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REPRINTED FROM
ADV. IN THE STUDY OF BEHAVIOR, VOL. 2

ACADEMIC PRESS INC., NEW YORK

The Neurobehavioral Analysis of Limbic Forebrain Mechanisms: Revision and Progress Report

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I. INTRODUCTION

The experimental analysis of the limbic forebrain began in earnest with the report by Klüver and Bucy (1937) that temporal lobectomy in monkeys produces a syndrome that includes taming, hyperoralism, hypermetamorphisis, psychic blindness, and hypersexuality. These dramatic results had on an earlier occasion (1888) been obtained by Brown and Schafer, but had somehow failed to engage further neurobehavioral exploration. The Klüver–Bucy experiments were quickly followed by those of Spiegel and his collaborators (1940) who produced rage by semichronic stimulations of the amygdala.

Simultaneously, a second avenue of interest was initiated by Papez (1937) and substantiated by Brodal (1947), who, on the basis of anatomical considerations, made the proposal that the circuitry of the limbic formations of the forebrain was incompletely described by its relation to olfaction; that indeed, this circuitry might constitute an ideal substrate for the emotional process.

During this decade another series of experiments was set in motion by the contributions of Bard and Mountcastle (1947) who demonstrated that ablations limited to the mediobasal structures included in the limbic systems affected the aggressiveness-tameness dimension of behavior. These investigators used cats as subjects and, contrary to the observations of Klüver and Bucy, rage was the result of removals of the medial

part of the temporal lobe—specifically, the amygdaloid complex—and hypersexuality did *not* occur. However, in other laboratories (Schreiner and Kling, 1953), amygdalectomy in cats *did* lead to taming and hypersexuality.

These three lines of evidence converged in the necessity for a systematic neurobehavioral analysis of the functions of the medial and basal portions of the forebrain, and for a contrast of these functions with those of the lateral isocortical mantle. To this end a program of experiments was carried out, much of it in my laboratories: (1) Klüver and Bucy's observation that "psychic blindness" follows temporal lobectomy was shown, in large part, to result from removal of the inferolateral isocortex of the temporal lobe and *not* to be related to mediobasal structures (Ades and Rabb, 1949; Blum *et al.*, 1950; Chow, 1951, 1952, 1954; Harlow, *et al.*, 1952; Mishkin, 1954; Mishkin and Pribram, 1954; Pribram, 1954; Riopelle and Ades, 1953; Riopelle *et al.*, 1951). (2) Disturbances of a similar nature in other sensory modes were produced by selective removals of isocortical areas in adjacent parts of the temporal, occipital, and parietal lobes (Bagshaw and Pribram, 1953; Blum *et al.*, 1950; Pribram and Barry, 1956; Weiskrantz and Mishkin, 1958; Wilson, 1957; Wilson *et al.*, 1960). (3) The mediobasal cerebral formations were subjected to physiological (by the method of strychnine neuronography) as well as anatomical classification (Lennox *et al.*, 1950; MacLean and Pribram, 1953; Pribram and Kruger, 1954; Pribram *et al.*, 1950; Pribram and MacLean, 1953). (4) The physiological effect of electrical excitation of the mediobasal cortex was thoroughly explored (Kaada, 1951; Kaada *et al.*, 1949; Livingston *et al.*, 1948; Pribram, 1961; Wall and Davis, 1951; Ward, 1948), with the result that the anterior portions of the limbic forebrain could be conceptualized to form a mediobasal motor cortex by contrast to the classical precentral motor cortex (Pribram, 1961).

II. The Visceral Brain Hypothesis

The contribution of this wealth of experimental evidence can be summarized as follows: The limbic structures of the forebrain appear to control those physiological and behavioral processes that are also controlled by various other core brain stem structures—e.g., hypothalamic and midline mesencephalic mechanisms. The conception that the limbic forebrain is an olfactory brain gave way to the idea that the limbic systems function as a "visceral" brain which regulates the instincts of self and species preservation (MacLean, 1949).

Some serious objections to this view immediately became apparent. If indeed the limbic systems exert their control largely via autonomic and visceral activities, this should describe a fairly exclusive relationship. The effect of stimulation of the mediobasal motor cortex on somatic and other striped muscles should be minimal. This is only partially so. True, the effects of this stimulation are not as precise as those obtained from the precentral motor cortex. However, eye movements, vocalization, head and body turning, and extension of extremities *are* obtained from mediobasal motor cortex excitation (Gloor, 1960; Kaada, 1951; Kaada *et al.*, 1949; Pribram, 1961). Conversely, the effects on autonomic and visceral activities of stimulation of the precentral motor cortex are well established (Bucy and Pribram, 1943; Fulton, 1951; Wall and Pribram, 1950). Perhaps a more-or-less exclusive relation between limbic formations and visceral-autonomic functions could be established on the basis of afferent connections. Vagal stimulation certainly gives rise to changes in the electrical activity of limbic structures (Bailey and Sweet, 1940; Dell, 1952; MacLean and Pribram, unpublished results quoted in Fulton, 1951). However, this change of electrical activity has also been observed to result from visual, auditory, and somatic nerve stimulations (Cadilhac, 1955; MacLean *et al.*, 1952).

These observations made it unlikely that the functions of the limbic systems could be simply described by the concept "visceral" brain. The relation between the limbic forebrain and visceral activities is *not* exclusive. Some more complicated connection between this part of the brain and "instinctive, self and species preservative" behaviors must obtain.

III. The Memory Hypothesis

In addition, ablations of the medial portions of the temporal lobe in man were found to produce a peculiar defect in memory – patients with such lesions were unable to recall their whereabouts at any given period after surgery, unable to remember interviews or even the fact that they had been interviewed. This occurred despite the fact that their performance on tests of digit span remained intact (Milner, 1954, 1958). Visceral and emotional disturbances were minimal.

And so, just as in the case of frontal lobe function (see Pribram *et al.*, 1964), two disparate views of the functions of the limbic systems came into vogue: one, that the memory mechanism is essentially involved; the other, that motivation and emotion, based on instinctive, self and species preservative processes, are crucially implicated. On the whole, both

views have proved popular and persuasive, and little has been done to reconcile them. The anatomical proximity of limbic structures and the hypothalamic and mesencephalic brain stem formations support one; the data on man the other.

IV. The Homeostat Hypothesis

Thus both laboratory and logic dictated dissatisfaction with these views (Miller *et al.*, 1960; Pribram, 1958, 1960, 1961). Most likely this dissatisfaction was enhanced by an inability to visualize precisely a mechanism by which sex and selection, aggression and alternation, rearing and remembering, ingestion and incestion, could all be controlled by the operations of limbic formations. Perhaps the dissatisfaction would be dispelled if the structure of such a mechanism could be worked out. And perhaps, also, the obvious discrepancies could in this manner be resolved.

I would like, therefore, to attempt to construct, to the extent that this is possible, a model based on neurobehavioral data gathered as a result of this dissatisfaction and to see what additional evidence is needed to make the picture clear.

The general thesis may be put forward in this manner: *The limbic formations partake in the neural organization of the homeostatic regulations of the organism.* So stated, this thesis has a superficial resemblance to the very notions already found unsatisfactory—but it is important to note this resemblance as an acceptable starting point common to a great many investigators in this area of research.

The differences between the suggestion made here and earlier ones become apparent only when specific hypotheses are made about the reach of what is meant by "homeostatic regulations," and about the way in which the limbic systems "partake."

Our earlier work (Fulton *et al.*, 1949; Pribram and Bagshaw, 1953; Pribram and Fulton, 1954; Pribram and Weiskrantz, 1957; reviewed in Pribram, 1958)—confirmed and amply supported by the results of others (Glees *et al.*, 1950; Hunt and Diamond, 1957; Smith, 1944; Stamm, 1955; Ward, 1948; for references to recent work, see Teitelbaum and Milner, 1963)—had established that whenever any of the limbic formations of the forebrain are damaged or artificially stimulated, only a selective range of behaviors is disturbed. These behaviors can be grouped under the categories of fighting, fleeing, feeding, and sex: the *four Fs* that enlarge on Cannon's fight and flight reactions. These instinctive, self and species preservative behaviors must, we reasoned, have some property in common if they are served by a common, albeit

internally diverse, neural substrate. The hypothesis that visceral-autonomic regulation is that property had served us well in generating direct experimental tests; however, as already indicated, the results of these experiments fell short by a considerable margin of fully supporting the hypothesis.

V. Behavior Sequences

In the search for alternatives, one in particular seemed worth pursuing. All of the behaviors concerned—fighting, fleeing, feeding, mating and maternal are directional, i.e., they demand the execution of sequences of actions, sequences in which an appropriate act in the sequence depends on the proper completion of an activity at an earlier point in the sequence. An example of the disorganization of maternal behavior that results from median cortex lesions points up this view. Stamm (1955) placed a mother rat with its young in an enclosure. A nest was in one corner of the enclosure. The babies were strewn about. A record was made of the time taken by the mother to retrieve her brood into the the nest. Next, lesions were made of the limbic median cortex, involving the cingulate gyrus of the mother. The mother was again placed in the situation, and the time for retrieval noted. After brain damage, retrieval time was extended indefinitely; and when the behavior of the mother was observed directly, the reason was clear to see. She picked up one infant, took it to the nest, fetched another, dropped it half-way back to the nest, picked up the one already safely ensconced and removed it on her way to still a third baby, etc. At the end of 15 minutes the infant-nest field was as disorganized as it was at the beginning, yet the individual actions of the mother had all been diligently, carefully, and skillfully accomplished.

The first question to be answered was therefore: Is the relation between the limbic forebrain and behavior sequences an exclusive one? If behavior sequences are disturbed only when these occur in the categories of instinctive, self and species preservative behavior, the argument loses force. If in addition, limbic lesions disturb behaviors that do *not* partake of the properties that critically determine behavior sequences, the hypothesis must be abandoned.

An experimental answer to the first of these questions was obtained: Behavior sequences that cannot be classed as self or species preservative *are* affected by limbic system lesions. The most rudimentary of such behaviors is a simple alternation task. The completion of one activity in the sequence (e.g., obtain a peanut from the left of two identically covered

food wells) must be accomplished before the next activity (obtain a peanut from the *other* well) in a sequence can be properly defined and undertaken. The effect of limbic system resections on alternation behavior was assessed in two experiments (Pribram *et al.*, 1962b; 1966).

The results were clear-cut. Resection of any of these limbic structures (hippocampal, cingulate, or orbitoinsolotemporal regions) impairs either the retention of a preoperatively learned delayed alternation performance or its initial learning, or both (Fig. 1 and Tables I–IV).

This simple case of a behavior sequence might possibly still be a case of instinctive, self and species preservative behavior—a laboratory example of hoarding behavior. To demonstrate greater generality, a more complicated sequence needed to be tested. An apparatus [Discrimination Apparatus for Discrete Trial Analysis (DADTA)], a small special purpose computer, was therefore designed in which one could readily assess behavior in more complex situations (Pribram *et al.*, 1962a).

The DADTA apparatus was so constructed that some information could also be simultaneously obtained about the second, or "negative," possibility: viz., that limbic system damage interferes with some basic property which is reflected in behavior other than the execution of behavior sequences:

Alternation defects have been attributed to impaired "recent memory." As already noted, a peculiar memory loss follows resection of the medial parts of the temporal lobe of man. The candidate most likely to defeat the hypothesis, therefore, was some form of general defect in immediate memory. I had already completed (Pribram, 1963a, 1964) an experiment in which monkeys were trained to perform in an operant conditioning situation on a 15-minute fixed-interval schedule of reinforcement. Removal of the hippocampal formation bilaterally failed to alter the "scallop" of the performance curves of two monkeys so trained; in addition, two monkeys were trained *after* bilateral hippocampectomy, and again their performance curves could not be distinguished from those of normal subjects. Furthermore, these lesioned monkeys attained smooth performance curves within the same number of days (100–160) of testing (6-hour sessions, 6 days a week) as did four controls (120–240 days). (See Fig. 2 for reconstruction of lesions.)

However, the argument can be made that the operant situation that uses a fixed-interval schedule of reinforcement is not the best test of memory functions—response chaining is accomplished in such closely approximated intervals that the events involved in making the response itself could bridge the temporal gap, especially since the situation is an extremely simple one.

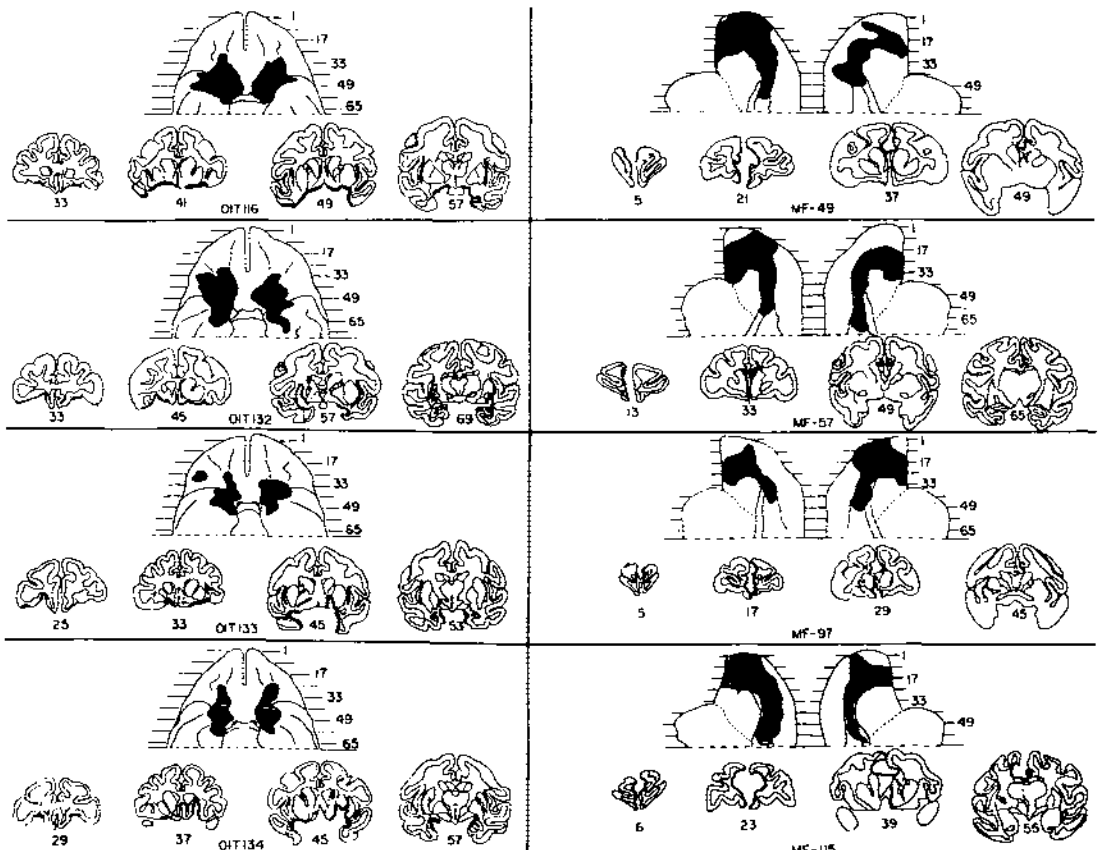


FIG. 1. Reconstructions of extent and sample cross sections of depth of cingulate and orbitoinsulotemporal resections. Lesions indicated by solid areas.

The DADTA apparatus was therefore equipped to allow variations of the temporal interval between actions; in other words, to make possible the spacing of the trials taken to solve problems.

Experiments were thus specifically designed to test the performance of monkeys on problems in which trials can be massed or spaced by varying the intertrial interval. In addition, the apparatus was used to

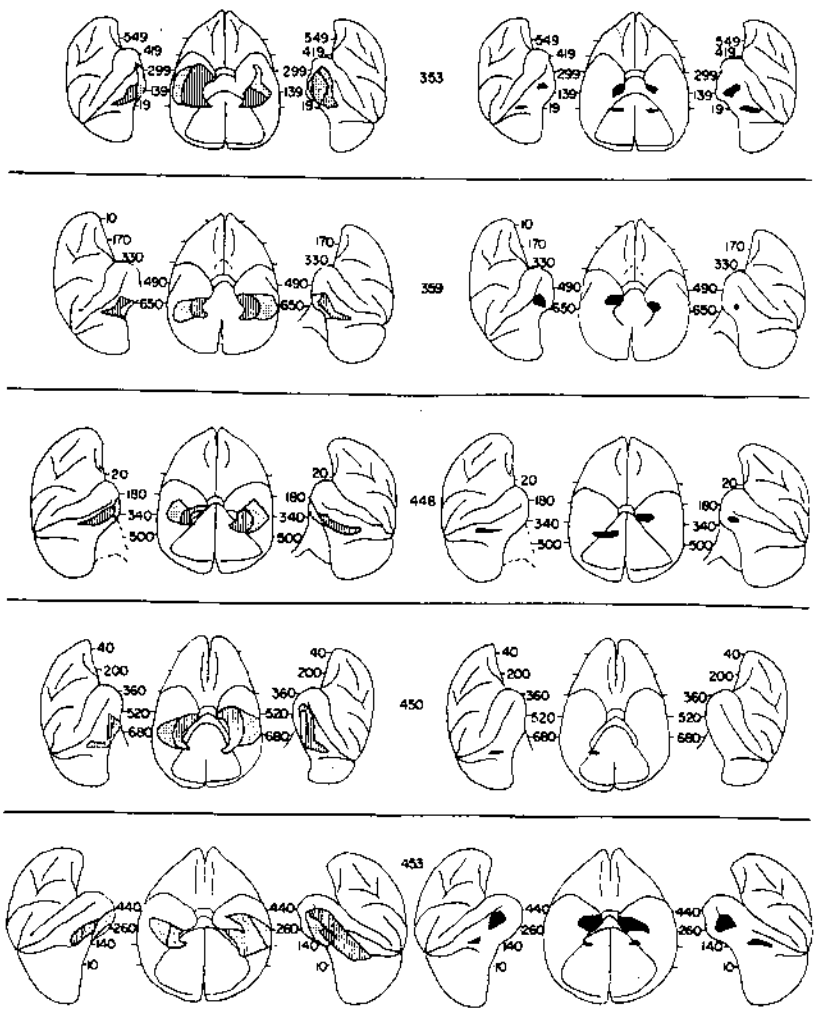


FIG. 2. Reconstructions of the extent of lesion (left) and the extent of remnants (right), after bilateral resections of the hippocampal formation. Stipled area indicates damage to superficial cortex; striped area indicates resection in depth. The remnants of spared portions of the hippocampal formation appear as solid areas.

TABLE 1
CLASSICAL ALTERNATION

	Preoperative learning to criterion		Postoperative relearning to criterion		Preoperative savings on retention		Postoperative savings on relearning	
Animal	Days	Errors	Days	Errors	Days	Errors	Days	Errors
MFC 49	16	99	8	8	14	99	8	91
MFC 57	27	257	2	3	21	253	25	254
MFC 97	5	9	4	8	3	9	+1	1
MFC 115	10	24	2	19	−2	0	8	+5
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 Average	16.2	100					9.8 (60.5%)	59 (59%)

TABLE II
GO NO-GO ALTERNATION

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

TABLE III

RIGHT-LEFT ALTERNATION

EXPERIMENT I-a. NUMBER OF TRIALS AND TOTAL ERRORS TO CRITERION

	Preoperative learning to criterion		Preoperative savings on retention		Postoperative savings on relearning	
Animal	Trials	Errors	Trials	Errors	Trials	Errors
H 448	580	186	370	136	Failed in 1250	301
H 450	240	101	240	91	115	62
H 453	575	270	575	263	−315	−47
Average	465	186	410 (97%)	176 (95%)	–	–
C 430	540	224	540	215	540	216
C 444	595	237	385	183	595	228
C 449	305	129	305	129	305	119
Average	480	197	410 (97%)	175 (95%)	480 (100%)	188 (97%)

TABLE IV
RIGHT-LEFT ALTERNATION
EXPERIMENT 1-b: NUMBER OF TRIALS AND TOTAL ERRORS TO CRITERION

	Postoperative Learning	
Animal	trials[a]	Errors
IT 368	445	150
IT 370	505	171
Average	475	160
MFC 281	1250 F[a]	449
MFC Y 65	1095	322
Average	Failed	–
H 353	1250 F[a]	589
H 359	880	328
Average	Failed	–

[a] 1250 F indicates that the subject did not reach criterion within 1250 trials.

present more complex problems that require the completion of behavior sequences for their solution.

The results (Kimble and Pribram, 1963) showed that bilateral hippocampal lesions interfere selectively with the acquisition of behaviors that involve the execution of sequential responses. There was no indication of "short-term" memory deficits with two-choice visual discriminations over inter-trial intervals up to 6 minutes, a result similar to that of Orbach *et al.* (1960), who found no retardation in the learning of a simple visual discrimination in widely separated trials by monkeys who had been given amygdala and hippocampal lesions. (See Figs. 3, 4, and 5, and Table V.)

These data make it unlikely that some simple memory storage or memory-trace-decay function is responsible for the disruption of behavior that follows limbic system damage. This does not mean that memory functions are not affected – but memory mechanisms are complex processes just as are behavior sequences. Whatever the basic defect that follows limbic lesions, an *exclusive* relation between limbic system function and some over simply conceived memory process becomes untenable. On the other hand, so far at least, the idea holds up that damage to the limbic formations of the forebrain results in disruption of a

variety of behaviors which have in common the fact that sequential acts are critically involved.

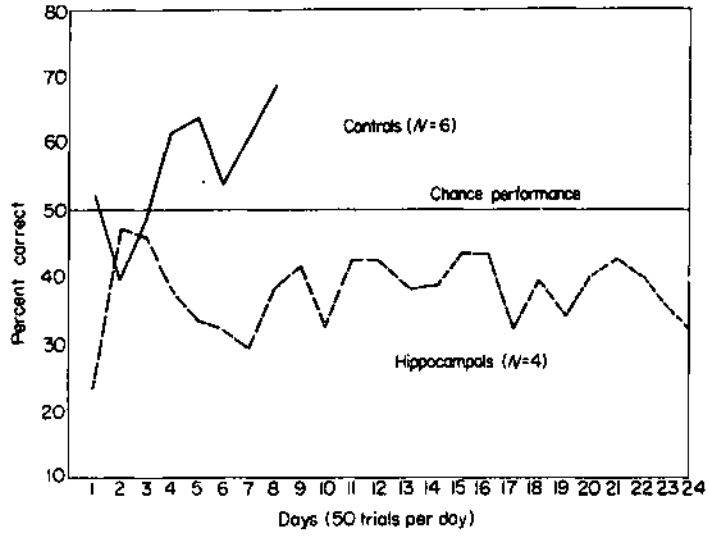


FIG. 3. Graph of performance on the "self-ordered" sequence task.

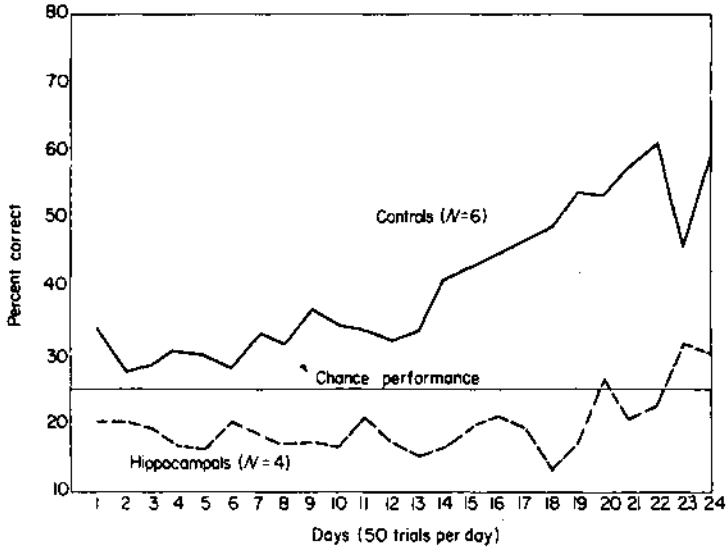


FIG. 4. Graph of performance on the "externally ordered" sequence task.

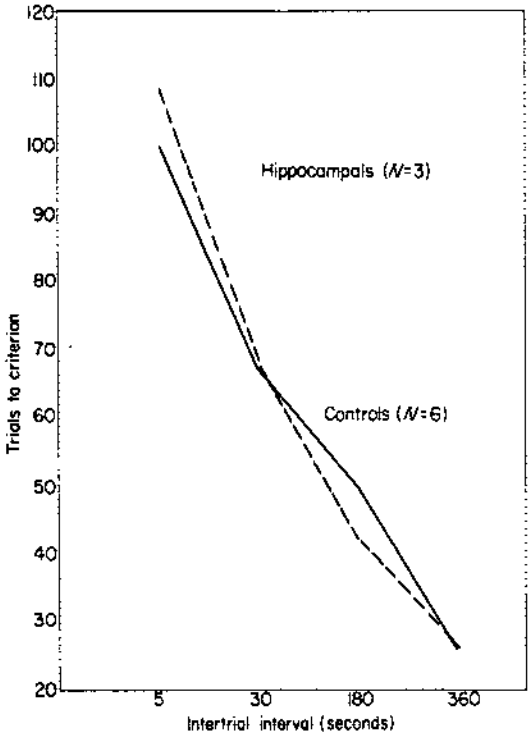


FIG. 5. Graph of discrimination performance with varying intertrial intervals. Note that spacing trials does not differentially affect the behavior of the monkeys with hippocampal resections.

TABLE V

EXPERIMENT II-a: NUMBER OF TRIALS AND TOTAL ERRORS TO CRITERION

Task	Resected subjects ($N = 4$)		Control subjects ($N = 6$)	
	Trials	Errors	Trials	Errors
"Self-ordered" sequence	932[a]	574	298	133
"Externally ordered" sequence	1897[b]	1476	1216	728
Discrimination trial spacing				
5 seconds	108	38	100	39
30 seconds	66	23	76	22
3 minutes	42	18	49	21
6 minutes	27	7	26	6

[a] Three of four did not reach criterion in 1200 trials.

[b] One of four did not reach criterion in 3000 trials.

VI. REINFORCEMENT AND THE TRANSFER OF TRAINING

The next question to be answered was "What process *is* disrupted by limbic lesions?" The process, when in successful operation, must be

responsible for the proper execution of behavior sequences. Its mechanism must take account of the known neuroanatomical and neurophysiological relations between the limbic forebrain and other structures in the central nervous system.

A clue was obtained in the last experiment: The hippocampally operated monkeys were shown to improve their performance when an extra "feedback" (in the form of turning off the "house light") was given the animals after each panel press. Feedback calls to mind those consequences of behavior that become the basis of reinforcement. There is already a body of evidence that relates limbic (as well as hypothalamic and core mesencephalic) formations to reinforcement: viz., the effects of electrical self-stimulation classically obtained from these structures (Olds, 1955); the interaction of amygdalectomy with the effect of deprivation (Schwartzbaum, 1961) and of changing the size of reward (Schwartzbaum, 1960a,b). In an initial form, therefore, a hypothesis was formulated which stated simply that limbic system lesions disrupt behavior sequences by altering the "reinforcing properties" of stimuli. In this simple form the hypothesis was *dis*confirmed: Changes *were* produced by amygdalectomy in the performance of a task – transposition – that did *not* simply involve the "reinforcing properties" of stimuli (Schwartzbaum and Pribram, 1960) (Table VI).

TABLE VI
EXPERIMENT III-a: NUMBER OF TRANSPOSED RESPONSES MADE ON TRANSPOSITION TESTS

Day	Normals					Amygdalectomized				
	439	441	443	447	Median	397	405	438	442	Median
1	6	5	6	6	–	2	5	2	4	–
2	5	5	5	6	–	3	6	2	2	–
Total	11	10	11	12	11.0	5	11	4	6	5.5

The transposition findings were consistent with the supposition that amygdalectomy impairs processes that are necessary for the generalization of a learned response. Given the training with a particular pair of stimuli, the animals with lesions did not respond normally to an overlapping set of stimuli on a brightness continuum. Indeed, their performance gave little evidence of the prior training. Whereas the normal animals markedly transposed their responses away from the previously positive stimulus, three of four amygdalectomized monkeys distributed their responses in what appeared to be a random manner.

These results make one conclusion inescapable: Amygdalectomy impairs functions that determine the discriminative as well as the reinforcing properties of stimuli. Whether or not these functions could be reduced to a common denominator in terms of generalization or transfer could be established only when the limits of the findings were further explored.

Several alternatives were opened as a consequence of these results: The first of these is that the experimental results have no general value, but are an artifact of the special conditions that obtained in this particular experiment. A second possibility is that the reinforcing properties of stimuli are a function of generalization; i.e., all stimuli are generalized by organisms, and stimuli consequent to behavior have their effect via such a process of generalization between cue and the consequence of behavior. Another possibility was also raised: that the behaviorally derived operational definitions of what constitutes reinforcement currently in vogue leave much to be desired. All three alternatives were explored. An experiment was designed to test the generality of the experimental result by investigating the effect of amygdalectomy on stimulus equivalence (Klüver, 1933). Another experiment was performed to report the effects of amygdalectomy on stimulus generalization, and a paper was prepared to detail the analysis of the problem of reinforcement (Pribram, 1964).

The results of the equivalence experiment disposed of the first of the alternatives posed by the outcome of the transposition experiment: The effect of amygdalectomy on transposition cannot be attributed to some special circumstance of the experiment—transfer of training whether in a transposition or a stimulus equivalence situation *is* affected by amygdalectomy (Table VII).

Further, the results of the generalization experiments (Hearst and Pribram, 1964; 1964b) show that the effect on transposition and equivalence can be dissociated from that produced on stimulus generalization (Figs. 6 and 7). In fact, this dissociation is double: Taken together with the report of Butter *et al.* (1965), the data show (1) that visual discrimination and generalization are both affected by inferotemporal lesions of the isocortex of the temporal lobe; (2) that these isocortical lesions have little effect on transfer in situations demanding transposition or equivalent responses to stimuli; and (3) that by contrast, amygdalectomy, while it does not disturb discrimination and generalization, markedly impairs transfer of training.

This is an interesting result in and of itself; however, the solution to the question about the nature of the disruptive effect of limbic lesions

TABLE VII

EXPERIMENT IV: NUMBER OF EQUIVALENT RESPONSES

Animal	Operation	ER's		Change	Group average change
		Preop.	Postop.		
33	None	5	5	0	–
49	None	4	8	+4	Normal
57	None	1	4	+3	+3.6
71	None	0	6	+6	–
97	None	4	9	+5	–
42	Amygdala	9	1	−8	Amygdala
55	Amygdala	6	2	−4	−6.3
76	Amygdala	8	1	−7	–
38	Inferotemporal	4	2	−2	–
43	Inferotemporal	3	1	−2	–
58	Inferotemporal	5	3	−2	Inferotemporal
51	Inferotemporal	1	2	+1	−0.8
98	Inferotemporal	4	2	−2	–
99	Inferotemporal	8	10	+2	–

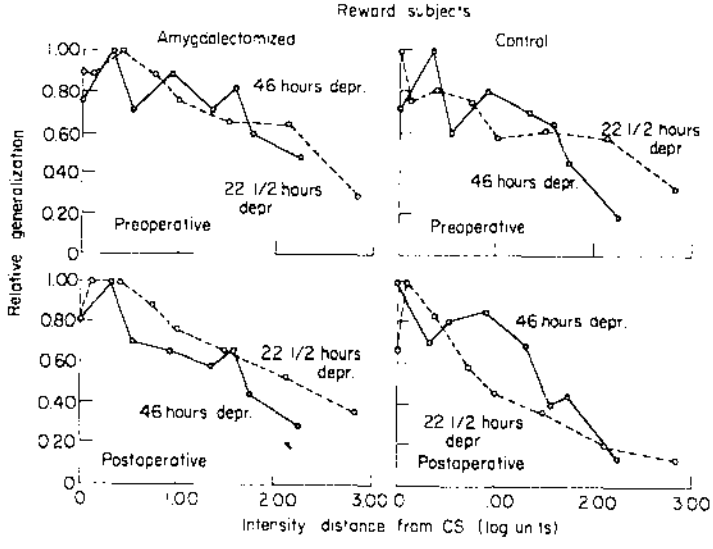


FIG. 6. Graphs of performance in generalization task in which food was used as reinforcement.

on behavior sequences, which initiated the experiments, appeared as remote as ever. What has transfer of training in common with reinforce-

ment? What is it about stimuli that gives them properties that are, perhaps in the same breath, equivalent *and* reinforcing?

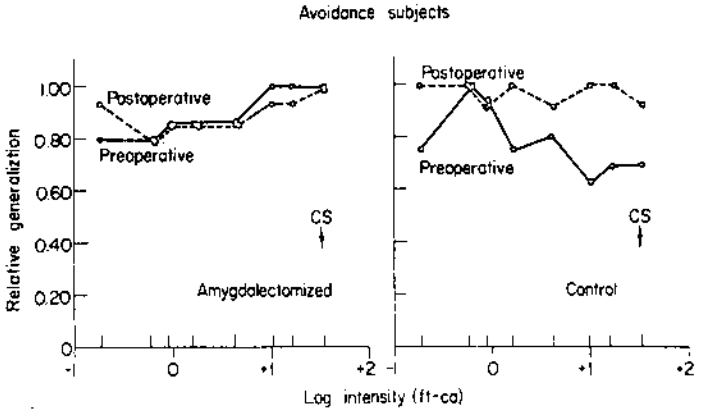


FIG. 7. Graphs of performance in a generalization task where shock was used as reinforcement. CS indicates intensity of training stimulus.

Perusal of the third alternative, that current conceptions of reinforcement are inadequate to our task, prevented complete confusion from taking over at this point in our studies. (Although the reader may already be beyond the point of no return we ask him to be patient, since we too felt, for a time, hopelessly lost among the interstices of experimental data which we had ourselves woven.)

As a result of this analysis (Pribram, 1963a), reinforcement is seen as a continuing process during which the *consequences* of behavior become ordered. A particular event is reinforcing only if it fits within the context of other events; reinforcers, to be effective guides for behavior, must be organized according to some schedule or program. Reinforcers are thus the *con-sequences* of behavior—sequences of events that fit within some prior, established context. When choices among unfamiliar alternatives are to be made (i.e., during learning), these *con-sequences* provide information about the alternatives. When, on the other hand, learning has already taken place, the *con-sequences* of actions place a value on performance (e.g., running speed will vary as a function of the number and density of distribution of reinforcing events).

The importance of this analysis for the problem of limbic system function is multiple. The reinforcing property of stimuli is shown to be dependent, first on their *sequential* occurrence; and, second, on the *temporal orderliness* (schedule or program) of these sequential occurrences.

The problem of reinforcement and that of behavior sequences thus become part of a larger question—how do sequences of events become organized into the structures that guide behavior (Miller *et al.*, 1960)?

Further, transfer of training is seen as a special case of this larger question. Reinforcement is seen as a process intrinsic to learning and to performance in a variety of contexts (Pribram, 1963a, 1964): These contexts may be the physiological needs of the organism; or they may result from the outcomes of earlier actions (i.e., from prior consequences per se) (Koepke and Pribram, 1967); in addition, the contexts may be furnished by exteroception—e.g., looking and listening are to be treated as perceptual performances. And so, stimulus familiarity and novelty—the problems of equivalence and transfer—become restatable as problems in perceptual reinforcement: the temporal ordering of the consequence of looking, listening, etc. Or to put it the other way around, classical reinforcement becomes the problem of familiarity and novelty, of equivalence and transfer, among the consequences of drives and of actions.

VII. The New Look in Homeostats

To pursue the identity of these processes that superficially seem so diverse leads to a search for identities in their organization. This search is more readily accomplished after a review of recent evidence concerning the structure of homeostatic processes. Cannon (1929, 1941) proposed the conception of a homeostat, a device which, by regulating the production of the substance to which it is itself sensitive, maintains control over the amount of that substance in the system. (The thermostat is, of course, the most familiar device of this sort.) Since Cannon, a number of specially sensitive detectors have been identified in hypothalamic and mesencephalic locations: Sensitivities to temperature (Ranson *et al.*, 1937), estrogen (Michael, 1962; Harris *et al.*, 1958), glucose (Anand, 1963), osmotic equilibrium (Andersson, 1953), and partial pressure of carbon dioxide (Meyer, 1957) are among these.

Homeostats, by definition, have the property that they control the apparatus which produces the substance (in the case of thermostats, heat) to which their receptors are specifically sensitive. Further, the control is so constituted that a sensed increase in substance shuts off production by the apparatus (a negative feedback loop).

As already pointed out elsewhere (Pribram, 1960) each homeostat must, in addition, be equipped with a device by which its set point can be adjusted. On the thermostats that regulate the temperature of our

houses, a small dial engraved with numbers (representing temperature) serves this function. By means of the dial, changes can be effected in the distance between the temperature-sensitive thermocouple contacts. These changes then bias the further operation of the homeostat—the system becomes tuned to the new setting.

In the mammalian thermostat a similar function is described:

> An interesting further suggestion from these experiments is that the brain-stem activating system, which is mainly controlled by nonthermoceptive projections, may influence the activities of the thermoregulatory effector systems, including the skeletal muscles as an independent reference mechanism (von Euler and Soderberg, 1958). If such were the case, a certain change in the intensity of function of the activating system might balance the coordination of the different heat-loss and heat-production mechanisms at a new level of body (or brain) temperature. This might, e.g., explain why hyperphagia and a steady hyperthermia may appear together after hypothalamic lesions (Mayer and Greenberg, 1953).
>
> From a conceptual point of view this suggestion is a definite advance. The central nervous thermoregulatory mechanism has sometimes been compared to a thermostat; in fever and during hard muscular work (Nielsen, 1938) when thermoregulatory balance is achieved (Bazett, 1951) at a higher level of body temperature than is normally the case at rest, the 'thermostat' is said to be reset. A given intensity of muscular work leads in man to an ultimate new level of body temperature (Nielsen, 1938) which is relatively independent of the surrounding conditions for heat loss, an observation demonstrating that the body regulates by adjusting its heat-loss mechanisms at the new level. If under such conditions the activities of the different thermoregulatory effector systems can be stated quantitatively and the central reference mechanism, the brain-stem activating system, responsible for the new thermoregulatory balance can be defined, the terms thermostat and resetting would certainly have an explicit meaning (Strom, 1960).

The Handbook of Physiology, in its volumes on neurophysiology (Magoun, 1960) contains many references consonant with the view that the activity recordable from the reticular formation reflects the set point around which the several homeostats operate (e.g., respiration, p. 1113; galvanic skin response, p. 961). The varied influences that play on the reticular formation and thus establish a bias have been emphasized repeatedly (Magoun, 1958). Here, the particular relation of the limbic forebrain to the operation of setting the bias of homeostats is of special concern.

However, it is not only the regulation of the organism's *millieu interieur* (Bernard, 1958) that is organized in this fashion. Recent evidence has made it necessary to conceive in the same terms of an organism's control over his field of action and his perceptions. Essentially, action takes place within a program or plan, a hierarchically organized complex of homeostatlike structures called "test-operate-test-exit" sequences

(Miller *et al.*, 1960). Basic to their organization is efferent control over inputs, as for instance by γ efferent activity on muscle spindle receptors (Pribram, 1960). Perception also proceeds by way of efferent control over receptors: a gating mechanism that controls its own input—the mechanism of perceptual readiness (Pribram, 1960). The concept homeostat, was, of course, invented to describe just this sort of structure: a system that controls the process to which it is sensitive. As control over the production of heat is such a mechanism, the concept was quickly applied in the engineering sciences to include control systems concerned with other varieties of energy: those used in communication processes. The techniques of cybernetics and information (amount of order) measurement are in turn useful to those working with the organization of neurobehavioral processes. The suggestion made is that, just as the mechanical thermostat became the model out of which the science of communication and control developed, the biological homeostat can become the nuclear conception from which neurobehavioral science derives impetus.

Let us return now, a long way back, to the thesis and hypotheses that motivated this research and theoretical analysis. The thesis is that the limbic systems partake in the homeostatic regulations of the organism. As was pointed out, this thesis, so stated, differs little from the idea that the limbic systems function as a "visceral brain." However, if the reach of what is meant by homeostatic regulations encompasses not only the neural regulation of the organism's internal environment, but the neural control (through the ordering process of reinforcement) of all sequentially occurring events, the thesis takes on new dimensions. Had this larger view of the homeostatic or reinforcing process been stated as an hypothesis, it would certainly have derived support from the experimental results described. Actually, the experimental and analytical procedures were so intermeshed that only now does the whole picture become at all clear.

This takes us to the second major concern voiced earlier: How do the limbic systems "partake" of the reinforcing process? Only when the details of "partake" are spelled out can the power of the above analysis be put to test. Only then can the necessary identities between the process of reinforcement and that of transfer really be teased out.

As a first step the following experiment was undertaken. All that has gone before points to the fact that limbic system lesions disrupt the way an organism handles sequences of stimulus events, whether these are derived from the receptors that surround the midline ventricular system or from exteroceptors. Could this disruption also take place at the very

simplest level, the mechanism by which events are admitted to context?

This simple mechanism is, of course, habituation of the orienting reaction. Experiments by Humphrey (1933) and more recently by Sokolov (1960) leave little doubt that habituation reflects the organization of a neural process (Sokolov's term: "the building of a neural model of the environment") against which current inputs are then matched. Sokolov's experimental demonstration was as follows: Human subjects were exposed to a tone of a certain intensity, frequency, and duration, repeated at irregular intervals. Galvanic skin response (GSR), heart rate, finger and forehead plethysmograms, and electroencephalograms were recorded. Initially, these records showed the perturbations that are classified together as the orienting response. After several repetitions of the tone, these perturbations diminish and finally vanish. This is habituation, a process which had been thought to reflect a *lowered* sensitivity of the central nervous system to inputs. Sokolov then *decreased* the intensity of the tone, leaving the other parameters unchanged. Immediately the subjects again showed full-blown orienting responses. Sokolov reasoned that the central nervous system cannot be *less* sensitive—it is only less sensitive to *sameness,* to *differences* the central nervous system has become *more* sensitive. He tested this idea by rehabituating his subjects, then shortening the tone without changing any other parameter. As predicted, his subjects then oriented to the unexpected silence.

The orienting reaction and habituation are thus sensitive measures of an organism's expectancy process—the process by which context is organized. We therefore initiated a series of experiments to analyze in detail the neural mechanisms involved in orientation and habituation. This proved not as easy as it at first seemed. The dependent variables—behavior, GSR, plethysmogram, and electroencephalogram—are not as dependable as one would like them to be. As others have found, dissociation between them is readily observed when one makes one or another experimental variation. In our experimentation, forehead plethysmography turned out to be especially tricky. We finally settled on behavior, the GSR, and the electrical brain manifestations (which we are at present pursuing) as most reliable. (We, of course, realize full well that the GSR can be used as an indicator of processes other than "expectations." Nonetheless, in the experiments reported here, the likelihood is that the GSR is truly one measure of the orienting reaction.)

The results of the first of these experiments (Schwartzbaum *et al.*, 1961) indicate that, under certain conditions, removal of the amygdaloid complex can selectively affect the persistence of locomotor activity in monkeys by minimizing or retarding decrements that normally occur.

The lesion thus produces a disturbance in the habituation of motor activity (Fig. 8).

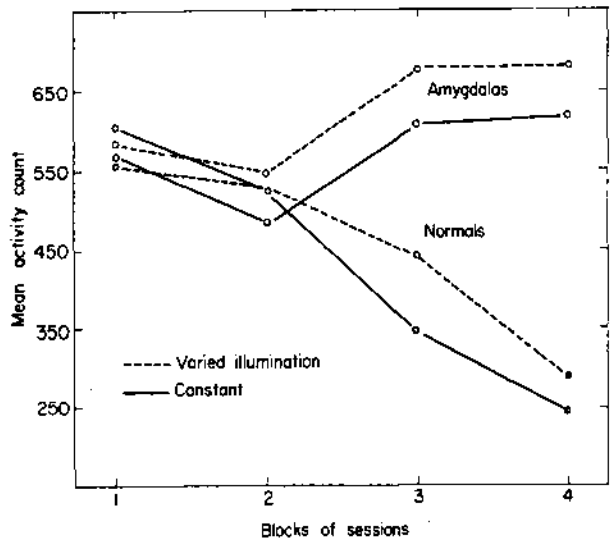
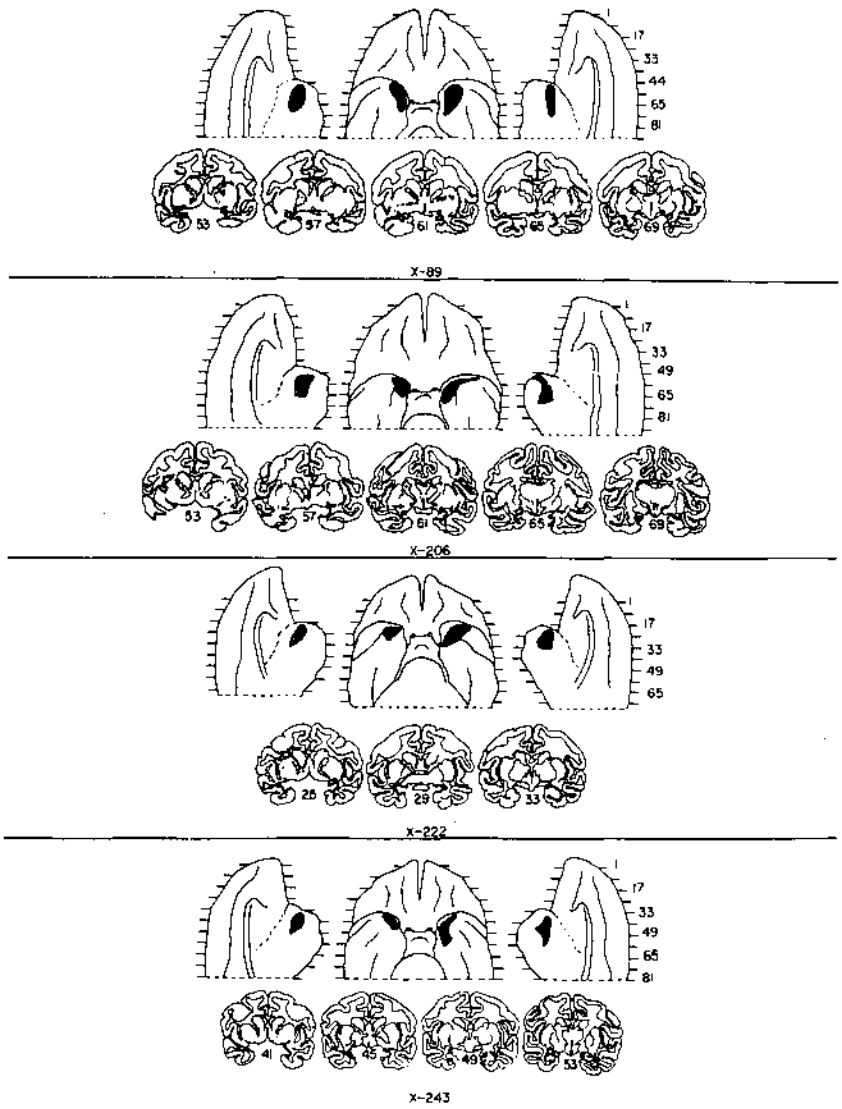


FIG. 8. Graph of progressive changes in activity. Note that normal *Ss* habituate to their environment and amygdalectomized *Ss* do not.

The results of the experiments on the habituation of the GSR component of the orienting reaction (Bagshaw *et al.*, 1965; Bagshaw and Benzies, 1968) also indicate clearly that amygdalectomy has an effect. (For reconstruction of these lesions, see Figs. 9A and 9B.)

The lesion interferes with the GSR in a situation in which the GSR is ordinarily obtained as part of the orienting reaction, although the GSR per se is not abolished (Fig. 10). From the previous experiment and others (Schwartzbaum, 1960a,b), we know that the habituation of behavior is severely altered by the lesions. Putting these results together, one could conclude that the GSR component of orienting is in some way crucial to subsequent behavioral habituation. If we could still hold the simple visceral-autonomic hypothesis of the role of the limbic systems, these results would certainly support it. However, as things now stand, the situation is more complicated. The only view possible at this time is that visceral-autonomic changes, as indicated by the GSR, function *integrally* in the production of habituation, i.e., in the organization of expectancies. How?



FIGS. 9A and 9B. Reconstructions of the extent of lesions, and sample cross sections of depth, of amygdalectomized brains. Solid areas indicate lesions. Striped areas in medial surface diagrams and in some cross sections indicate spared amygdala.

VIII. The Model

The problem is posed by Sherrington in the following quotation:

We note an orderly sequence of actions in the movement of animals, even in cases where every observer admits that the co-ordination is merely reflex. We see one act succeed

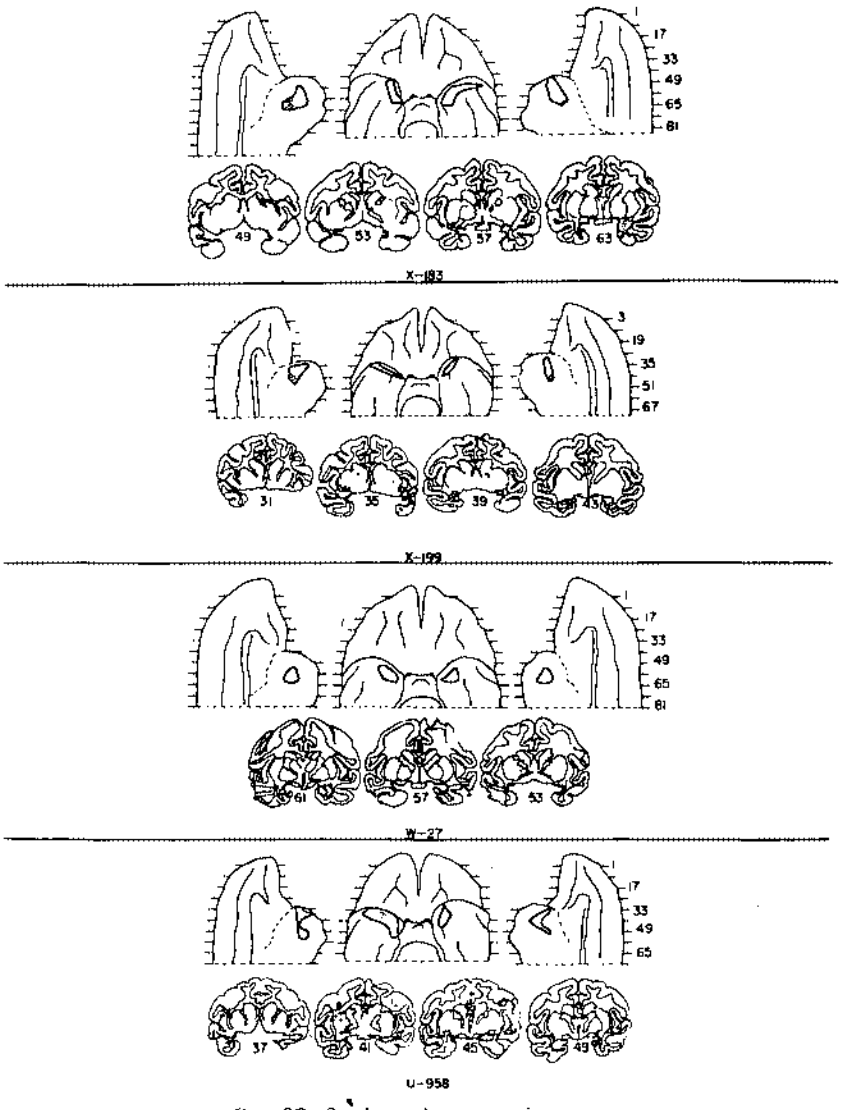


FIG. 9B. See legend on opposite page.

another without confusion. Yet, tracing this sequence to its external causes, we recognize that the usual thing in nature is not for one exciting stimulus to begin immediately after another ceases, but for an array of environmental agents acting concurrently on the animal at any moment to exhibit correlative change in regard to it, so that one or another group of them becomes—generally by increase in intensity—temporarily prepotent. Thus here dominates now this group, now that group in turn. It may happen that one stimulus ceases coincidently as another begins, but as a rule one stimulus overlaps another in regard

to time. *Thus each reflex breaks in upon a condition of relative equilibrium, which latter is itself reflex.* In the simultaneous correlation of reflexes some reflexes combine harmoniously, being reactions that mutually reinforce (Sherrington, 1947, p. 120; italics added).

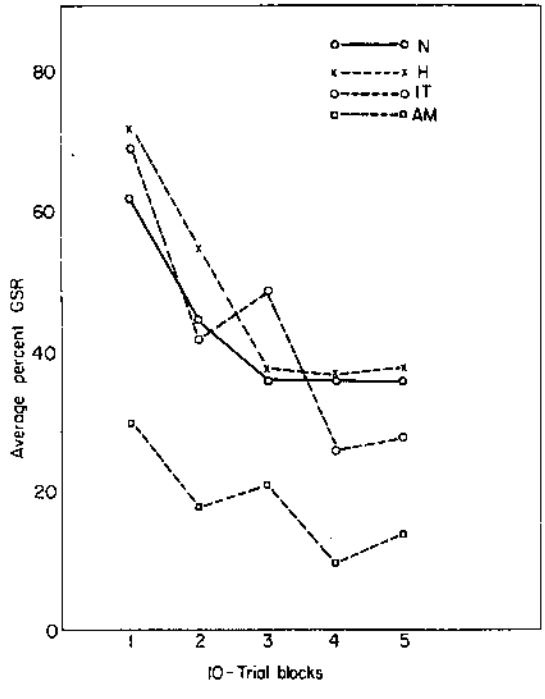


FIG. 10. Percent GSR responses to a pure tone stimulus across 50 trials for a normal group of monkeys, compared with three groups who had selective lesions in the temporal lobe.

The suggestion arises that this "condition of relative equilibrium" is a condition necessary both for the operation of "reactions that mutually reinforce" and for one or another of ". . . an array of environmental agents acting concurrently on the animal at any moment to exhibit correlative change to it" and so become temporarily prepotent. A condition of equilibrium is thus already conceived by Sherrington to be fundamental both to reinforcement and to transfer. It is as if the organism's transactions with the environment resembled an iceberg: "the condition of relative equilibrium, which . . . is itself reflex" acting as a stabilizing mass which allows the perturbations caused by the "array of environmental agents" acting on its exposed portion to be organized and thus dealt with promptly and without upsetting the entire structure.

The further suggestion to be entertained here is that this condition

of equilibrium, this stabilizing reflex mass, is in large part, though not wholly, produced in an organism through habituation to any repetitiously recurrent events and that visceral-autonomic processes form a major category of such events. These repetitious processes are thus to be distinguished from the unexpected, i.e., the novel occurrences that the organism experiences. The model can now be spelled out more fully.

Lacey and Lacey (1958) have presented evidence that supports the suggestion that the GSR and other visceral-autonomic indicators can be used to gauge an organism's stability or lability in a variety of situations. Autonomic "stabiles" react sluggishly to their environment; "labiles" react rapidly, but make more errors. However, labiles maintain maximal readiness to respond longer than do stabiles.

Ashby (1960) has portrayed the structure of the stabile and labile system: A completely joined set of homeostats is ultrastable. It reacts sluggishly to perturbations, and when these are iterated, adaptation time is interminable. The ultrastable system can, however, be disjoined if the reaction to the perturbation is isolated from the rest of the system so that homeostasis of this part alone can be achieved. Each part-stability thus achieved further disjoins the system. "Constancies cut the system to pieces" (Ashby, 1960). Thus adaptation is relatively rapidly achieved if it can occur serially, sequentially.

The question is raised, therefore, how the galvanic skin "orienting" response functions as an indicator of the amount of join of the homeostatic system? Could it be that the autonomic components of the orienting reaction (which signals discrepancy between an expectation and the concurrent input) can give some index of whether the perturbation resulting from the discrepancy is isolated or not? In order to make clear how this might be so, a more complete description of the model of homeostats and how they are joined, i.e., synchronized, must be detailed.[1]

Each interaction between environment and organism involves at least two components: (1) discrete interaction by way of the brain's sensory-mode-specific classical projection systems and its core homeostats; (2) a "nonspecific," relatively diffuse, interaction by way of reticular and related formations. As discussed earlier, the activity generated in the nonspecific systems acts as a bias on the specific reactions—i.e., the set point or value toward which a specific interaction tends to stabilize is set by the nonspecific activity. Visceral feedback, which is by the nature of its

[1] This description owes much to the one proposed by Ashby (1960). However, his design is somewhat modified and the specifics of the neural function are made explicit (see Pribram, 1963b, 1967).

receptor anatomy and afferent organization particularly diffuse, certainly constitutes a major input to this biasing mechanism: an input which can do much to determine its set point. However, in addition to providing a major source of input, visceral and autonomic events have the characteristic that they are, in the history of the organism, repetitiously redundant. They vary recurrently, not from occasion to occasion as do the external changes. The visceral-autonomic feedback is thus a major determinant of bias and contributes through the process of habituation, a large mass of the "expectancy" structure of the organism.[2] Such a stable substructure is necessary if more specific expectancies are to develop. Expectancies can become independent iterations, sequences, only to the extent that they are stabilized by the operation of some structure that does not change value with each perturbation. Only with the aid of the constancy of this basic substructure can specific experiences be disjoined.

This massive basic part of the expectancy structure has its own organization and when constancies are developed within it, regulation of visceral-autonomic activities proceeds with alacrity. However, should these basic ("essential") constancies be disrupted, as for instance by amygdalectomy, two consequences would follow: (1) Visceral regulation would become sluggish and capricious; (2) the total system would become hyperstable with the result that the organism's discrete interactions with the environment through the operation of the classical sensory motor system would, if anything, become more disjoined, desynchronized, immediate.

Evidence supports these inferred consequences. A series of studies (Brennan, 1955) has shown that bizarre sluggishness of visceral reactions follows limbic lesions: e.g., gastric motility is slowed by half; intravenously administered sugar will produce a maintained abnormally high blood glucose level for as long as a week, etc. The GSR has also been shown to display this sluggishness (Grueninger *et al.*, 1965). With regard to the immediacy of experience, the hypermetamorphosis observed by Klüver and Bucy, the repetitious taking and mouthing of a proferred lighted match as well as the delayed behavioral habituation are indicative that this is indeed what occurs.

The suggestion is therefore that amygdalectomy produces hyperstability by disrupting the temporal organization which has developed

[2]This massive "apperceptive" base of the expectancy structure corresponds, in function, to the set of step mechanisms in Ashby's model. As he points out (pp. 198–199), if join between iterated perturbances is by way of the step mechanisms, and *not* by way of the reacting parts of the organism, stability can be achieved relatively rapidly.

by habituation to regularly recurring events such as those derived from visceral activity. Organization of this neural system depends, as elsewhere in the nervous system, on the development of past constancies which keep the perturbations involved in any specific action from becoming general both spatially and temporally. Failure of amygdalectomized monkeys to display an orienting GSR is, in the light of this model, attributed to the fact that the organization of the GSR-controlling system has been disrupted and therefore is too sluggish to react while the neural responses mediated through the classical systems are, if anything, too discrete to be registered.

Should this model prove viable, certain consequences for a neural mechanism of memory follow. Weiskrantz (1967) has shown that an extensive temporal lobe lesion (which combines hippocampal ablation with that of the isocortex involved in discrimination) leads to a memory impairment in monkeys comparable to that found in man after extensive medial temporal resection (Milner, 1958). Thus limbic-isocortical relations appear to be involved in producing the total syndrome. The model proposed here emphasizes the role of the limbic mechanism in allowing disjoin, desynchronization, to occur among memory units, i.e., among expectancies, the neuronal models of experienced inputs. If disjoin among memories is indeed important to the processes of memory storage and retrieval, the suggestion arises that such processes must occur in a system arranged in parallel. Such a system would allow simultaneous access to a great number of units provided they were disjoined and thus obviate the necessity for a time-consuming sequential scan.[3] Blurring of the boundaries of memory units would drastically impair such a simultaneous access mechanism: Both the addressing procedure and the limited search demanded by the model depend on unit boundaries since an address depends on the coordinates given by such boundaries and search is completed only when the boundaries of a unit have been reached.

In summary, then, the organism's expectancy structure is conceived to resemble an iceberg whose submerged portion stabilizes the perturbations to which it is subject through its exposed surface. This submerged portion, a massive "apperceptive" base, results from habituation to repetitiously recurrent experiences and makes possible discrete, prompt reactions to novelty. Limbic system lesions are conceived to disrupt the organization of this massive base and this disruption is assumed to have several consequences: the regulation of recurrent regularities such as

[3] A description of a somewhat similar mechanism for the operation of a computer content addressable memory has already been proposed (Lee and Paull, 1963).

visceral processes becomes sluggish and capricious. Discrete interactions with the environment via the classical sensory-motor projection systems becomes more immediate, less deliberate, more stimulus bound. Finally, to the extent that additional disruption of discrete neural organization is produced, join among memory units occurs with severe disturbance of memory processing.

The thesis that limbic formations partake of the neural organization of the homeostatic regulations of the organism has thus taken on precision. Actions and perceptions as well as drives are seen organized homeostatically. Homeostats have been provided with an active, movable bias that allows shifts in set point; the homeostat thus becomes more properly a "homeorhetic mechanism" [to use Waddington's (1957) term] i.e., a mechanism that deals as much with coming stabilities as with return to prior ones. Yet, the original meaning of homeostasis takes on new importance as well. Visceral-autonomic events, being for the most part relatively diffusely organized and repetitiously redundant, provide through the mechanism of habituation a stable substructure upon which reactions to the unexpected can be built. This fresh view of the import of visceral-autonomic activity accounts for the proven heuristic value which the concept "visceral brain" held for investigators of the limbic forebrain. At the same time, the peculiar disturbance of the memory process of man and monkey can be understood. If each major environmental change alters the contexts (the loci of disjoin, the set points, the set of values) within which stimuli are organized, very little can be transferred from one situation to another. Behavior sequences become disrupted, since they are dependent on hierarchically organized programs, plans, which in turn are constructed from such transfers.

Many new questions and new versions of old ones immediately arise, of course. Just how do limbic processes protect the organization of the organism's constancies? What role does each of the limbic structures have in the maintenance of redundancy? What is the precise relation between "expectancy" as defined by habituation of the orienting reaction and "error" when incongruity results from an unexpected consequence of an action? Neurobehavioral experiment and analysis is not yet done.[4]

[4]Especially cogent to the argument presented here are the following recent publications: Bagshaw and Benzies (1968), Bagshaw and Coppock (1968), Bagshaw and Pribram (1968), Barrett (1969a,b), Douglas (1967), Douglas and Pribram (1966), Gerbrandt (1965), Pribram (1967, 1967a,b), Pribram *et al.* (1966), Pribram and Melges (1969), and Pribram and Tubbs (1967). These experimental studies and formulations (as well as many of those cited in the text) have been undertaken and completed since this material was presented

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