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# The Limbic Systems, Efferent Control of Neural Inhibition and Behavior

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The functions of the limbic forebrain have been characterized either in terms of their influence on response regulation (McCleary, 1961; Miller *et al.*, 1960), on emotion (MacLean, 1950; Pribram and Kruger, 1954), or on memory (Milner, 1958; Penfield and Milner, 1958). In and of themselves these characterizations have so far failed to provide the key to the essential nature of the limbic contribution to behavior and to psychological experience.

In part, this failure can be attributed to an absence of precision in the concepts invoked. With this in mind the question has been raised whether a clearer picture might be obtained if a possible relationship between limbic function and information processing were pursued. Perhaps with this relationship worked out, the puzzle of the importance of the limbic systems to response regulation, to emotion and to memory will also come into focus.

To this end a series of neurophysiological and neurobehavioral experiments were undertaken. The results of the neurophysiological experiments were such that they suggested a model of limbic system function. I shall first introduce the model, then present the neurophysiological and behavioral data which generated it. Finally, I will attempt to summarize the model in its current form.

## SOME NEUROPHYSIOLOGICAL DATA AND A MODEL

The model regards inhibitory neural processes — inhibition defined as a reduction in the excitation of a neural unit. Two major types of neural inhibition are recognized. The first is inherent in afferent activity: active afferent neurons inhibit their neighbors. This lateral or surround inhibition operates through collateral processes distributed among neurons or via amacrine-like cells and is well-demonstrated in the visual (Hartline *et al.*, 1956), auditory (Von Bekesy, 1957), and somatic (Mountcastle, 1957) systems, both at peripheral and central stations. This type of afferent neural interaction corresponds to Pavlov's 'external' inhibition. The second type of afferent inhibition is recurrent (Asanuma and Brooks, 1965; Brooks and Asanuma, 1965a, b). It takes two forms, presynaptic and postsynaptic. Both result in the regulation of afferent activity via negative feedback. In the case of postsynaptic recurrent inhibition,

interneurons of the Renshaw type are assumed via recurrent inhibitory fibers, to function as dampers which control the excitability of active neurons as a consequence of their own activity.

The model focuses on the interaction of these two forms of afferent inhibition. The *collateral* type acts to *accentuate* the difference between active and less active sites while the *recurrent* type tends to *equalize* such differences. Any patterned change in the system will be enhanced by collateral inhibition; recurrent inhibition works against change, tending to stabilize the status quo. The collateral type is thus conceived to be a labile mechanism sensitive to input and concurrent activity. The recurrent type, on the other hand, works more slowly, countering the rapid fluctuations in the patterns of neural activity that would otherwise occur and stabilizing the changes once they have occurred.

The chief concern of the model is with efferent control exerted over this interaction. This control is primarily cerebrofugal. Mechanisms which enhance and inhibit afferent inhibition are assumed to converge upon the afferent pathways. Because of this site of operation, a 4-fold mechanism of efferent or cerebrofugal control should in theory be distinguishable: (a) enhancement of collateral inhibition; (b) enhancement of recurrent inhibition; (c) inhibition of collateral inhibition; and (d) inhibition of recurrent inhibition.

There is already available evidence for corticofugal control over both the presynaptic and postsynaptic forms of recurrent inhibition. Repetitive stimulation of a variety

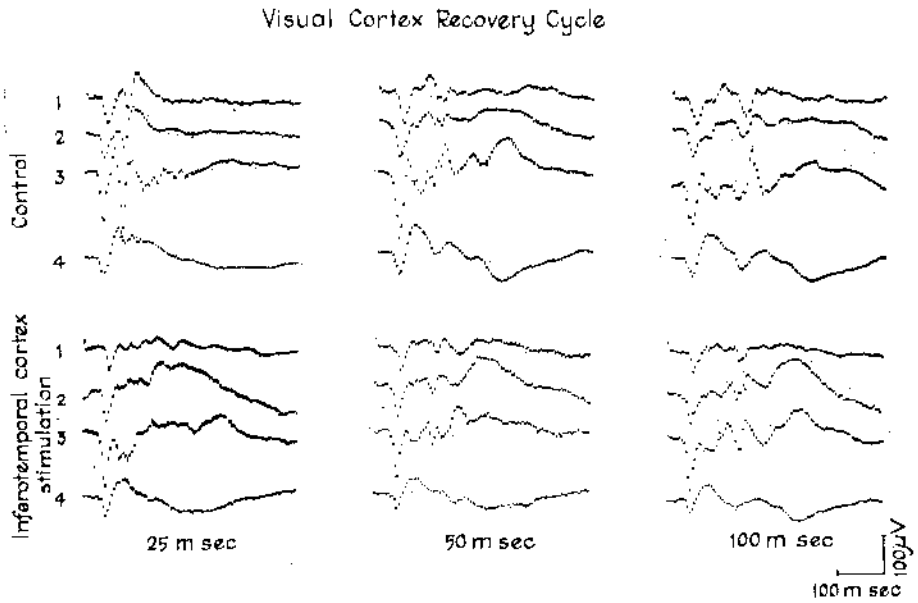


Fig. 1. A representative record of the change produced in visual evoked responses by chronic stimulation of the inferotemporal cortex. Upper set of records was taken before stimulation; lower set, during stimulation. All traces were recorded from the visual cortex; in the first column are responses produced by a pair of flashes separated by 25 msec; flash separation is 50 msec in the second column and 100 msec in the third.

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of sensory-motor points on the lateral cortex influences presynaptic inhibition at the spinal level (Andersen *et al.*, 1962; Andersen and Eccles, 1962; Eccles, 1962). And the effect of hippocampal stimulation on visual evoked activity has also been recorded (Fox, 1966).

The evidence for efferent control of collateral inhibition has been gathered in my own laboratory, in collaboration with Dr. D. N. Spinelli (Spinelli and Pribram, 1966). I will present these studies in detail and then continue to develop the model which is so extensively rooted in these data.

Experiments were performed on fully awake monkeys implanted with small bipolar electrodes and a device which allows chronic repetitive stimulation of one of the electrode sites.

The monkeys were presented with pairs of flashes and the interflash interval was varied from 25 to 200 msec. Electrical responses were recorded from the striate cortex and the amplitude of the responses was measured. A comparison of the amplitude of the second to the first response of each pair was expressed and plotted as a function. The assumption underlying the interpretation of this function is that when the amplitude of the second of the pair of responses approximates that of the first, the responding cells have fully recovered their excitability. In populations of cells such as those from which these records are made, the percent diminution of amplitude of the second response is used as an index of recovery of the total population of cells — thus the smaller the percent, the fewer the number of recovered cells in the system.

Chronic stimulation (8–10/sec) of several cerebral sites alters this recovery function. When the inferotemporal cortex of monkeys is stimulated, recovery is delayed (Figs. 1,

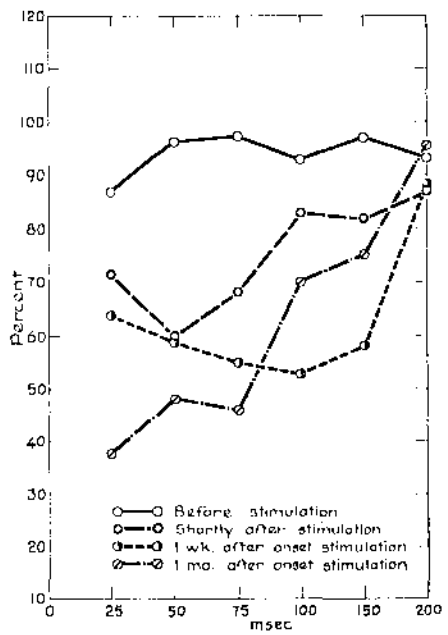


Fig. 2. A plot of the recovery functions obtained in one monkey before and during chronic stimulation of the inferotemporal (I.T.) cortex.

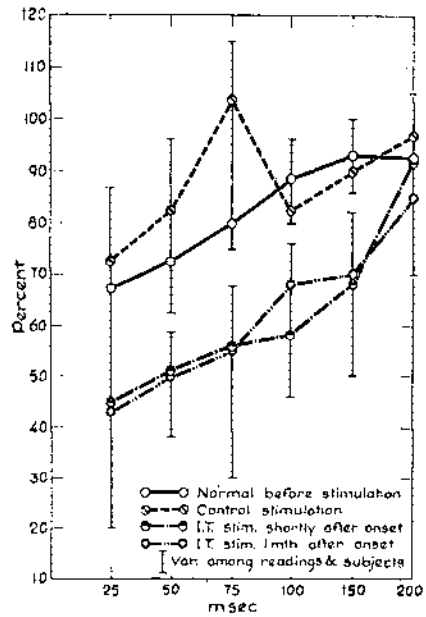


Fig. 3. A plot of the recovery functions obtained in 5 monkeys before and during chronic cortical stimulation.

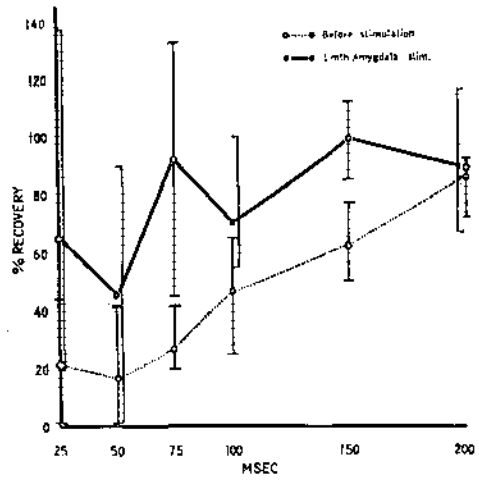


Fig. 4. The effect of chronic stimulation of the amygdaloid complex on recovery function. The dotted line indicates the function before, the solid line after, 1 month of stimulation. Bars perpendicular to the curves show variability among subjects. Each curve is based on the average response of 4 subjects.

2, 3). Stimulation from control sites (precentral and parietal) has no such effect. Nor does the stimulation of inferotemporal cortex alter auditory recovery functions. These, however, can be changed by manipulations of the insular-temporal cortex, as was shown in a parallel experiment performed on cats. Here the crucial cortex was removed and recovery functions obtained on responses recorded from the cochlear

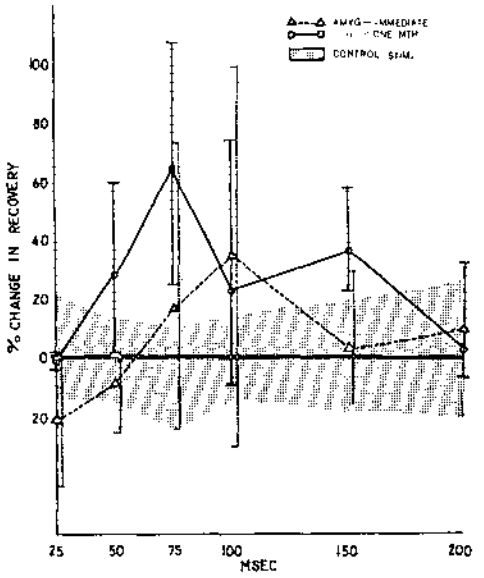


Fig. 5. This figure represents the same data as those represented in Fig. 4. However, here % change in recovery is plotted. Shaded area indicates range of recovery for *un*stimulated subjects.

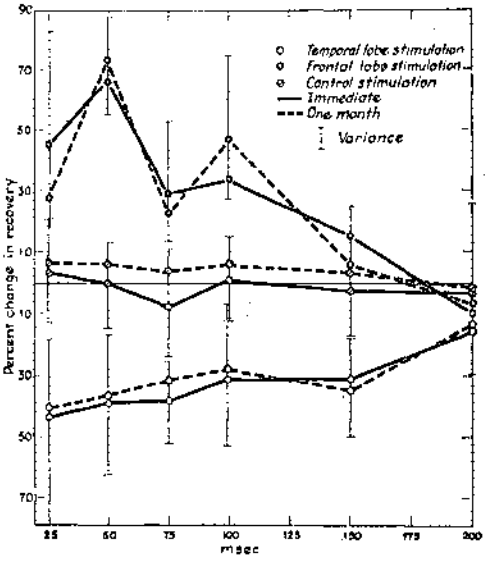


Fig. 6. This figure plots the % change in recovery for all subjects in the various experiments. It is thus a summary statement of the findings.

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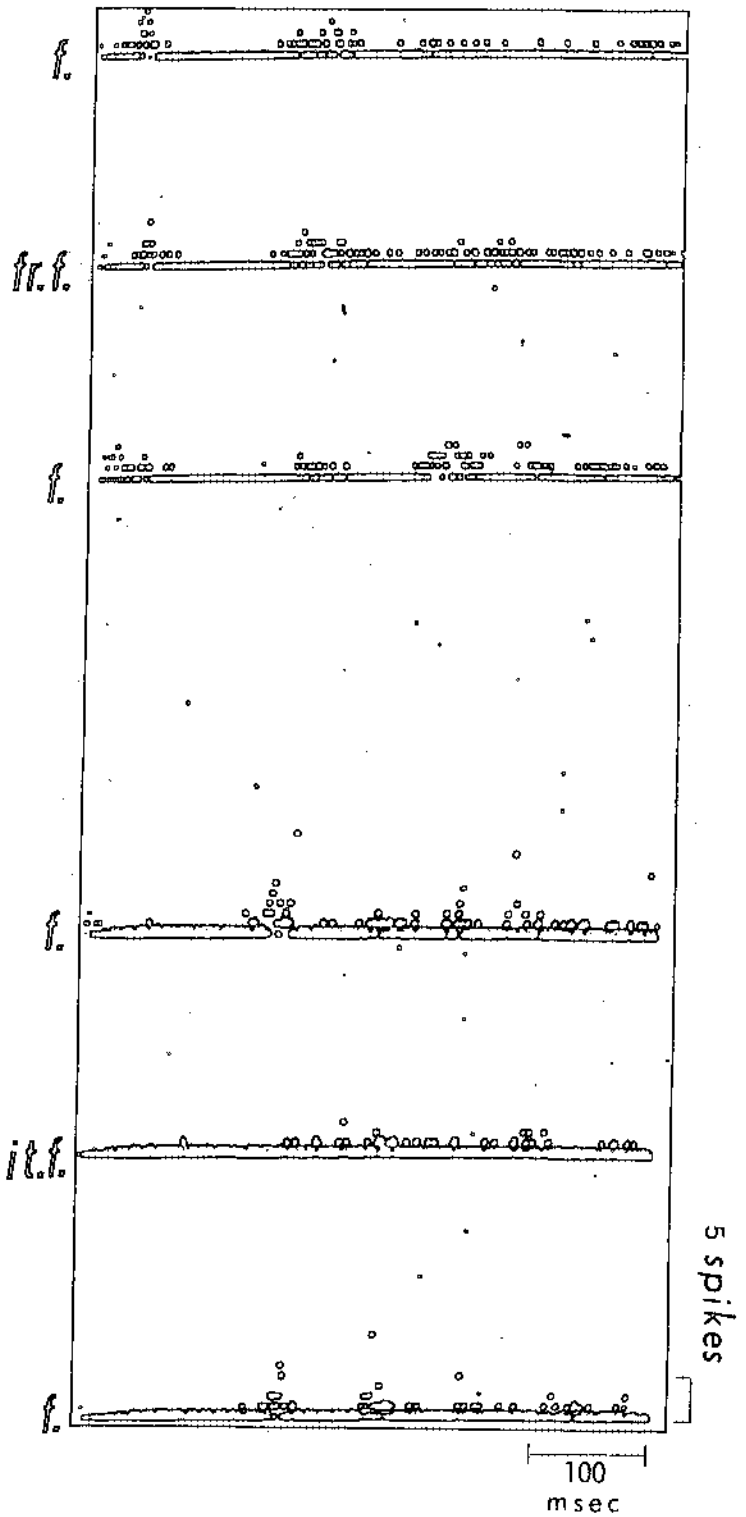
Fig. 7. This figure is made up of photographs of a pulse histogram derived from a readout from a computer for average transients. Each vertical segment represents the number of impulses recorded from a neural unit during a 1.24 msec period. The upper three traces show the effects of concurrent stimulation of the frontal (fr.), the bottom three traces the effects of concurrent stimulation of the temporal (it)., cortex of cats on the unit activity evoked in the striate cortex to repeated flashes (f.). The first and last trace in each trial are controls; the middle traces were recorded during concurrent stimulation. Note that the first silent period is lengthened by concurrent temporal, and shortened by concurrent frontal, cortex stimulation.

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nucleus (Dewson III *et al.*, 1966). Removal of insular-temporal cortex shortens recovery in the auditory system.

A great many neurobehavioral experiments have shown the importance of these isocortical temporal lobe areas (and not others) to visual and to auditory discrimination. These studies are reviewed elsewhere (Pribram, 1954, 1966). What concerns us here is that a corticofugal, efferent mechanism is demonstrated and that this mechanism alters the rapidity with which cells in the visual and auditory afferent systems recover their excitability. Further, since stimulation delays and ablation speeds up recovery, the inference is that the normally afferent inhibitory processes which delay recovery are enhanced by the ordinary operation of these temporal lobe isocortical areas.

But the opposite effect — namely inhibition of afferent inhibition — can also be obtained when cerebral tissue is chronically stimulated. In these experiments the cortex of the frontal lobe and the cortical nucleus of the amygdala were chronically stimulated and recovery of cells in the visual system were shown to be speeded. This result has suggested that the frontal and anterior medio-basal portions of the forebrain function as efferent systems which inhibit afferent inhibitory processes (Figs. 4, 5, 6).

The antagonistic effect of these two efferent control systems is best illustrated by data obtained at the unit level.

These unit recordings were made from the striate cortex of flaxidilized cats to whom flashes of light were presented. Note that the silent period of a cell can be lengthened by concurrent inferotemporal stimulation. Note also that concurrent frontal stimulation can shorten this silent period. Finally, note the unit whose silent period is lengthened by inferotemporal, and shortened by frontal, stimulation (Figs. 7,8).

In summary, the model is based on neurophysiological evidence of two forms of afferent inhibition: collateral and recurrent. The reciprocal interaction of these two forms is spelled out. Data are presented which indicate that afferent inhibition is under efferent corticofugal control. Further, such efferent control is shown to be balanced: both efferent enhancement and efferent inhibition of afferent inhibition were found to converge so as to regulate the activity of a single system and even a single cell. The major assumption of the model is that separate forebrain systems can be found to regulate collateral and recurrent afferent neural inhibition.

One of the consequences of this model of efferent control over afferent inhibition is a plausible neural explanation of the orienting reaction and its habituation. A series of studies has shown (1) that orienting can be identified by a specific pattern of behavioral and physiological indices; and (2) that habituation of this set of indices is not a function of a raised neural threshold to input, but to change in some neural configuration against which input is matched (Sokolov, 1960). The reasonable suggestion can be made that habituation reflects increments in recurrent inhibition and that the orienting reaction manifests an override on habituation which takes place whenever collateral inhibition is enhanced. There is at least preliminary evidence at the neurophysiological level which is congruent with this suggestion (Thompson, 1966). The following data at the neurobehavioral level can also be interpreted to be in accord with the model.

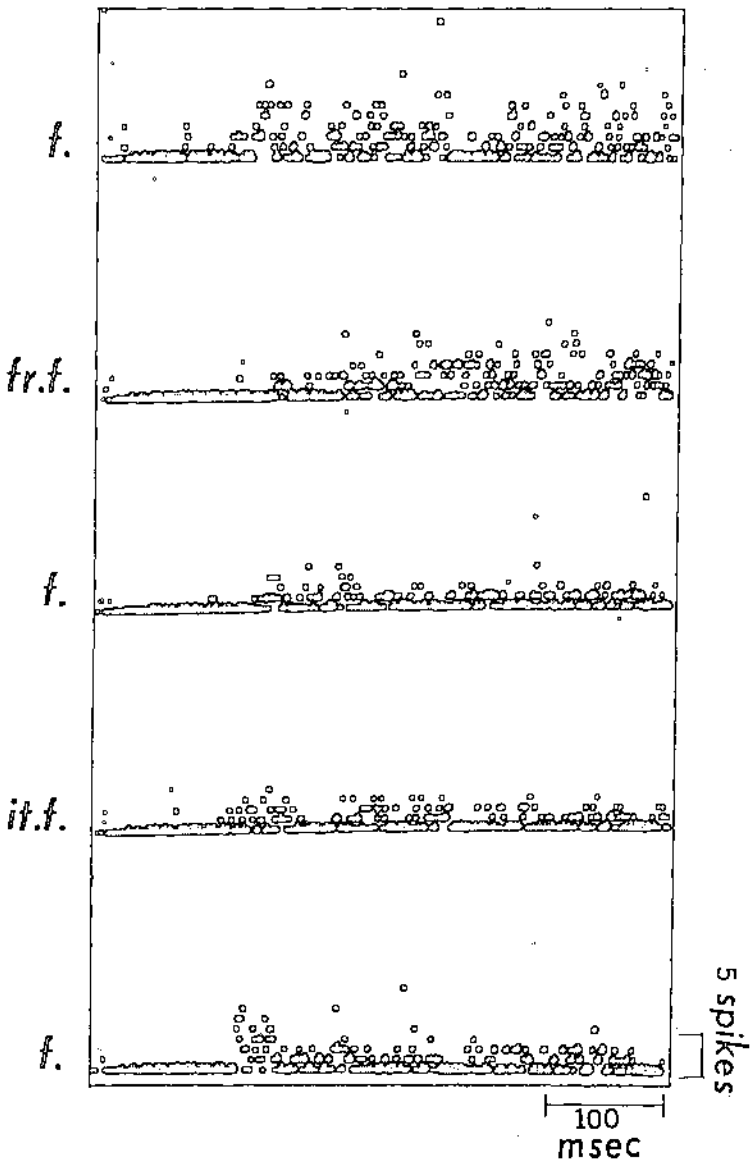
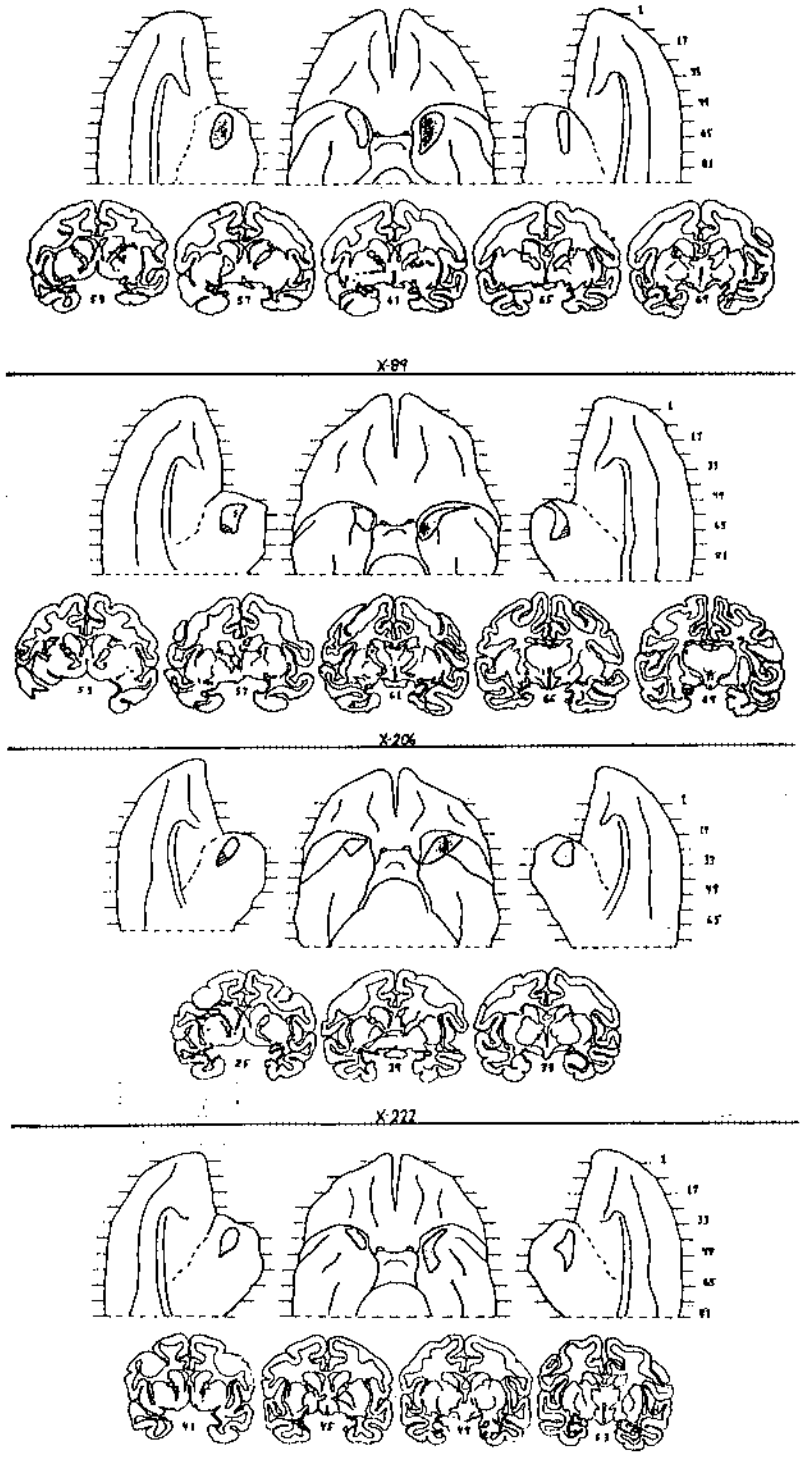



Fig. 8. A pulse histogram obtained in the same fashion as that reproduced in Fig. 7. Here the influence of concurrent temporal (it.) (2nd trace) and concurrent frontal (fr.) (4th trace) cortical stimulation on the flash (f.) evoked activity of the same single unit is shown. Note that the first silent period is lengthened by concurrent frontal, and shortened by concurrent temporal, cortex stimulation.

## THE REGULATION OF ORIENTING AND THE AMYGDALOID COMPLEX

Bilateral amygdalectomy (Fig. 9) interferes drastically with the orienting reaction as gauged by the galvanic skin response (G. S. R., Bayshaw *et al.*, 1965; Kimble *et al.*,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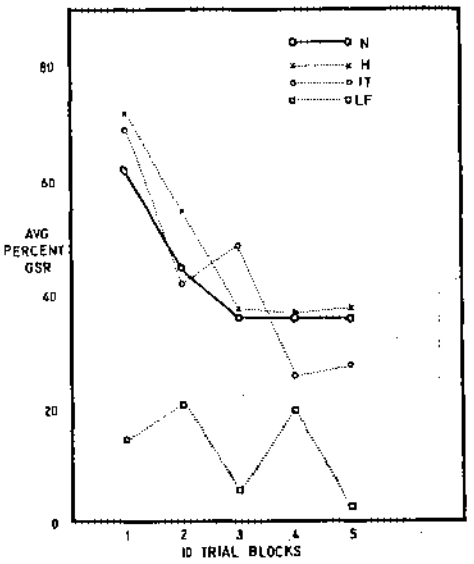


Fig. 10. Curves of average (AVG) % GSR response to the first 30 presentations of the original stimulus for the normal and three experimental groups.

1965; Koepke and Pribram, 1966. See Fig. 10). However, the behavioral effect of this interference is not simple. In a variety of discrimination learning tasks, some of which amygdalectomized monkeys found more difficult than their controls, a behavioral measure of orienting was taken (Bateson, submitted to *Science*). This measure consisted of noting the flick of the monkey's ears during the time the cues were presented. Normal monkeys show this flick of theears while they are learning; once a task has been mastered this ear response no longer occurs. Amygdalectomized monkeys show a longer total time during which such ear flicks occur, especially in those tasks in which they showed impairment.

These results led to the idea that orienting was made up of two components — one an alerting reaction indicated by the ear flick, the other a focusing function which allowed registration of the event which produced the alerting. In is this second stage which involves the amygdala and is signalled by the appearance of a GSR.

The two phases of orienting fit the model presented. The first phase, alerting, can be explained as a consequence of initial disinhibition of collateral inhibition. In the absence of a secondary controlling mechanism this reaction would overcome the stabilizing mechanism provided by recurrent inhibition. Events would continually be noticed but *adjustment* of the stabilizing mechanism (habituation) precluded. This is believed to be the case after amygdalectomy. By contrast, in normal subjects, collateral inhibition is in turn inhibited by the operation of the amygdaloid mechanism. This provides the reaction with a stop mechanism which increases the likelihood that its specific

Fig. 9. Reconstructions of the bilateral lesions of the amygdaloid complex. Black areas denote the lesion.

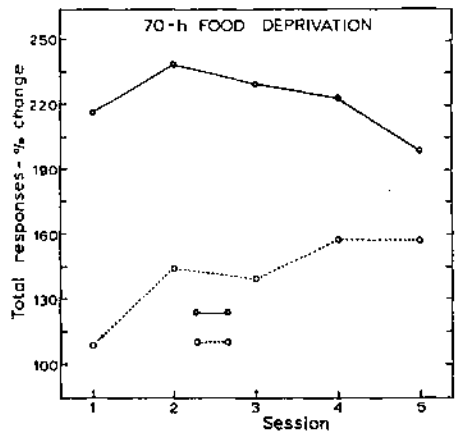


Fig. 11. Mean percentage changes in total responses of test sessions which followed prolonged deprivation of food. The values in the legend refer to the range of total responses for the three preceding control sessions on which the percentage changes are based.

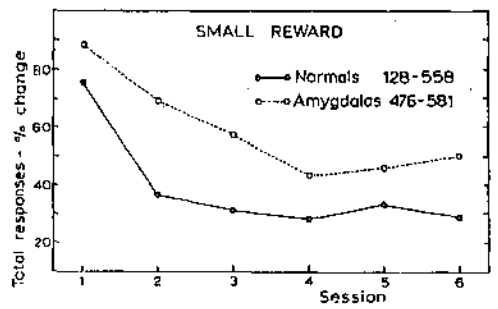


Fig. 12. Mean percentage changes in total responses in test sessions with the small reward. (The values in the upper right refer to the range of total responses for the three preceding large-reward sessions on which the percentage changes are based; group differences are not statistically significant).

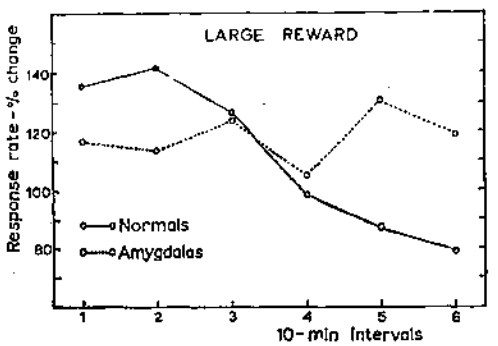


Fig. 13. Mean percentage changes in total responses within test sessions with the large reward.

configuration will be stabilized, *i.e.* registered. A difficulty in registering events should show up behaviorally in a variety of ways. One of them certainly would be in direct measures of habituation. Short term measures should show an increased speed of habituation; on the other hand, longer term measures should show that such habituation had failed to incorporate the orienting experience. This is exactly what has been found (Schwartzbaum, 1964). Another consequence of difficulty in registration would be the relative inefficacy of reinforcement. And, indeed, a series of experiments has shown that changing the amount of reward or its size (Schwartzbaum, 1960a, b) or the distribution of its occurrence (Schwartzbaum, 1961), has considerably less effects on amygdalectomized monkeys than on their controls (Figs. 11, 12, 13).

## THE HIPPOCAMPAL FORMATION AND HABITUATION

Douglas (Douglas and Pribram, 1966) formulated in precise behavioral terms a theory that I have taken the liberty of incorporating into my model. He suggested that the amygdala system operates as a reinforce-register mechanism and that the hippocampal formation serves to evaluate error. Several ingenious experiments were devised to test hypotheses derived from the theory. I shall present three of these. All were performed in an automated discrimination apparatus which allowed programming of tasks by a special purpose computer which could also be used for data reduction and analysis (Pribram *et al.*, 1962; see Figs.).

Douglas modified a standard behavioral testing procedure to his purpose. The procedure is called probability matching and in it subjects are trained to discriminate

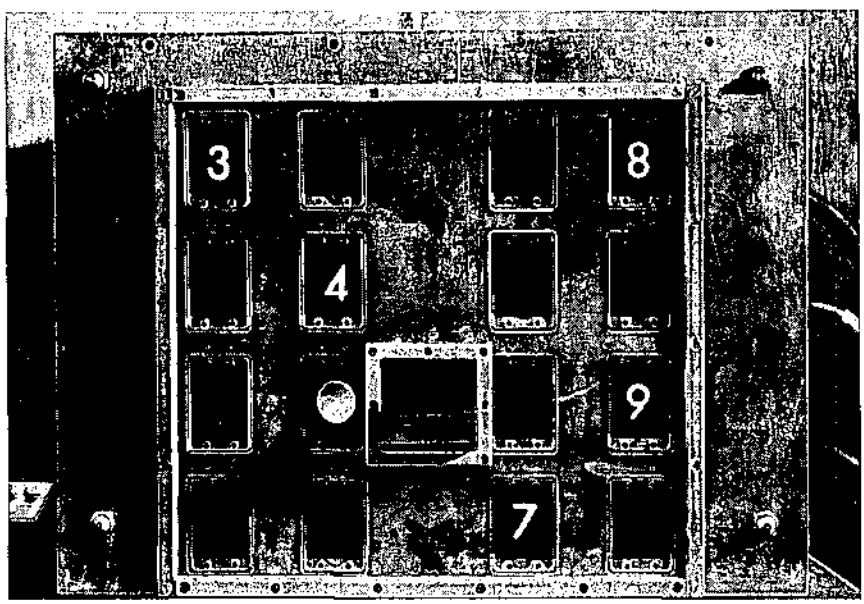


Fig. 14. Display panel of the automated discrimination apparatus. Note 16 clear hinged windows through which patterns can be displayed, and central tray attached to feeder mechanism.

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between two cues. Ordinarily one cue is rewarded 100% of the time and the other is never rewarded. In a probability matching task, however, one cue is rewarded some percentage less than 100 — say 70% — while the other cue is rewarded on the remaining occasions — in this instance, 30% of the time. This task is, of course, more difficult than the ordinary discrimination. The probability test is more interesting, however, since different organisms demonstrate different strategies in solving the problem. Douglas trained monkeys (bilaterally amygdalectomized, hippocampectomized and sham operated controls) in such a probability matching situation and then paired a *novel* cue with either the most- or the least-rewarded of the familiar cues. His results were striking.

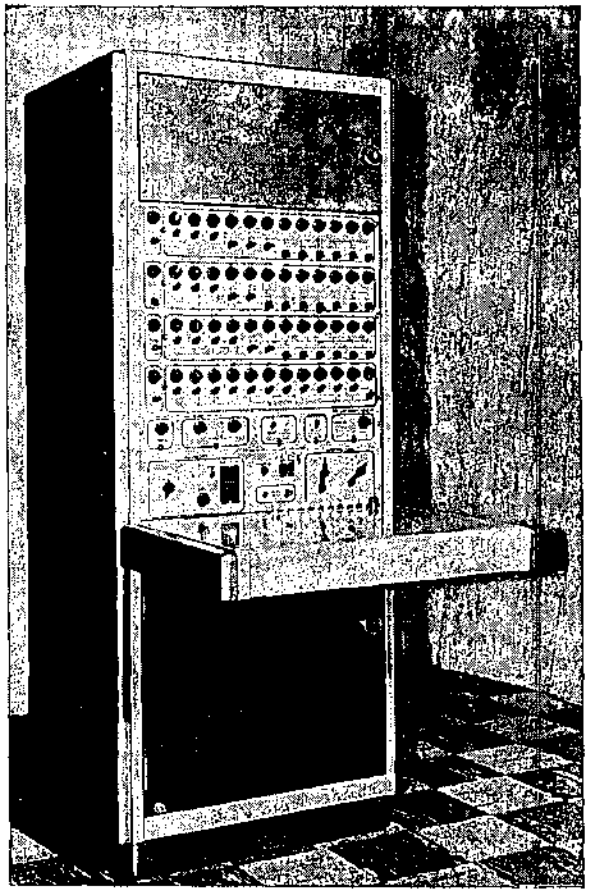
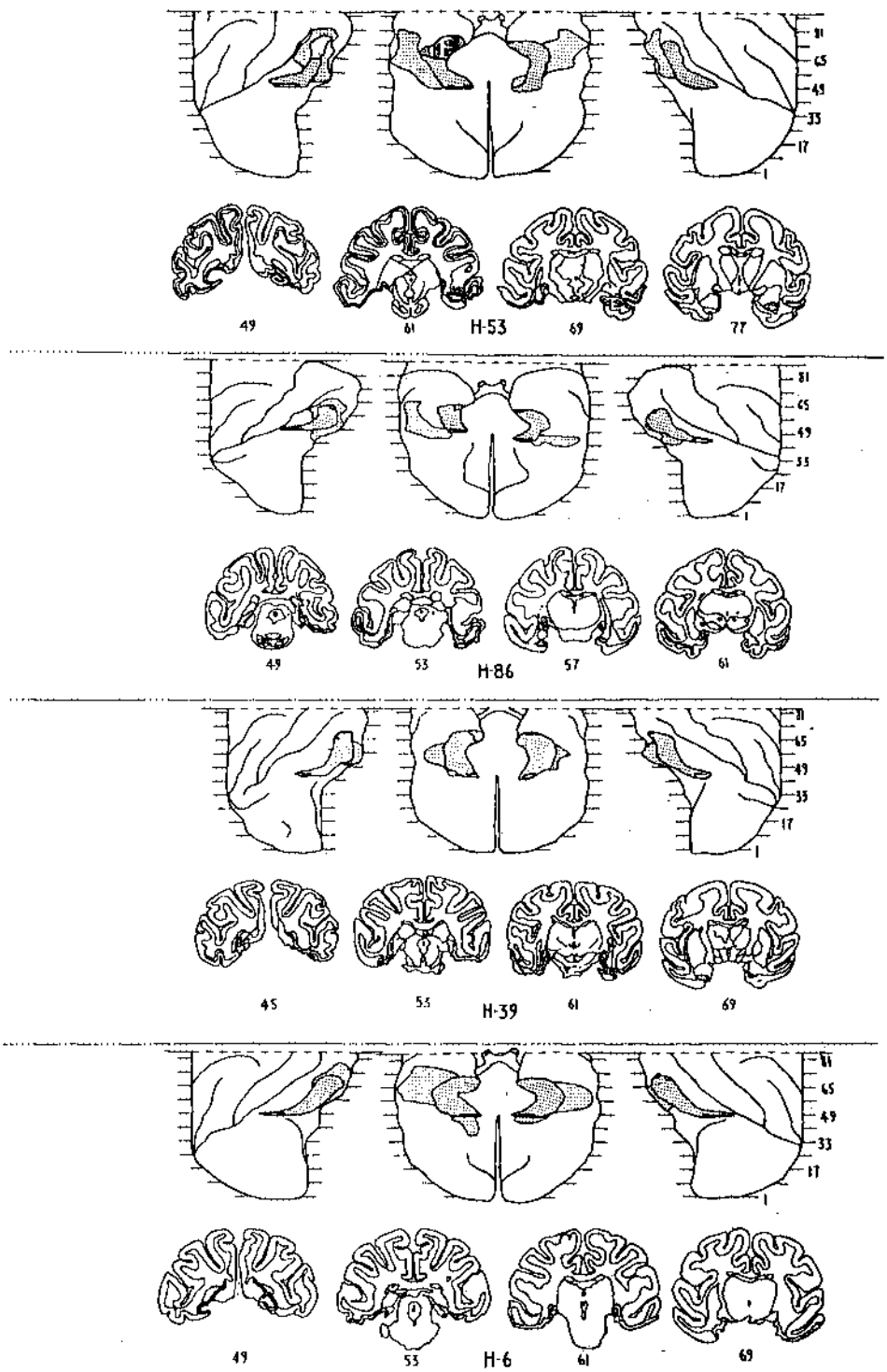


Fig. 15. Control console and special purpose computer for the automated discrimination apparatus which allows programming of tasks as well as data reduction and analysis.

Fig. 16. Reconstructions of the bilateral lesions of the hippocampus. Note that in this figure dashed areas on the reconstructions denote the lesion, black areas denote sparing. Dotted areas show the overlying cortex removed in the approach. Heavy lines on the cross-sections show the extent of the lesion on the ventral surface.

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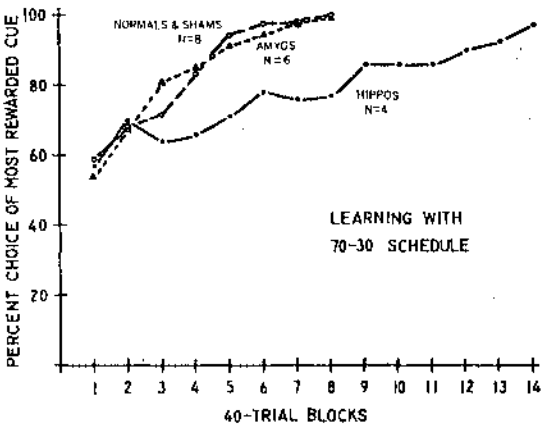



Fig. 17. Percent choice of most rewarded cue in probability task involving learning with a 70–30 schedule.

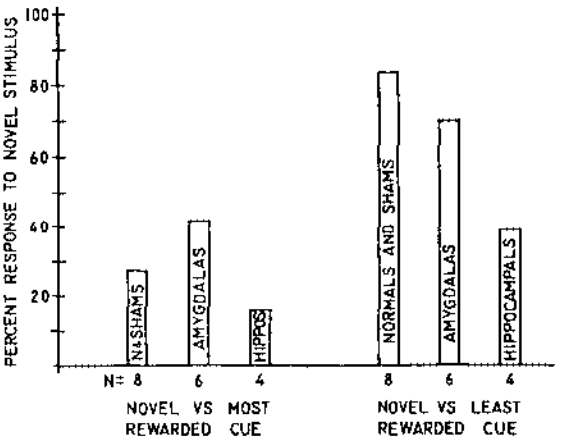



Fig. 18. Percent response to novel stimulus in groups of novel vs. most rewarded cue, compared with groups of novel vs. least rewarded cue.

First, monkeys with hippocampal lesions learned the probability task more slowly than did the other groups. This slower learning is interpreted as consonant with an impaired error-evaluate system in the hippocampectomized monkeys (*cf.* Figs. 16, 17).

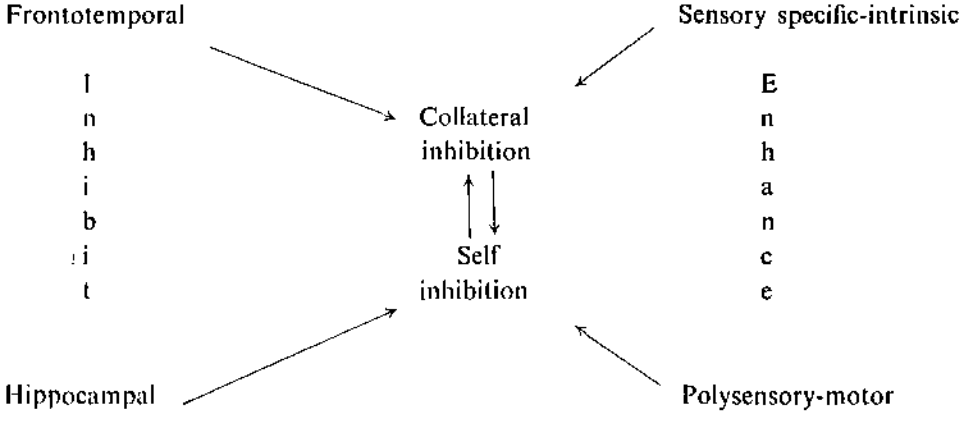
Second, monkeys with hippocampectomies, when compared with the other groups, chose the familiar cue more often when this was paired with a novel cue, irrespective of whether that familiar cue had been reinforced on 70% or 30% of the trials. The choice of the familiar is also consonant with an intact reinforce-register function and an impaired error-evaluate mechanism (Fig. 18).

Finally, the cues used in the probability matching task were again presented, this time without reinforcement. As could be predicted, control subjects quickly shifted their responses away from the previously rewarded cue since these responses were now erroneous. And again, hippocampally ablated monkeys came to the support of the theory by failing to shift their responses on the basis of error (Fig. 19).

As already noted, the behavioral process invoked to explain these results is an error-evaluate mechanism. On the basis of the model and data presented, the hippocampus is suggested to provide this mechanism. By inhibiting recurrent inhibition, the erroneous experience is allowed to register. In the absence of the hippocampus, the stabilizing effect of recurrent inhibition is assumed to be sufficiently strong to overcome the registration of nuances: the system of afferent inhibitory processes tends to revert to the status quo ante. This hyperstability is overcome only if the orienting events are overwhelming or if they recur regularly. Probabilistic occurrences, such as errors, fail to 'get through'. According to this view, short term habituation should be slowed by hippocampectomy and registration limited to regularly recurring events. There is evidence in support of both of these statements (Douglas and Isaacson, 1964; Roberts *et al.*, 1962; Kimble, 1963).

## SUMMARY AND CONCLUSION

The model is now complete. Collateral and recurrent afferent inhibition are bucked against one another, forming a primary couplet of neural inhibition within afferent channels. Four forebrain mechanisms are assumed to provide efferent control on this primary couplet:




Two of these, frontotemporal and sensory specific-intrinsic (which includes the inferotemporal cortex), work their influence by regulating collateral inhibition; two others, hippocampal and polysensory-motor, regulate recurrent inhibition. The sensory specific-intrinsic and polysensory-motor 'association' cortical systems exert their control by enhancing, while the frontotemporal and hippocampal systems exert control by inhibiting afferent neural inhibition.

According to the model, orienting is a function of changes in the pattern of collateral inhibition; habituation is due to recurrent inhibition elicited in response to this changed pattern. Registration of experience is a function of habituation. Complex problem-solving is dependent on evaluating erroneous experiences: these, because they are

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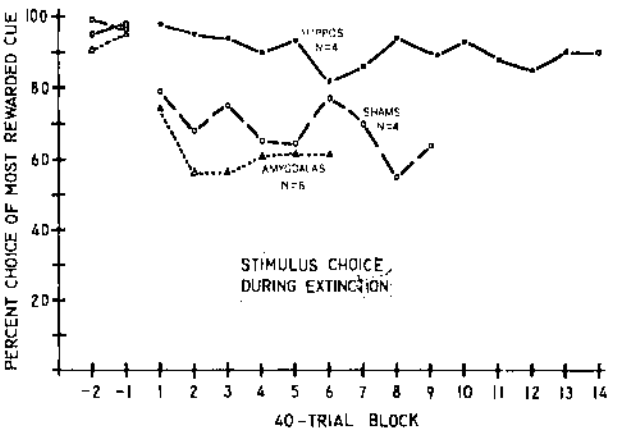


Fig. 19. Percent choice of most rewarded cue in 40-trial block, where cues used in the probability matching task were again presented, this time without reinforcement.

nuances, can only be registered if the hyperstabilizing influence of recurrent inhibition is in turn inhibited.

Data were presented to show that the amygdaloid complex regulates the orienting reaction. These data were interpreted, according to the model, as indicating that the amygdala ordinarily originates a process of efferent inhibition of collateral inhibition. This process stops the change in pattern of collateral inhibition from proceeding at such a rate, and to such an extent, as to preclude revision of the pattern of recurrent inhibition. Registration occurs only when such revision has taken place.

Data were also presented to show that the hippocampal formation is involved in the handling of erroneous experience. A case was made to the effect that ordinarily negative instances of experience must be evaluated. According to the model, the hippocampus, by efferently inhibiting recurrent inhibition, ordinarily provides a mechanism for allowing the registration of nuances. Here again, revision of the pattern of recurrent inhibition is dependent on an efferent inhibitory mechanism. In this instance, however, efferent inhibition overcomes the tendency of recurrent inhibition toward hyperstability.

This neurological model of information processing has helped me considerably in understanding the wealth of neurophysiological and neuropsychological data available. Some of this understanding has been brought to bear here on the problem of limbic system function. Aside from further tests of the model, the job ahead is to devise experiments which will allow extension of the model to such problems as the regulation of action, memory and emotion.

ACKNOWLEDGEMENT

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