a backward direction from criterion, the learning process can be seen to be discontinuous: rapid increments in acquisition are separated from each other by plateaus of stationary performance (Blebert, 1966). To date we have found no brain lesion which will selectively influence the slope of the incremental portion of the discrimination learning curve. Impaired or deficient learning is always reflected in the length of the period of stationarity which may cover a multiplicity of ongoing processes necessary but not intrinsic to the discrimination function. More of this in a moment.

Evidence for a selective effect on discrimination learning is obtained, however, from another approach. Marked deficits occur when multiple alternatives are present in a situation to be discriminated (Pribram, 1960) or when the cues are multidemensional or vary along several parameters in one dimension (Butter, 1968), and the posterior (intrinsic) cortex associated with the various primary sensory systems is ablated. Under these conditions the monkeys with this brain damage sample fewer of the alternatives, fewer of the distinctive features of the cues. This deficit should be reflected in the backward learning curve paradigm when applied to complex discriminations but as yet this has not been adequately put to test.

The period of stationarity in such curves is in part attributed to hypothesis formation and testing (Zeaman & House, 1963). The suggestion has been made that eye movements, reflecting visual observing behavior or "attention," might prove a good indicator of the course of hypothesis testing. We have therefore undertaken studies of eye movement under conditions of discrimination learning (Bagshaw, Hackworth & Pribram, submitted) and found that in fact the duration of fixation on any one stimulus feature is shorter for the lesioned than for

[Figures 3 and 4]

the control monkeys although the number of items sampled is the same. Further analysis of these data is underway in an attempt to discover which specific strategy of hypothesis testing is impaired by these lesions. From the earlier studies already noted, the suggestion would be that these lesions influence the selective aspect of the sampling strategy (which searches and selects the distinctive features to be attended). But this needs to be tested further in the present series of experiments.

Transfer Learning

I mentioned above that a variety of brain resections will lengthen the period of stationarity of a discrimination learning curve. This is especially true when the subject is examined for the first time in a discrimination situation or when a variety of transfer tasks such as discrimination reversal and equivalence are given. In these situations lesions of the posterior intrinsic (nonassociated) cortex have little effect. By contrast, the frontolimbic formations of the forebrain pro

especially important to transfer learning. Again, as noted, the deficit following resections is seen in the prolonged

[Figure 5]

stationary period of the learning curve. We turn therefore to other behavioral analyses for suggestions as to which part of the overall hypothesis formation and testing strategy (the attentional mechanism) becomes impaired.

We take a monkey who has learned a discrimination task and ask him to transfer his experience to a situation in which one of the familiar cues is paired with a novel one (Schwartzbaum & Pribram, 1960; Bagshaw & Pribram, 1965). He will quickly master the new task unless he has a lesion of the limbic forebrain. If his hippocampi have been resected

[Figure 6]

the familiar cue will be normally effective only if it had previously been the rewarded one. The previously unrewarded cue will be reacted to as if it also were novel--as if it had been completely ignored in the original discrimination problem. Just the opposite occurs when a monkey has been amygdalectomized. Now effective familiarity relates to non-reward (S^{Δ} ; negative instances); the previously rewarded cue is treated as novel in the transfer situation (Douglas & Pribram, 1966).

A variety of other problem situations have demonstrated this relationship between hippocampus and the previously non-reinforced (non-salient) aspects of a situation and between amygdala and prior reinforcement. Multiple choice (Douglas,

[Figure 7]

Barrett, Pribram & Cerny, 1969) and distraction (Douglas & Pribram, in press) experiments have been especially illuminating.

[Figure 8]

In all instances, as in the reversal situation, whenever the reinforcing contingencies become insufficiently distinct, or the distractions sufficiently powerful, limbic lesioned subjects fail to persist in a strategy that had proved useful in prior situations. Attention and search are no longer directed (programmed) by previous experience; hypotheses are no longer pursued (Pribram, Douglas & Pribram, in press).

The frontolimbic forebrain has, of course, also been shown important to other recall tasks such as delayed response and delayed alternation (Pribram, Wilson & Connors, 1962; Pribram, 1961). All of these involve a temporal distribution of the reinforcing contingencies, the scheduling of feedback, the programming of the recurrent regularities, the temporal redundancies in the situation (Pinto-Hamuy & Linch, 1963; Pribram, Lim, Poppen & Bagshaw, 1966; Pribram & Tubbs, 1967).

[Figure 9]

One of the major tasks facing my laboratories now is to develop electrophysiological instruments with which to probe how these parts of the brain effect this programming.

Learning Skill

However, we have already made some initial steps in this direction. Thus both the selective and directive strategies involve input controlling functions of the brain. For instance, recovery functions in the primary visual and auditory systems have been influenced by electrical stimulations of the sensory specific associated and the frontolimbic systems (Spinelli & Pribram,

[Figure 10]

1966). This influence is a function of the attentive state of the monkey (Gerbrandt, Spinelli & Pribram, submitted).

[Figure 11]

Visual receptive fields have also been shown to become altered by such stimulation (Spinelli & Pribram, 1967). Finally, the

[Figure 12]

pathways from the associated and frontolimbic formations to the primary input systems have been in great part delineated (Reitz & Pribram, in press). Perhaps the most surprising finding of these studies is that input control is to a large measure effected through structures which had hitherto been thought of as regulating motor function.

This brings me to a consideration of the brain as the instrument with which we develop learning skill. The brain as we know it now is considerably different from the one that early learning theorists thought they were working with. Most formulations of learning depended heavily on the concept of associative strength based on contiguity and number. Configural variables were relegated to perception and perceptual learning was, until the past two decades, denied or ignored. Further, the configural and differentiative aspects of perceptual learning had not been teased apart. Nor, until recently, has the distinction between the selective and directive mechanisms of the learning process been adequately portrayed. The use of ROC analysis, perhaps more than any other device, has allowed this

portrayal. We are at present plotting data in these terms (Spevack, in preparation). Even here it is not altogether clear to what the directive (incentive, criterial) property of cues (their salience) is to be attributed. Is this property exclusively a function of their reinforcement history (including the genetic factors involved therein) as my data lead me to believe, or is a more extensive catalogue of attributes demanded?

An even more pervasive difficulty with classical learning theory is its dependence on the reflex-arc, stimulus ——→organism ——→ response model of brain function. We now know that the brain is organized along servomechanism principles. The discovery of the function of the γ efferent fibers of motor nerves made it necessary to modify our conceptions of the organization of the reflex and therefore of behavior. The data on input control cited above indicate that even the "highest" systems of the brain exert their influence via the input to the brain rather than via its output. In fact the control over input is exercised via motor structures which themselves influence behavior by "setting" the muscle spindle receptors by means of the γ loop. Servoprocesses are selective by virtue of tests, matches between configurations in memory and in input. Servoprocesses are directive by virtue of feedback. But as we have seen, feedback makes matching possible and matching (or mismatching) initiates feedback. There is a loop not an open arc in central nervous system organization. Sensory

functions are controlled by motor systems; behavior is regulated not by a piano keyboard control over muscle contraction but by servocontrol of the setting of muscle receptors. In such a brain learning is hierarchic and constructional: the brain must build up programs to organize perceptions and compose a behavioral repertoire. Associative learning plays a small role in the progressive development of programs. Laws of learning through configural matching and of the accretion of skills through practice (the development of subroutines) are demanded by what we know of today's brain. And experimental psychology has been singularly silent on these subjects.

Linguistic Learning

All of this the non-human primate brain has taught me. What then distinguishes man's brain, identifies him as human? The psychopathology of human memory processes--the amnestic syndromes--has almost universally been interpreted in terms of intracortical connections. All we have learned from experiments on non-human primate brains (e.g. the data noted above) mitigates against the importance of such connections. Either the interpretation of the basis for the amnestic syndromes in man is in error or else we have through our efforts stumbled on the difference between man's brain and that of his primate relatives. Thus it becomes paramount to review and test out once again, from this new vantage, the clinical evidence. The next decade promises to be an exciting one in this respect. My hunch is that linguistic skills just as other

learning skills will be found to rely heavily on the brain mechanisms responsible for configural learning and program development and not on those giving rise to associative chaining--what else can the evidence of the past decade of psycholinguistics convey to the student of brain function?

The converse of this approach should also prove fruitful. The crowning glory of man's brain is his linguistic mind. Experiments have already been initiated to test the linguistic abilities, one by one, of non-human primates. Where are the limits, the disparities that make the difference? Perhaps something about these can also be learned from a study of the development of linguistic capacities in children and from a relationship of earlier forms of linguistic structuring to earlier forms of brain organization.

In Conclusion

As detailed in the sections on learning skill and linguistic learning, I believe the key to all of these investigations is the fact that learning, and its operational counterpart remembering, is in large part the development of Configuration and Incentive, of Discrimination and Direction, in short operations of the brain. There has been in both psychology and biology and even more so in the simulation efforts of the computer sciences too great an emphasis on the quantitative aspects of memory storage to the exclusion of the equally important problem of efficiency. Efficiency depends on perception and on planning--on ways of coding information so as

to make it accessible. My data lead me to believe that all brains are prime coding instruments and that man's is distinguished by the power of his coding abilities. Just as ruminants spend their time munching cud, so man ruminates his codes. The resulting product is vastly different in the two cases: the beast's activity degrades structure into dung; man's productivity constructs and reconstructs his universe.

Research on brain functions has in these results shown me that to learn is to code, that learning is not mere associative storage but a productive activity making available alternatives. Multiple constructions, options among alternatives, these are the hallmarks of human learning. Thus enriched through learning man's brain, in time, creates his freedoms.

References

Bagshaw, M. H. and Pribram, K. H. Effect of amygdalectomy on transfer of training in monkeys. J. comp. physiol. Psychol., 1965, 59, 116-121.

Bateson, P. P. G. Changes in chicks' responses to novel moving objects over the sensitive period of imprinting. Animal Behav., 1964, 12, 479-489.

Blehert, S. R. Pattern discrimination learning with rhesus monkeys. Psych. Reports, 1966, 19, 311-326.

Butter, C. M. The effect of discrimination training on pattern equivalence in monkeys with inferotemporal and lateral striate lesions. Neuropsychologia, 1968, 6, 27-40.

Douglas, R. J., Barrett, T. W., Pribram, K. H. and Cerny, M.C. Limbic lesions and error reduction. J. comp. physiol. Psychol., 1969, 68, 437-441.

Douglas, R. J. and Pribram, K. H. Distraction and habituation in monkeys with limbic lesions. J. comp. physiol. Psychol., 1969, 68, 203-209.

Gerbrandt, L. K., Spinelli, D. N. and Pribram, K. H. Recovery cycles and evoked response measures of excitability in primate striate cortex. Electroenceph. clin. Neurophysiol. submitted).

Grandstaff, N. and Gerbrandt, L. K. Habituation I: Analysis of changes in evoked potentials with visual stimulation. Electroenceph. clin. Neurophysiol. (in press).

Kraft, H., Obrist, W. D. and Pribram, K. H. The effect of irritative lesions of the striate cortex on learning of visual discrimination in monkeys. J. comp. physiol. Psychol., 1960, 53, 17-22.

Pinto-Hamuy, T. and Linck,P. Effect of frontal lesions on performance of sequential tasks by monkeys. Exp. Neurol. 1965, 12, 96-107.

Pribram, K. H. The intrinsic systems of the forebrain. In J. Field, H. W. Magoun and V. E. Hall (eds.) Handbook of Physiology, Neurophysiology II. Washington, American Physiological Society, 1960, 1323-1344.

Pribram, K. H. Some dimensions of remembering: steps toward a neuropsychological model of memory. In J. Gaito (ed.) Macromolecules and Behavior. New York, Academic Press, 1966, 165-187.

Pribram, K. H. The neurophysiology of remembering. Scientific American, 1969, 220, 73-86.

Pribram, K. H. Four R's of remembering. In K. H. Pribram (ed.) On the Biology of Learning. New York, Harcourt, Brace and World (in press).

Pribram, K. H. A further experimental analysis of the behavioral deficit that follows injury to the primate frontal cortex. Exp. Neurol., 1961, 3, 432-466.

Pribram, K. H., Blehert, S. R. and Spinelli, D. N. Effects of visual discrimination of crosshatching and undercutting the inferotemporal cortex of monkeys. J. comp. physiol. Psychol., 1966, 62, 358-364.

Pribram, K. H., Douglas, R. J. and Pribram, B. J. The nature of non-limbic learning. J. comp. physiol. Psychol. (in press).

Pribram, K. H., Lim, H., Poppen, R. and Bagshaw, M. H. Limbic lesions and the temporal structure of redundancy. J. comp. Physiol. Psychol., 1966, 61, 368-373.

Pribram, K. H., Spinelli, D. N. and Kamback, M.C. Electrocortical correlates of stimulus response and reinforcement. Science, 1967, 157, 94-96.

Pribram, K. H. and Tubbs, W. E. Short-term memory, parsing and the primate frontal cortex. Science, 1967, 156, 1765-1767.

Pribram, K. H., Wilson, W. A. and Connors, J. The effects of lesions of the medial forebrain on alternation behavior of rhesus monkeys. Exp. Neurol., 1962, 6, 36-47.

Reitz, S. L. and Pribram, K. H. Some subcortical connections of the inferotemporal gyrus of monkey. Exp. Neurol. (in press).

Schwartzbaum, J. S. and Pribram, K. H. The effects of amygdalectomy in monkeys on transposition along a brightness continuum. J. comp. physiol.Psychol., 1960, 53, 396-399.

Sokolov, E. N. Neuronal models and the orienting reflex. In M.A.B. Brazier (ed.) The Central Nervous System and Behavior. New York, Josiah Macy, Jr. Foundation, 1960, 187-276.

Sperry, R. W., Miner, N. and Meyers, R. E. Visual pattern perception following subpial slicing and tantalum wire implantations in the visual cortex. J. comp. physiol. Psychol., 1955, 48, 50-58.

Spevack, A. and Pribram, K. H. ROC analysis of the effects of limbic lesions (in preparation).

Spevack, A. and Pribram, K. H. ROC analysis of the effects of lesions of the inferotemporal cortex (in preparation).

Spinelli, D. N. Evoked responses to visual patterns in area 17 on the rhesus monkey. Brain Research, 1967, 5, 511-514.

Spinelli, D. N. and Pribram, K. H. Changes in visual recovery functions produced by temporal lobe stimulation in monkeys. Electroenceph. clin. Neurophysiol., 1966, 20, 44-49.

Spinelli, D. N. and Pribram, K. H. Changes in visual recovery function and unit activity produced by frontal cortex stimulation. Elecgroenceph. clin. Neurophysiol., 1967, 22, 143-149.

Stamm, J. S. and Knight, H. Learning of visual tasks by monkeys with epileptogenic implants in temporal cortex. J. comp. physiol. Psychol., 1963, 56, 254-260.

Stamm, J. S. and Pribram, K. H. Effects of epileptogenic lesions in frontal cortex on learning and retention in monkeys. J. Neurophysiol., 1960, 23, 552-563.

Stamm, J. S. and Pribram, K. H. Effects of epileptogenic lesions in inferotemporal cortex on learning and retention in monkeys. J. comp. physiol. Psychol., 1961, 54, 614-618.

Stamm, J. S., Pribram, K. H. and Obrist, W. The effect of cortical implants of aluminum hydroxide on remembering and on learning. Electroenceph. clin. Neurophysiol., 1958, 10, 786 (abstract).

Stamm, J. S. and Warren, A. Learning and retention by monkeys with epileptogenic implants in posterior parietal cortex. Epilepsia, 1961, 2, 220-242.

Zeaman, D. and House, B. J. The role of attention in retardate discrimination learning. In N. R. Ellis (ed.) Handbook of Mental Deficiency. New York, McGraw-Hill, 1963, 159-223.

Fig. 1

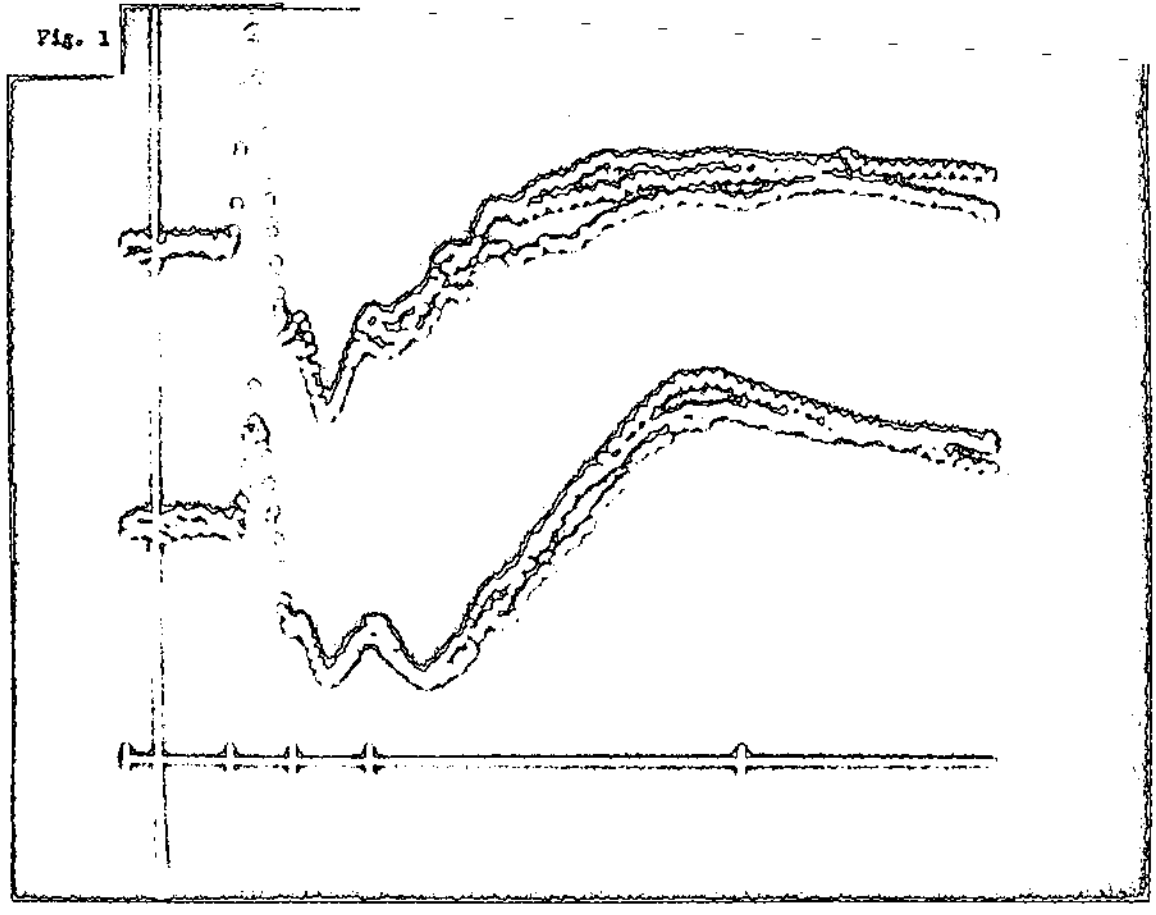
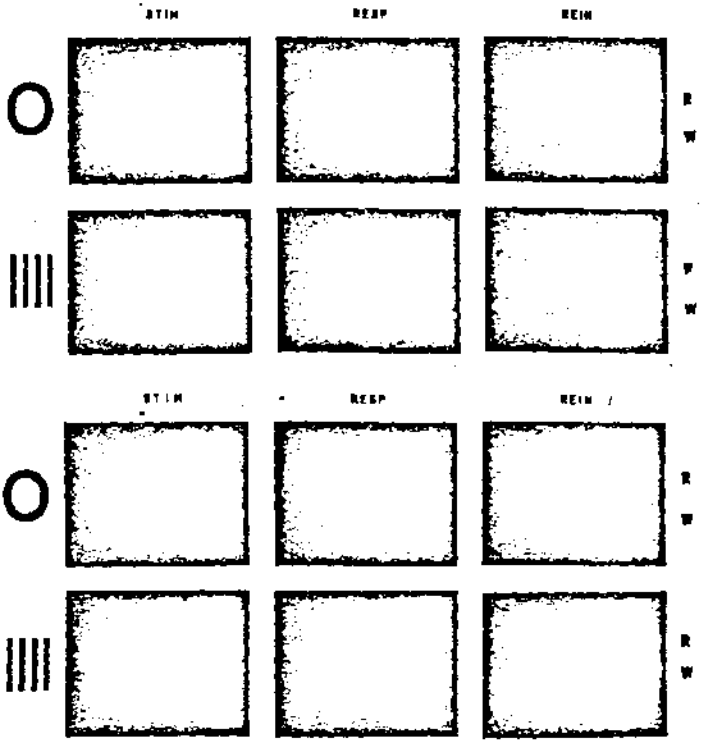


Fig. 2



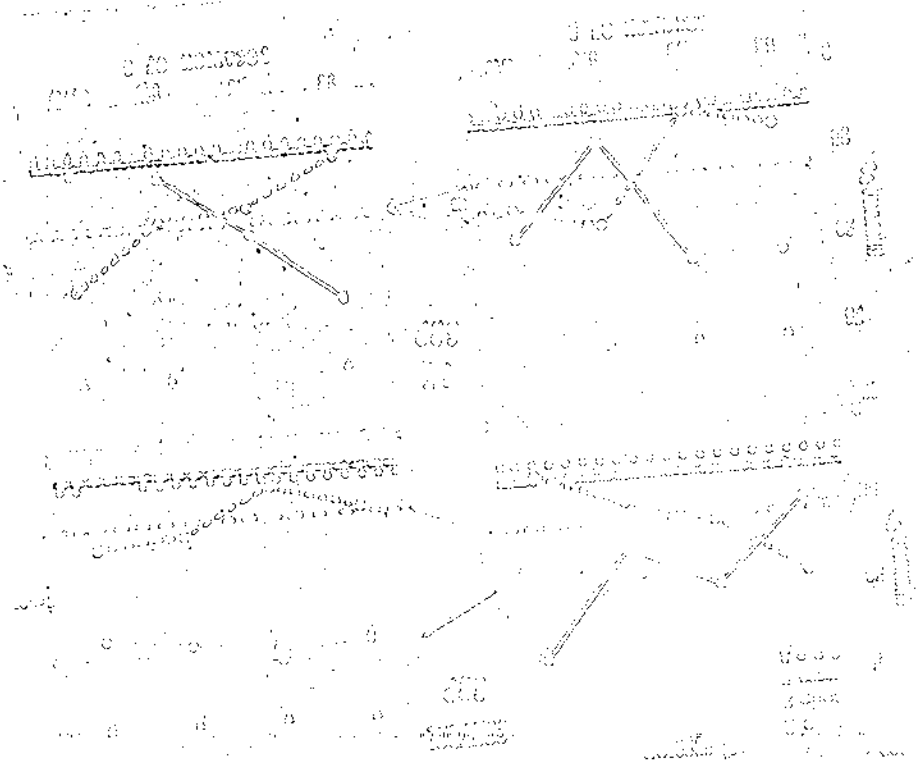
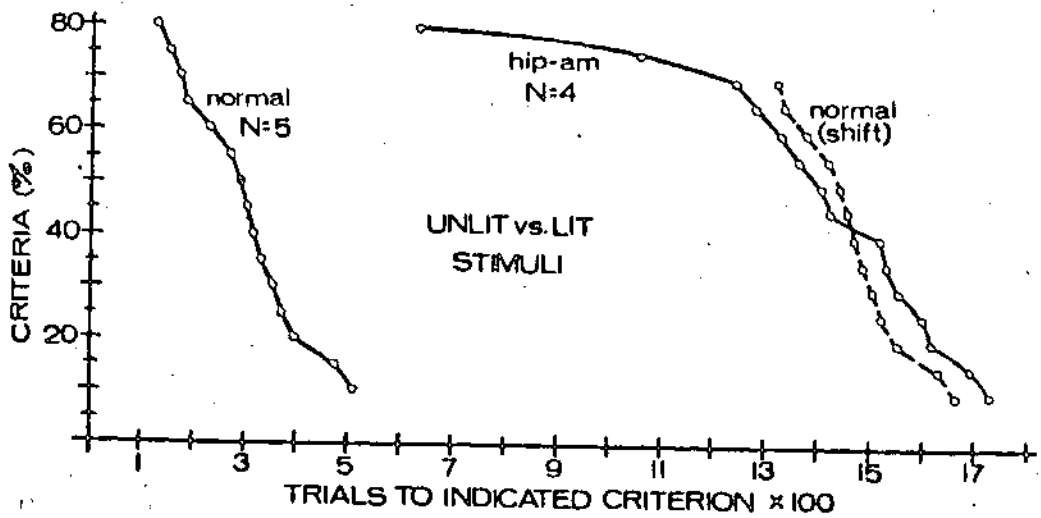


Fig. 3


80
60
40
20
CRITERIA (%)
normal
N=5
hip-am
N=4
normal
(shift)
UNLIT vs. LIT
STIMULI
1
3
5
7
9
11
13
15
17
TRIALS TO INDICATED CRITERION x 100

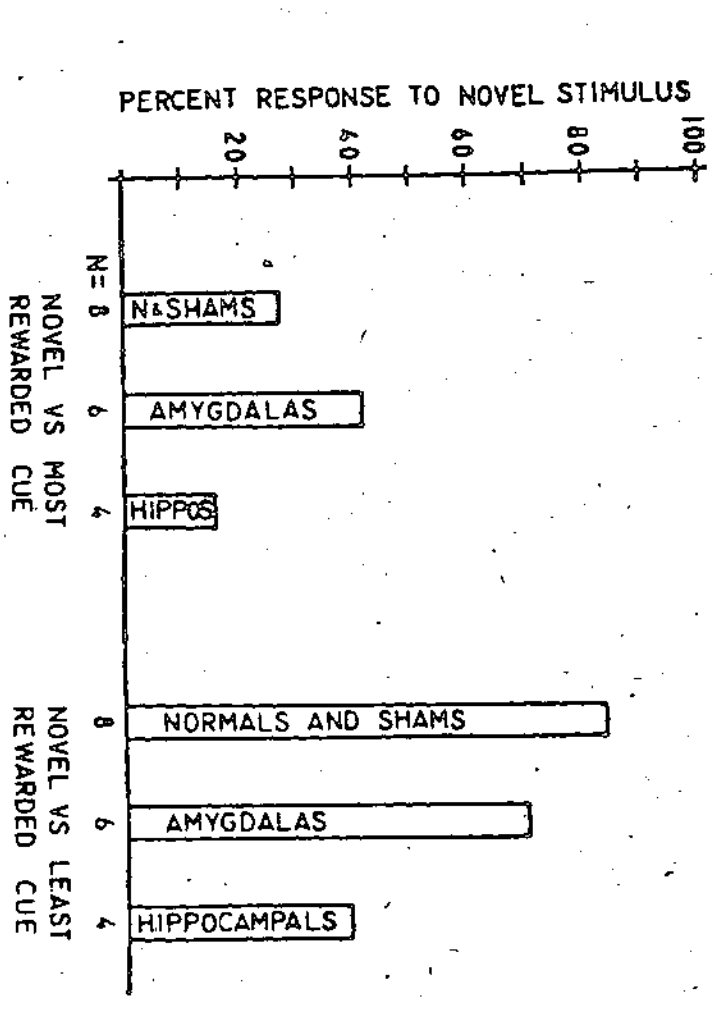

PERCENT RESPONSE TO NOVEL STIMULUS
20
40
60
80
100
NOVEL VS MOST REWARDED CUE
N=
8
N&SHAMS
6
AMYGDALAS
4
HIPPOS
NOVEL VS LEAST REWARDED CUE
8
NORMALS AND SHAMS
6
AMYGDALAS
4
HIPPOCAMPALS

Fig. 6

Fig. 7

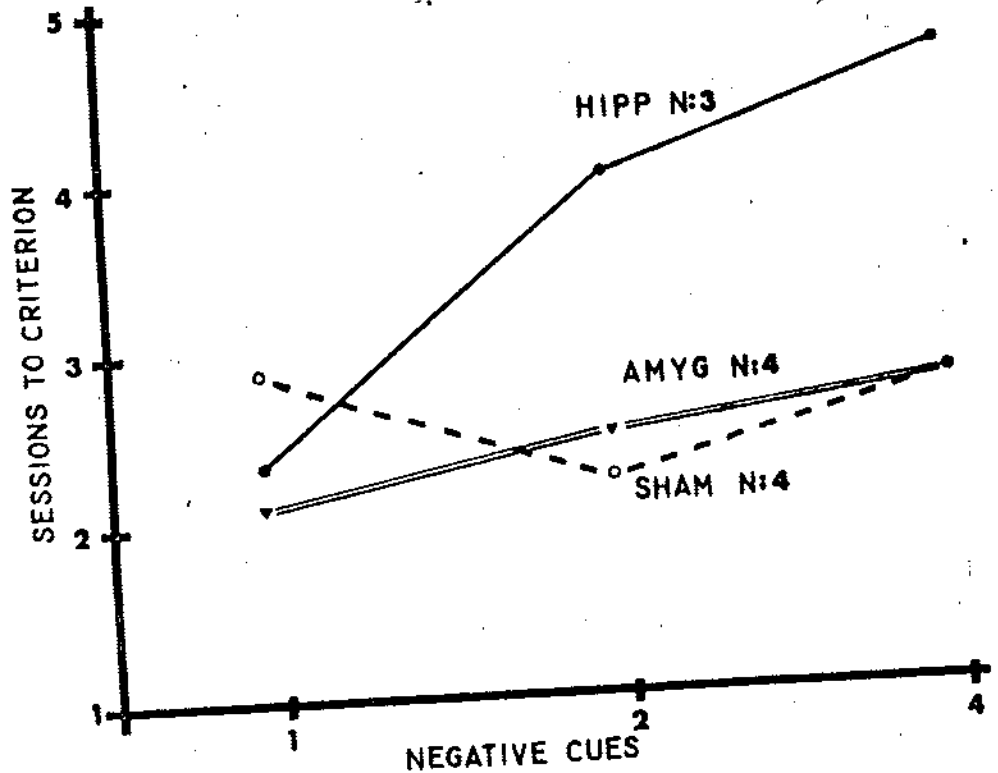

5
4
3
2
1
SESSIONS TO CRITERION
HIPP N:3
AMYG N:4
SHAM N:4
1
2
4
NEGATIVE CUES

Fig. 8

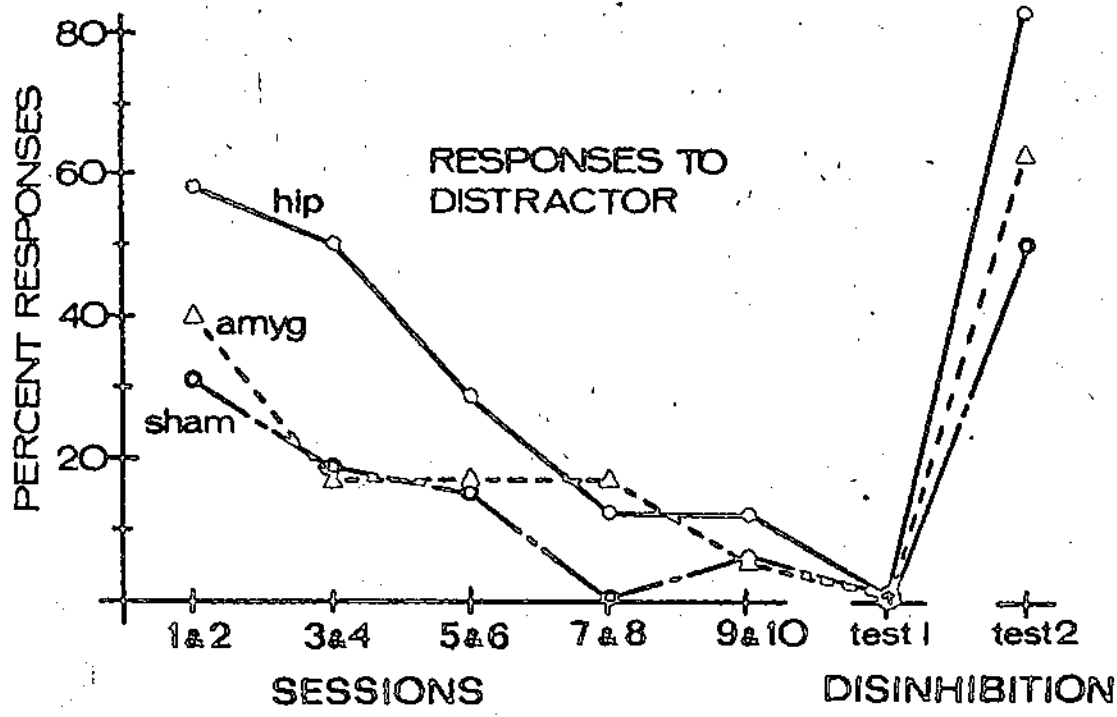

RESPONSES TO
DISTRACTOR
hip
amyg
sham
PERCENT RESPONSES
80
60
40
20
1&2
3&4
5&6
7&8
9&10
test I
test2
SESSIONS
DISINHIBITION

Fig. 9

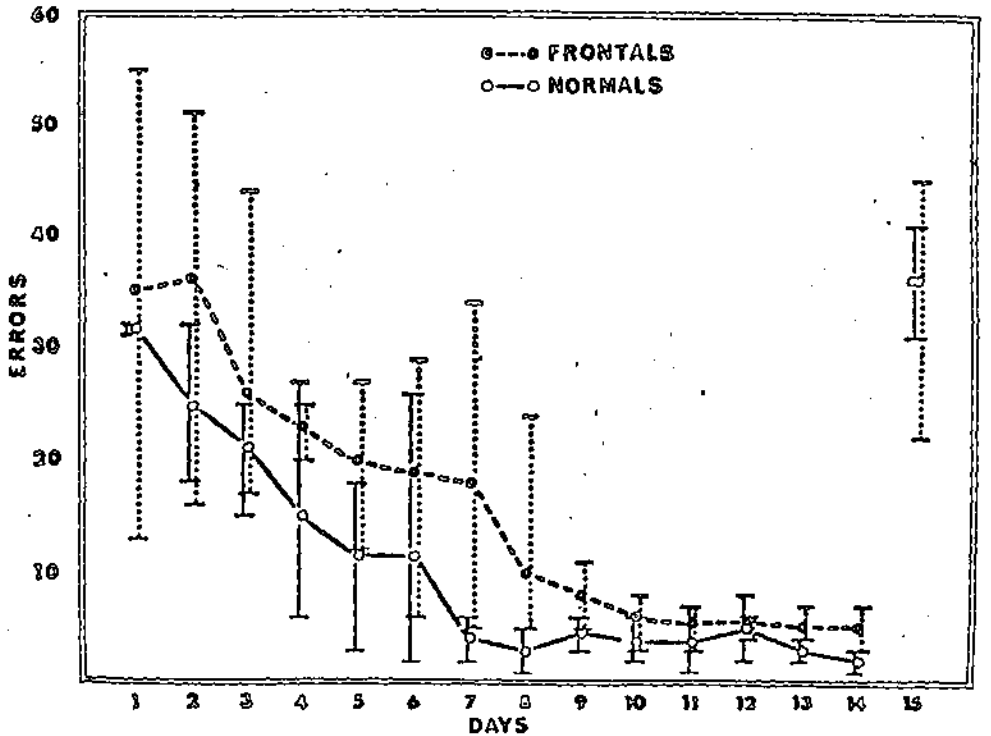

o---o FRONTALS
o—o NORMALS
ERRORS
60
50
40
30
20
10
1
2
3
4
5
6
7
8
9
10
11
12
13
14
15
DAYS

Fig. 10

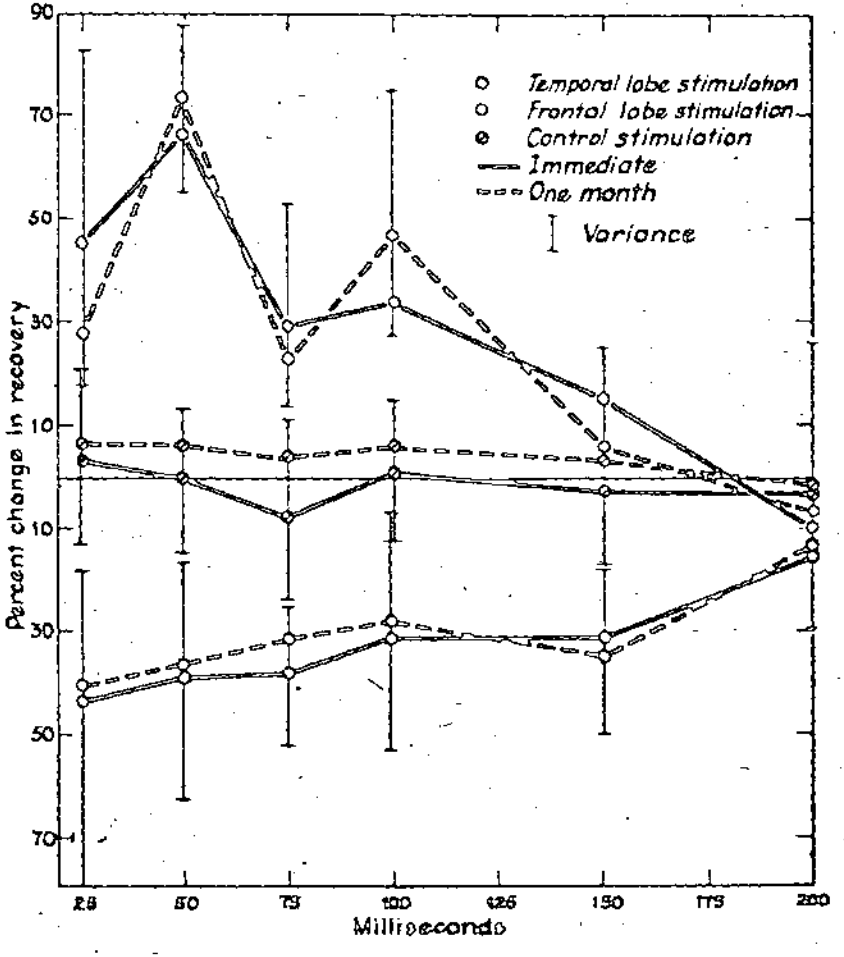

Temporal lobe stimulation
Frontal lobe stimulation
Control stimulation
Immediate
One month
Variance
Percent change in recovery
90
70
50
30
10
10
30
50
70
25
50
75
100
125
150
175
200
Milliseconds

Figure Legends

Figure 1

From top to bottom: (a) three averaged waveforms obtained when the circle was flashed (50 responses each) are shown superimposed; (b) three averaged waveforms, obtained when the stripes were flashed. The vertical marker shows when the stimulus was presented; on the horizontal marker the breakpoints are shown. Statistical analysis shows that the greatest and most significant difference between the two waveforms is in component No. 4, as was also apparent from visual inspection. (From Spinelli)

Figure 2

Averaged recordings of electrical activity obtained from occipital cortex of monkeys performing a differential discrimination: circle as opposed to vertical stripes. A standard 500 msec. of activity is represented in each trace; the amplitude represented is variable, however, and depends on how many signals were averaged in order to make the record; for example, many more signals were obtained when the monkey made a correct response than when he made an error during criterion performance. The records under STIM are the waveforms evoked by a display lasting 1 msec.; the records under RESP were generated just prior to the response; the records under REIN were generated after the response and during the period when reinforcing events occurred. The upper six panels were made from records obtained while the monkey was performing at chance; the lower six panels were made from records obtained after the monkey attained an 85 percent criterion (200 consecutive trials). The records in line with R were made when the monkey performed correctly; those in line with W were made when the monkey was wrong. The waves generated just prior to response (the intention waves) are similar whenever

the monkey is about to press the right half of the panel, regardless of whether this response proves to be correct or wrong.

Figure 3

One typical frame of 16 mm movie film showing one of the displays reflected on the cornea when a monkey was fixating on the numeral 8. (From Hackworth and Bagshaw)

Figure 4

Distribution of fixations of the right eye during the first (left side) and fourth (right side) sessions of training a control group (upper figures) and an inferotemporal lesion group (lower figures) to preferentially fixate the figure "8" as opposed to the figure "3". The control group succeeded by the fourth session while the IT group failed. Note that the IT group adopted a position preference for the SE (right lower quadrant) as indicated on the abscissa.

Figure 5

Reduction of presses of unlit panels. Y axis shows criteria decreasing by 5 percent intervals, and X axis shows mean trials to each criterion. Dotted line is normal curve superimposed on lesion group curve.

Figure 6

Responses to novel vs. rewarded stimuli.

Figure 7

Graph of the results of changing the number of negative cues in a set of discrimination problems. Note the effect on the hippocampectomized monkeys.

Figure 8

Overt responses to the distracting stimulus.

Figure 9

Graph of the average number of errors made by monkeys having ablations of the frontal cortex and by their controls. Bars indicate ranges of errors made. For day 15 are shown records of the number of errors made on the return to the classical 5-sec. alternation task.

Figure 10

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

Figure 11

A record of flash recovery, after either small or large responses in the striate cortex produced by the LGB probe stimulation, is shown at four interflash intervals (60, 90, 120, and 180 msec.) in § 29. Marks on the time axis below each pair of waveforms indicate the onset of the response to each flash. The amplitude calibration marker represents a 100 μv deflection.

Figure 12

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