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LEARNING AND LIMBIC LESIONS*

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Abstract—A model is presented which identifies the amygdala and hippocampus with two distinct attention-directing processes. The process to which the amygdala contributes is postulated to heighten awareness of experiences as a function of previous reinforcement, while the process to which the hippocampus contributes acts to diminish awareness of experience as a function of the probability of non-reinforcement. The manner in which these processes govern problem-solving behavior is outlined, and a number of hypotheses generated. These hypotheses were tested using monkeys with hippocampal and amygdaloid lesions. The results of these experiments were generally in agreement with the model, and the two lesion groups were found to be as distinct from each other as either group was from sham-operated controls.

1. INTRODUCTION

IT IS the belief of the present authors that progress in the field of physiological psychology has been greatly retarded by the lack of appropriate theoretical development. There is at the present time no general model of problem solving which contains the variety of variables necessary to explain the results of many types of brain lesions. It is possible that the present approach in which the effects of lesions are described in terms of established theory should be reversed. A "backwards" procedure of constructing learning models based on variables abstracted from ablation or electrophysiological data might prove to be more fruitful. The present authors have attempted to make a start in this direction, however shakily, through the presentation of a model derived from the behavior of animals with hippocampal and amygdaloid lesions, and the confirmation obtained in the results of a series of experiments which were carried out to test empirically the validity of the model. While this model was designed to operate at the behavioral level, it can also be related to variables which refer to events at the cellular level, and converges with the studies carried out in this laboratory on the efferent control of sensory reception. There are, in fact, three levels to the model, and the model must be tested on all three before its general validity can be established.

The development of the theory began with an attempt to show that the behavioral changes resulting from hippocampal lesions could be ascribed to the loss of a single process which normally contributes to problem solving. In describing this process, however, it soon became apparent that additional processes had to be taken into account in order to explain the behavior of which hippocampectomized animals *are* capable. One additional process, especially relevant, appeared to be ascribable to the amygdala. These two processes

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were tentatively described and a series of experiments performed to check on some of the important points. Thus, the development of the model and the data gathering went hand in hand so that the current form of the theory presented here was attained only after these studies were completed. Each experiment was, however, a test of one of the hypotheses generated by the rudimentary model. This leads to problems of presentation, and for this reason the more complete model will be presented before the results of the experiments which helped to determine it will be discussed. No pretense is made that all of these details were formulated before the results were in.

Problem solving usually begins in a situation to which the subject is habituated. Until recently, habituation was thought to be due to an increased neural threshold to input. Recent experiments have, however, radically changed this view (SOKOLOV [1], HAIDER *et al.* [2], BAGSHAW *et al.* [3], KOEPKE and PRIBRAM [4]). There can no longer be much doubt that habituation is evidence for the organization of a complex neural process—a neuronal configuration against which the subsequent inputs are compared. This neuronal configuration is a highly specific encoded representation of the situation experienced by the organism.

The first major postulate of the theory states that any aspect of the situation leading to a perturbation of this neural configuration is experienced by the organism. Such perturbation is signalled by an orienting reaction. Several physiological indices of orienting identify that it has occurred. Repetition of the experience leads in turn to its habituation, i.e. its registration. Only when experience is registered can it effectively guide behavior. However, once habituated, experience is no longer experienced. Data will be presented to support the view that the amygdala is important to the system allowing experience to be registered.

The second major postulate of the theory is also concerned with the registration of experience. This postulate states that the neuronal configuration against which inputs are compared displays considerable inertia. The tendency of the mechanism is to return to the *status quo ante* after perturbation. This characteristic disposes against the registration of experiences, especially if they are probabalistic rather than overwhelming or regular in their recurrence. Errors are an example of such probabalistic experiences. Data will be presented to support the view that the hippocampal formation is part of a system which imposes an override on the inertia of neuronal configurations and thus allows the registration of errors.

2. THE REINFORCE-REGISTER PROCESS

(1) This process acts to *increase* the probability that an experience becomes registered.
(2) Increments are added to the probability of registration whenever an experience is followed closely in time by an environmental change which has "significance" to the subject at that time. Such a change is usually produced by the subject's own response, though this is not the only condition for registration. Nonetheless, reinforcers, the consequences of action, are the most ubiquitous registrants.
(3) Registration of experience alters the way subsequent experiences are experienced. This residual impact of experience is termed *impellance*.
(4) The size of the increment of increase in impellance due to a single experience is related to the magnitude of the reinforcement and the amount of work or effort necessary to produce it. Total impellance is related to the number of such experiences plus innate factors. Since impellance increments for constant events or rewards are assumed to be

constant in size, each reinforcement adds relatively less to the total, as each increment is smaller in relation to the total. Thus, the first few increments should produce a greater change in impellance than the later ones. For this reason the reinforce-register system should be of greatest effectiveness in the very early stages of learning. This is true because if two stimuli are simultaneously present the probability that the first of them will be experienced, rather than the second, is proportional to the ratio of their impellances.
(5) Impellance increments are assumed to be permanent and resistant to disruption. However, the effects of registration can be overridden by the error-evaluate process to be discussed below. The reinforce-register system is insensitive to errors, as these constitute a lack of something, rather than a positive experience, and they are handled by the second system. Errors are not to be confused with punishment, which is a positive event, and which does act to increase impellance much as a reward does. There is, however, a possibility that after regular reinforcement the withholding of reward may be punishing.
(6) The amygdala is assumed to be involved in the *increasing* of impellance, but not necessarily in its storage. Experience which has gained impellance may retain it after removal of the amygdala, although this point has not been fully established. The identification the neurological system important to the reinforce-register process is based on the frequent association of the amygdaloid complex with the reinforcing properties of stimuli.
(7) At the primary neuronal level, increases in impellance are suggested to involve inhibitory control over normally occurring collateral inhibitory processes in the afferent systems. Evidence from the study of recovery functions in afferent systems in our laboratory indicates that recovery of neuronal sensitivity to recurring stimuli in the afferent systems is appreciably shortened by concurrent chronic stimulation of the amygdala.

The suggestion is that orienting is a function of the enhanced contrast produced by collateral inhibition. When rapidly repeated changes in input are accentuated by this contrast-enhancing mechanism, the recurrent form of inhibition which opposes the changes and stabilizes the inhibitory patterns within the afferent channels is in danger of being disrupted. The configuration of recurrent inhibition, conceived as the basis of habituation, must be gradually altered, not disrupted, if progressive registration of inputs is to be accomplished. The function of the amygdaloid complex is to provide a stop to the collateral disinhibitory process in rapidly changing situations, thus securing the possibility for gradual and effective change in the afferent inhibitory mechanism.

3. THE ERROR-EVALUATE PROCESS

(1) This process acts to *decrease* the probability that an event will be experienced.
(2) This system becomes effective in two stages. In stage 1 experience is registered, and in stage 2 behavior is determined by the previous experience. During the first stage the error-evaluate system is inactive, as far as the determination of behavior is concerned, as this is the registration stage. The length of stage 1 is a function of the complexity of the situation and the past history of the subject in similar situations, both of which determine the amount of experience which must be registered before this system can become effective. Stage 2 begins when the subject, on the basis of events experienced in stage 1, begins to actively "ignore" recurrences of the formerly experienced events. These seemingly ignored events are said to be *gated out* of awareness. When an event is gated out it is not experienced.
(3) The error-evaluate system is sluggish, as compared to the rapidly acting reinforce-register system, due to the necessity for stage 1. Further, this system is assumed to be the

more sensitive of the two to disruption or override. The configuration of the gating process is labile, and can be removed or shifted on the basis of further experience.
(4) The error-evaluate system is assumed to play a part in most, if not all, classes of behavior in which formerly experienced events are seemingly ignored. The functions of this process in behavior become apparent in extinction, discrimination reversal, passive avoidance conditioning, habituation, alternation, and some types of sequential behavior. This process is operative in general, then, in tasks which must be solved by the avoidance of behavior which results in "errors", i.e. which is not reinforced.
(5) The hippocampus is suggested to be a key structure in the neurological system important to the error-evaluate process since its removal results in impaired behavior on tasks such as those above.
(6) The primary neuronal mechanism involved is thought to be an inhibitory control over normally acting recurrent inhibition in afferent systems. Such control would allow more subtle changes occurring in stage 1, orienting, to influence stage 2, the habituation or gating process. Thus, the tendency towards hyperstability of the afferent inhibitory mechanisms would be modulated. Without such a controlling influence, only regularly recurring or disrupting orienting experiences could become registered.

In summary, we proposed two processes important to the problem-solving mechanism. The first regards events which produce *increments* in awareness. On the basis of the organism's current experiences with the problem, attention is focused on relevant aspects which then become stimuli. The second system is concerned with behavior which produces *decrements* in awareness. On the basis of the subject's prior experience irrelevant aspects of the situation become ignored. Both processes are modulated so that their operation does not overwhelm the problem-solving mechanism. The basic mechanism is assumed to involve orienting and its habituation, which are conceived neurologically as the afferent inhibitory processes of collateral and recurrent inhibition. Modulation is conceived neurologically to concern efferent controls over afferent inhibitory processes. This presentation is an attempt to spell out the roles of the hippocampus and amygdala in these efferent control mechanisms.

4. GENERAL METHOD

4.1. *Subjects*

Subjects were twelve immature rhesus monkeys. Four of these were subjected to one-stage bilateral removal of the hippocampus by suction through an opening in the entorhinal cortex as described by PRIBRAM and WEISKRANTZ [5]. Six monkeys had their amygdalas removed bilaterally by suction in a manner described by PRIBRAM and BAGSHAW [6]. Four animals had sham operations which involved removal of the same skull and manipulations of the areas normally removed prior to hippocampectomy and amygdalectomy. All animals had been "shaped" and tested in several situations before the operation, and all could be considered to be highly sophisticated subjects.

4.2. *Apparatus*

All behavioral testing was done in the Automated Apparatus for Discrete Trial Analysis (DADTA), which is described in more detail by PRIBRAM *et al.* [7]. In this apparatus the subject sits before a group of sixteen small depressable panels upon which various stimuli can be projected. Depression of that panel upon which the correct stimulus is presented results in the delivery of a peanut reward to a centrally located food cup. The testing

enclosure is just large enough (2 × 2 ft) to contain the transport cage and eliminates all direct communication between the experimenter and the monkey. The subject could, however, by seen by the experimenter through a one-way glass mirror. The enclosure was illuminated by an overhead fluorescent light, and ventilated by means of a blower. The experimenter and the computer which regulates presentation of stimuli and rewards were located in a room adjacent to the one containing the testing enclosure. The monkey's performance was recorded on punched tape by the computer and in a protocol based on direct observation.

4.3. *Testing procedures*

All problems involved the projection of two different stimuli onto a randomly determined pair of panels within the middle two rows of panels (eight possible panels). Within these rows stimuli could be either side by side, one over the other, or diagonal to each other. An exception to this procedure was in the preoperative training and the post-operative retention testing of two animals, in which all panels were used, and the stimuli were not paired in space. The reasons for the restriction in location of stimuli will be discussed later. Stimuli consisted of either letters of the alphabet, numbers, or colored geometric symbols.

Two types of reward schedule were used. One was the conventional schedule in which one stimulus is always rewarded while the other one never is. The other schedule consisted of rewarding one stimulus 70 percent of the time and the other one 30 percent of the times it was pressed (Differential Partial Reinforcement). This was accomplished by drawing up a schedule of rewards in advance of training. On a block of forty trials the most rewarded stimulus would be rewarded, if pressed, on twenty-eight trials, with the least rewarded stimulus being rewarded, if pressed, on twelve trials. The sequence in which the potential rewards were made available was limited by the condition that in every two blocks the least rewarded stimulus would be "correct" three times in a row only once; twice in a row on five occasions; and in isolation eleven times. These limitations guarded against long sequences in which one or the other stimulus would be continuously rewarded and also reduced a possible source of irrelevant variability from problem to problem. The intertrial interval was always 8 sec unless otherwise specified. The order in which experiments were performed, and the stimuli used, are presented in Table 1.

4.4. *Histology*

Histological examination has not been undertaken as yet because these animals are scheduled for further testing. However, identical operations by the same surgeon in the past have resulted in nearly complete destruction of the target areas, with minimal damage to the surrounding tissue (see PRIBRAM and WEISKRANTZ [5] and PRIBRAM and BAGSHAW [6]). Since the present lesions were made with visual guidance, there can be little doubt that the given areas were subject to massive damage, but the extent of removal, or the degree of damage to surrounding areas cannot, of course, be known for certain.

5. EXPERIMENT I: LEARNING WITH DIFFERENT REINFORCEMENT SCHEDULES

The conventional discrimination task, in which one stimulus is always rewarded while the other one is never reinforced, can be seen to be merely one end of a continuum on which two stimuli are rewarded with varying degrees of probability. At one end of the

Table 1. Order in which tests were given

Test	Order	Group tested	Stimuli
Learning (70–30)	1	Pre-op. Hip. Shams	6 vs. 4 S vs. R
Novelty test	2	Pre-op. Hip. Shams	6, 4 vs. 0, 1, 2, 5, 7, 9 S, R vs. A, B, E, F
Retention (10-day)	3	Hip. Shams	6 vs. 4 S vs. R
Novelty test	4	Hip. Shams	6, 4 vs. 0, 1, 2, 5, 7, 9 S, R vs. A, B, E, F
Learning (70–30)	5	Hip. Shams Amyg.	S vs. R X vs. N S vs. R
Novelty test	6	Hip. Shams Amyg.	S, R vs. A, B, E, F X, N vs. A, B, E, F S, R vs. A, B, E, F
Extinction	7	Hip. Shams Amyg.	S, R X, N S, R
Paired vs. scattered	8	Hip. Shams Amyg.	9 vs. 2 A vs. E A vs. E
Reversal (70–30)	9	Hip. Shams Amyg.	2 vs. 9 E vs. A E vs. A
5 vs. 20 sec. interval	10	Amyg.	X vs. N Green cross, blue square
Learning (100–0)	11	Hip. Shams Amyg.	Large vs. small yellow circle, or *vice versa*
Reversal (100–0)	12	Hip. Shams Amyg.	Same as above

continuum (the 100–0 end) a discrimination problem should be learnable with great efficiency if only an intact reinforce-register process were used, with any contribution by the error-evaluate process being largely redundant. This should be the case because the recurring regularity of the situation leads to certain increase in impellance. Further, according to the model, the first few rewards should have a relatively large effect. The error-evaluate system, on the other hand, should be relatively unimportant since it requires a period of information gathering during which the problem could already have been solved by means of the other process. This would be true, however, only if the Ss entered the problem without an advance 100–0 expectancy or set. Were such a set in operation a single error might suffice to initiate gating. In the present experiment set was manipulated so as to minimize this possibility by using the 100–0 schedule after all other tests had been finished. Thus it was expected that those animals with intact reinforce-register processes

would learn with the 100–0 schedule in fewer trials than those in which the processes were uncontrolled. In other words, the sham-operated and the hippocampectomized Ss were expected to learn faster than the monkeys with amygdala lesions.

At the other end of the continuum, a discrimination problem could be given which would be learned only with extreme difficulty were an intact reinforce-register process the only one in operation. As an example, suppose that one stimulus was rewarded 51 percent of the time while the other was rewarded 49 percent of the occasions on which it was pressed. Since the impellance of the most rewarded stimulus should grow faster than that of the least rewarded, the reinforce-register system should by itself lead to eventual maximization. However, since the consequences of responding to both stimuli increase impellance, the difference between the two should grow only with a rate best described as painfully slow. The rate of learning (or maximization) could be greatly speeded up, however, through gating-out the least rewarded stimulus. According to the model an intact error-evaluate process would be expected to function in just this fashion to speed up the learning process, and so the hippocampectomized Ss in which the process is assumed to be impaired would be greatly retarded in learning the differential partial reinforcement task. In the present experiment the extreme example mentioned above was not used because it might prove too difficult. Instead, a more moderate schedule was used in which one stimulus was rewarded 70 percent of the time, and the other 30 percent. The model would predict eventual maximization for all groups, with the hippocampectomized Ss lagging far behind those with amygdala lesions and sham operations.

5.1. *Method*

In the 100–0 problem the stimuli consisted of yellow circles of varying sizes (either 1 in. dia. vs. $\frac{3}{4}$ in., or $\frac{3}{4}$ in. vs. $\frac{3}{8}$ in.), with half of the Ss in each group having the larger circle positive, and half the smaller circle. All Ss were trained until they had reached a criterion of 37 out of 40 trials correct. An attempt was made to train all Ss in one session, but several animals took so long to learn that two sessions were necessary (all belonged to the amygdalectomized group).

In the 70–30 problem, four groups were actually tested, rather than three, as the hippocampectomized Ss were trained pre-operatively before any surgery was performed, tested for postoperative retention, and then trained with a new set of stimuli. Although the sham operation had already been completed before these animals became available for this study, they were given the same training experience as the hippocampals, including a 10-day "postoperative" retention test. Groups were compared only on the first 70–30 problem learned with their given brain lesion or condition, with the second problem of the sham group not counted in the analysis. Stimuli used in this problem were "S" vs. "R" for the operated groups, while the unoperated Ss were trained to "6" vs. "4". All Ss were trained to a criterion of thirty-six responses to the most rewarded stimulus in a block of forty trails. Ss were usually given one hundred and twenty consecutive trials in a daily session, with rewards determined in the manner outlined in the General Discussion section.

5.2. *Results*

On the 100–0 problem the results were as predicted, with the sham- and hippocampally-operated Ss learning at about the same rate (see Fig. 1), and with the amygdalectomized group requiring significantly more trials to criterion than the other Ss ($t=2.2$, $P<0.05$).

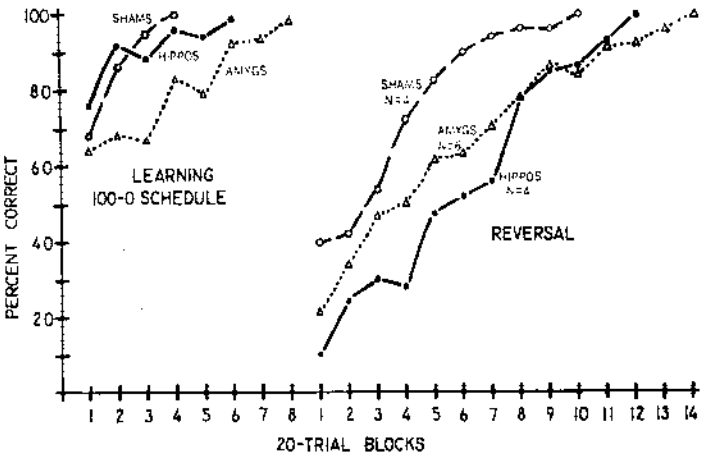

FIG. 1. Learning and reversal with 100–0 schedule.

In addition to a general slowing of acquisition, the amygdalectomized group tended to show more variability in their progress from one block of trials to the next; however, this difference fell short of significance. The failure to find a difference between the hippocampally- and sham-operated Ss in this type of problem confirms other reports in the literature (KIMBLE [8], KIMBLE and PRIBRAM [9]). Slower learning on a problem of this type has also been previously reported for amygdalectomized monkeys (SCHWARTZBAUM and PRIBRAM [10]).

While the 70–30 problem proved to be more difficult than the 100–0 for all groups, it was especially so for the hippocampal group, as can be seen in Fig. 2.

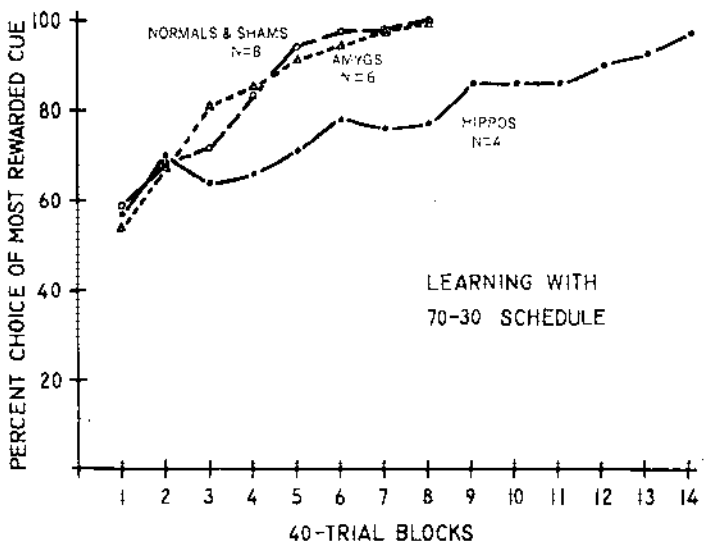

FIG. 2. Learning with 70–30 schedule.

The hippocampectomized Ss required significantly more rewards on the most-rewarded stimulus before reaching criterion than did the sham-operated and normal groups ($t=2.7$, $P<0.05$) or than did the amygdalectomized Ss ($t=2.5$, $P<0.05$). Sham-operated and normal Ss were combined into one group, as they had intact brains and did not differ from each other in performance on this task. The amygdalectomized group did not differ significantly from this sham-normal group in performance.

5.3. *Discussion*

While these results were just about as expected on the basis of the model, the data were further analyzed in an attempt to decide if the shapes of the learning curves were also in accordance with the model. "Backward" learning curves were plotted for all groups on both problems in an attempt to determine the course of the learning process in more detail. These curves showed that all groups except the hippocampal had abrupt increases in the level of their performance just prior to achieving criterion. In contrast, the hippocampal group showed a relatively gradual improvement which confirms the expectations based on the model. The sudden jump in the learning curves of the sham, normal, and amygdala groups is assumed due to the initiation of the gating process.

The results of backward plotting of behavior in the 100–0 problem were like those obtained in the 70–30 situation. Even though the amygdalectomized Ss were relatively slow in learning this task, they did not learn gradually, as did the hippocampectomized Ss in the 70–30 problem, but instead, suddenly improved after a long period of only slight improvement. The hippocampal group also behaved according to expectations based on the model in that their improvement was relatively greater in the earlier stages than in the later. The sham-operated group, however, did not act as expected. Their improvement was much faster than would be expected if their learning was based on the same process as that of the hippocampal group. This suggests that even though the error-avoidance process may not be necessary for fast learning in a 100-0 type of task, it is in operation anyway. Learning curves obtained from other studies in this laboratory indicate that a sudden jump in performance is often found in 100–0 tasks when the discrimination involved is a difficult one (BLEHERT [11]). This suggests either that gating is always used in problems of this type, but that its appearance is masked when learning is extremely rapid, or that gating processes are more critical when discriminative stimuli are more similar. The latter would imply that the reinforce-register process is more subject to stimulus generalization than is the error-evaluate process.

6. EXPERIMENT II: REACTION TO NOVEL STIMULI

While the group differences obtained in Experiment I basically confirm the predictions of the model, the present experiment was carried out in order to further determine whether the 70–30 problem had been acquired by the different groups in the *manner* suggested by the model. It was reasoned that if the sham and amygdalectomized groups had made use of the error-evaluate process in learning this problem, then these groups should appear to ignore the least-rewarded stimulus, when this was subsequently paired with an indifferent or novel stimulus, while the hippocampectomized group should not. The hippocampal group should, in fact, be expected to show an inability to ignore the least-rewarded stimulus since experience with it should have gained impellance through the reinforce-register process by being rewarded on many occasions. On the other hand, the sham and

hippocampectomized groups, having made use of the reinforce-register process, might be expected to show an apparent affinity for the most-rewarded stimulus when this was subsequently paired with a novel stimulus, while amygdalectomized Ss should find it no more impelling than the new stimulus and thus show little preference between the two. These hypotheses were tested in the following manner.

6.1. *Method*

Immediately after reaching criterion in the 70–30 problem each S was given a series of twenty-four trials on which either the most-rewarded stimulus or the least-rewarded stimulus was paired with one of a group of novel stimuli and the animal allowed to press one or the other. On odd-numbered trials the most-rewarded stimulus was paired with one of the novel stimuli, while on even-numbered trials the least-rewarded stimulus was paired with the novel. The S was rewarded no matter what response he made. In the case of one group, six different novel stimuli were each used four times, but in all other groups only four novel stimuli were used, with six exposures each. The stimuli used were: normal Ss, 6 or 4 vs. 0, 1, 2, 5, 7, 9; sham-operated, hippocampectomized and amygdalectomized Ss, S or R vs. A, B, E, F. As noted in the General Method section, some groups were tested more than once on this task, but because of procedural differences only the results of each group's first test will be used here. An attempt was made to keep the intertrial interval at 8 sec, but this was not always possible in all instances.

6.2. *Results*

The results of the novelty test are shown in Fig. 3.

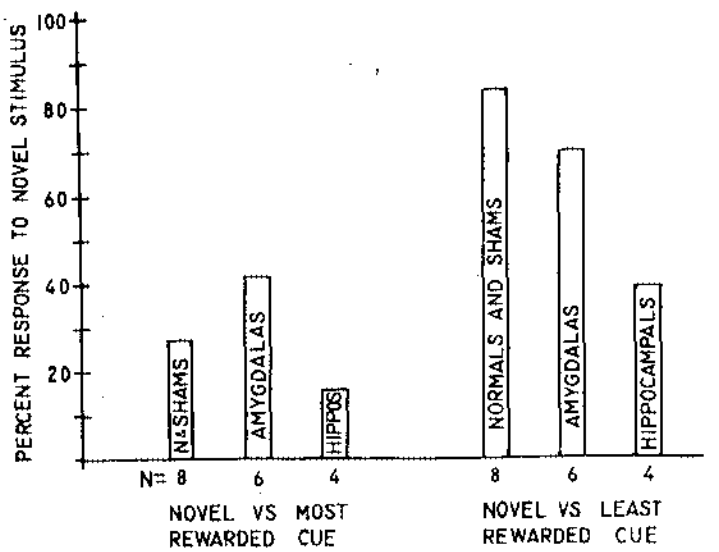

FIG. 3. Responses to novel vs. rewarded stimuli.

Since the sham-operated and normal Ss behaved almost identically on this test, the groups were collapsed. It can be seen that the results conformed to expectations based on

the model. All animals except those in the amygdalectomized group showed marked tendencies to respond to the most-rewarded stimulus when it was paired with a novel stimulus. The response of the amygdalectomized group to the most-rewarded stimulus occurred at a significantly lower rate than that of the sham-operated and normal groups ($t=1.84$, $P<0.05$) or that of the hippocampectomized group ($t=3.6$, $P<0.01$). The amygdalectomized Ss, in fact, chose the most-rewarded stimulus at a rate which was close to a chance 50 percent (58.3).

When the least-rewarded stimulus was paired with a novel stimulus the sham-operated, normal and amygdalectomized Ss, in contrast more often responded to the novel, rather than to the least-rewarded stimulus. The hippocampal group chose the novel stimulus at a rate significantly lower than that of the sham-operated and normal group ($t=7.8$, $P<0.01$) or that of the amygdalectomized group ($t=3.4$, $P<0.01$). The difference between the performance of the amygdalectomized, the sham-operated and normal Ss did not approach significance.

6.3. *Discussion*

These results again generally confirmed the model. In addition, some of the differences which did not reach significance indicate the direction in which the model can be further elaborated. For example, the hippocampectomized Ss did tend to choose the most-rewarded stimulus *more* often that did the sham-operated and normal Ss. Furthermore, observation of these normal animals during testing led to a distinct impression that they appeared to take notice of the new stimuli, while the hippocampally-lesioned Ss gave no indication that the situation had been changed. This may indicate that the error-evaluate process may have an additional function of dishabituating an organism whenever the "gate" does not match the input (a gating mismatch effect). A gating mismatch effect could be produced whenever recurrent inhibition was sufficiently inhibited so that a rebound would be produced in the process of collateral inhibition. This could well happen since the model assumes that the two processes of afferent inhibition do balance each other. Such a modification of the model would account for the lack of distractibility in hippocampectomized rats (WICKELGREN and ISAACSON [12]).

Another incidental test of the model occurred when the hippocampal Ss were given the novelty test using as rewarded stimuli some which had been learned before surgery. Under this condition, response to novelty was very much like it had been before the surgery. In other words, the abnormal reaction to novelty shown by the hippocampal group in the present experiment occurred only when learning had taken place in the absence of their hippocampi. This suggests that the gating itself occurs in the absence of the hippocampus, but that this structure is necessary for the original initiation of the process.

Even though the results of this experiment showed a remarkable homogeneity of individuals in each group, the novelty procedure is not as simple and clear-cut as it may appear. There is some evidence (unpublished) that different results would be obtained if *radically* new stimuli were used, or if varying amounts of overtraining had been experienced. Monkeys react more erratically when some types of changes are made in the stimuli to which they have become accustomed (BATESON, unpublished data). The present results should therefore not be taken as indicative of reactions by normal and operated monkeys to all kinds of novel stimuli, nor should it be assumed that the present results would have been obtained after prolonged overtraining.

7. EXPERIMENT III: EXTINCTION

In the model, the error-evaluate process is assumed involved in extinction. The reason for this is that the error-evaluate process directs attention away from the irrelevant aspects of a situation—irrelevancy being a function of non-reinforcement. According to the model, therefore, an organism whose error-evaluate system is impaired should be unable to switch responses from one of two presented stimuli and start responding to the other one in the absence of *differential* reinforcement. For example, if monkeys with hippocampal removal were trained to maximization on a 70–30 problem and then all rewards were withheld, these Ss should press only the previously most-rewarded stimulus, if they press at all. Those animals with intact error-evaluate systems, however, would be expected to begin gating out the previously most rewarded stimulus as it no longer leads to reward. This could lead to an absolute, and would certainly lead to a relative *increase* in responses to the previously *least*-rewarded stimulus; when both stimuli were shown equally irrelevant to reward, the animal should stop responding to them altogether. Thus, there should be a significant difference between the hippocampal-operated monkeys and those in the other groups in the frequency with which the most rewarded stimulus is pressed during a prolonged period of non-reinforcement. In addition, the hippocampals might be expected to extinguish more slowly to both stimuli. These hypotheses were tested in the following manner.

7.1. *Method*

Upon completion of the novelty test, all Ss were retrained on the preceding 70–30 task until all had received eighty trials of overtraining past the block on which they had reached criterion. This was done in order to insure a very high and stable level of performance. At this point all rewards were withheld, and each S allowed to press panels in the usual manner until extinction occurred. The criterion for complete extinction was five consecutive trials on which no response was made within 60 sec after presentation of the stimuli. Response latency was recorded on the last forty rewarded trials, as well as on all extinction trials.

7.2. *Results*

Figure 4 shows the mean latency (log) for all groups during extinction and on the preceding forty rewarded trials.

It can be seen that the curves for all groups are clearly distinct, with the amygdalectomized Ss having the longest latencies and the hippocampals the shortest. All groups were scored for the number of presses made prior to the cessation of responding at extinction. The only difference which was found to be statistically significant was that between the hippocampectomized group and a combination of the other two groups, with the hippocampal group requiring more presses to extinction ($t=1.92$, $P<0.05$). Thus, the hippocampal group tended to make more responses during the extinction procedure and to make these responses with a shorter latency than the other groups. Both observations indicate that monkeys with hippocampal lesions are relatively slow to extinguish.

Large differences were found between groups on the measure of the rate at which the most rewarded stimulus was preferentially responded to during extinction. Not counting the trials on which no response was made, this rate was 91.8 percent in the hippocampal group, 71.5 in the sham-operated group, and 61.9 in the amygdalectomized Ss. Significant differences were found between the hippocampal group and sham-operated Ss

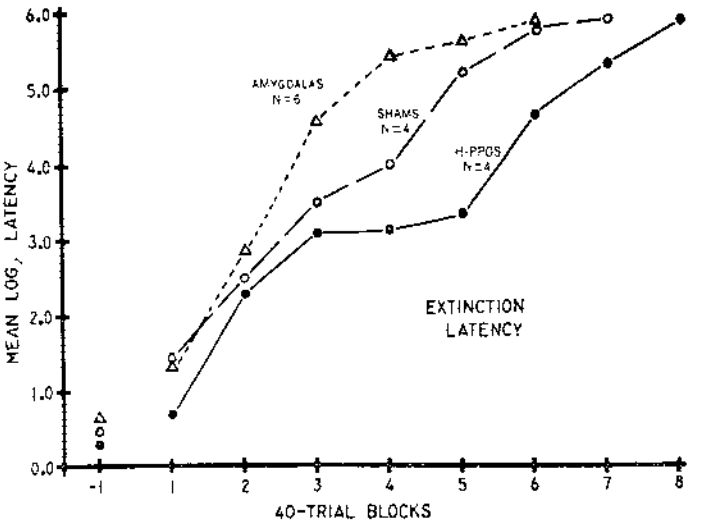

FIG. 4. Response latency during extinction.

($t=7.0$, $P<0.01$). The difference between the sham-operated and amygdalectomized groups was not found to be statistically significant, but further testing might substantiate the trend shown. These results are shown in Fig. 5.

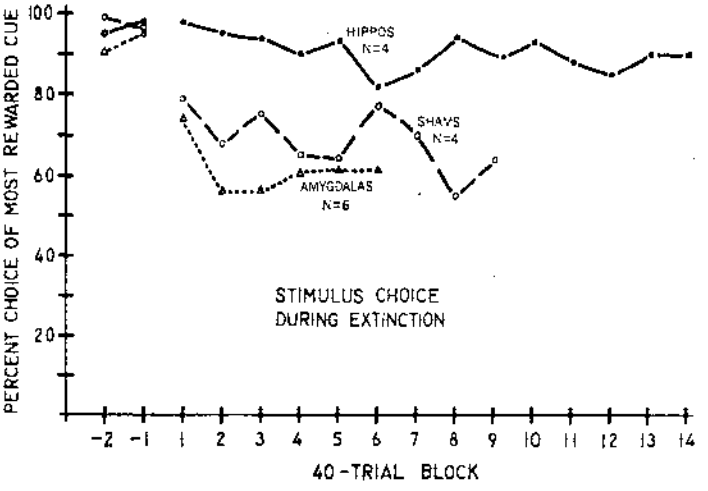

FIG. 5. Stimulus choice during extinction.

An unexpected difference was found between groups on the pattern of response latencies during the rewarded trials just prior to the start of the extinction procedure. While the groups did not differ significantly in mean latency during this period, they did differ in the frequency with which they made responses of longer than 2.0 sec. The typical response latency of these animals was either 1.0 or 1.5 sec, and observation revealed that a response of a longer latency was almost invariably accompanied by a definite hesitation, as if the monkey were undecided or being extra careful in his choice. If a hesitation response

is defined as one of longer than 2 sec, the rate at which these hesitation responses were found to occur in the different groups was: hippocampectomized Ss, 4.4 percent; sham-operated Ss, 12.5; and amygdalectomized Ss, 22.1. The probability that these groups could differ this greatly if all animals had been drawn from the same population is less than 0.01, as determined by the Kruskal–Wallis test ($H = 16.4$).

7.3. *Discussion*

Hippocampal lesions have been reported to retard extinction in the rat (ISAACSON *et al.* [13], NIKI [14], JARRARD *et al* [15]) and in the cat (PERETZ [16]). Thus, it is likely that the results of the present experiment are generally applicable to different species.

The finding that the amygdalectomized Ss were more prone to make long latency responses on the rewarded trials may be related to BATESON's (unpublished data) observation that amygdalectomized monkeys cock their ears more often during the training procedure than do normal monkeys, which show a much greater decrement in ear movements as training progresses. This result may also be related to the observation reported by SCHWARTZBAUM and PRIBRAM [10] that amygdalectomized Ss hesitate on test trials in a training progresses. This result may also be related to the observation reported by transportation task. All of these observations suggest that the reinforce-register process ordinarily functions by inhibiting undue orienting—which is borne out by the fact that the hippocampectomized Ss rarely made such long latency responses.

Although the amygdalectomized Ss extinguished faster than the sham-operated group on all measures of extinction, none of the differences were found to be statistically significant. The present authors suspect that the trend may be real, however, and that further testing with larger groups might result in the findings of significant differences. Faster than normal extinction has previously been reported in amygdalectomized monkeys, although on different tasks (WEISKRANTZ [17]). In the reversal tasks to be reported below the amygdalectomized Ss were also found to be faster in losing the originally trained response. These observations further support the model: undue orientation tends to disrupt the stability of the afferent inhibitory mechanism.

The choice of stimuli pressed during extinction differentiated the groups more clearly than did any other measure, with the results generally conforming to the expectations based on the model. However, the suggestion that the sham-operated and amygdalectomized Ss would fall to 50 percent or even below that in their rate of responding to the previously most rewarded stimulus was not entirely confirmed. When the results are plotted "backwards" from the point of complete extinction they reveal that the 50 percent level was never reached by the sham-operated group, although the amygdalectomy group did drop below that level on one block of trails. Both groups, however, increased responding to the previously most rewarded stimulus after this low point, and were up to about 65 percent just prior to extinction. Since no reward was involved and this final rate was found in both the sham-operated and amygdalectomized groups, it cannot be explained simply in terms of the reinforce-register process. A possibility is that the previously least rewarded stimulus by virtue of prior error-evaluation was being more intensely gated than the previously most rewarded stimulus. The model needs to be further specified, however, to encompass clearly this finding of differential gating. Such specification would assume that the modulation of recurrent inhibition is not just a quantitative process but is programmed. Evidence for such programming must be sought by further neurophysiological research.

8. EXPERIMENT IV: PAIRED vs. SCATTERED STIMULI

We mentioned earlier that presentation of stimuli was changed from random location to a condition in which the two stimuli were always paired spatially. The reason for the change was that when the first two hippocampectomized Ss were being tested for post-operative retention they did very poorly. An independent observer noted that these animals often had their faces very close to the panel, and when a stimulus appeared before their noses the other one was often out of sight. In these cases the hippocampal Ss appeared to press the visible stimulus, whichever it was. At this point in the experiment all Ss were immediately switched to a spatially paired stimulus presentation, a switch which benefited the hippocampectomized Ss greatly. On the basis of the model, the possibility of this occurring should have been suspected, since the hippocampectomized Ss would be expected to press the more impelling stimulus. If only one stimulus (lighted panel) is in sight they should, of course, press it and, in the absence of a normally operating error-evaluate system, continue to do this. This effect of the lesion was not formally tested, however, until after the extinction tests had been complete, since these had a higher priority for us.

8.1. *Method*

For this experiment a new tape was punched to govern stimulus presentation. With this tape, twenty consecutive stimulus presentations were allowed in which the two stimuli were either side by side, one above the other, or diagonal to each other. These pairs were presented only in the middle two rows of the panel, approximately at eye level. Following the twenty paired presentations were twenty trials in which the stimuli were presented at opposite sides of the panel, at the top and bottom, or diagonally. In both situations all possible locations were used within the limits described above. All Ss were then trained on a new 70–30 problem for either two hundred and forty trials or until a criterion of 36/40 was reached. Half of each group started with the paired stimuli and half with the scattered, with a switch being made after each block of twenty trials. The stimuli used were: hippocampal group, 9 vs. 2; sham-operated and amygdalectomized groups, A vs. E. Each S was given a score which indicated the difference in performance on the two types of stimulus presentation. This score consisted of the deviation from 50 percent correct on a given block of twenty paired stimulus trials minus the deviation from 50 percent on the preceding or following twenty scattered stimulus trials. The total score for each individual consisted of the mean difference for all blocks. This score was then converted to a measure of the average improvement per trial which was found when stimuli were paired, as compared to when they were scattered.

8.2. *Results*

It was found that the hippocampectomized Ss averaged 0.15 more correct responses per trial on the paired stimulus condition. This could amount to a large difference in the number of correct responses on a 40-trial block and is consistent with the earlier observation. The hippocampectomized Ss mean score was found to be significantly above the chance level of 0.0 ($t=2.5$, $P<0.05$). The mean scores of the amygdalectomized and sham-operated groups were 0.03 and 0.01 respectively, and neither group was affected by the change in stimulus location above a chance level.

8.3. *Discussion*

Since tasks of this general type are usually said to involve "discrimination" learning, the scores of the sham-operated and amygdalectomized Ss on this test are rather surprising.

If this test was really measuring the ability of monkeys to discriminate the difference between two stimuli, then this discrimination should have been more readily achieved when the stimuli were juxtaposed than when they were widely separated in space. The results suggest, rather, that the monkeys had no difficulty in differentiating one stimulus from the other, but they had to learn which of these stimuli to ignore. Ignoring, as we have seen, is not a simple process, however—ordinarily behavior is still guided by the ignored stimuli (as for instance during habitual performance). Only when the gating process is insufficiently modulated does ignoring become so overriding that only what is literally in front of the organism's nose determines his behavior. The performance of the hippocampectomized group is therefore in consonance with expectations based on the model.

However, other explanations can also be forwarded to account for these data. The present results, as well as all other data in this series of experiments, indicate that hippocampal lesions result in animals which have difficulty in withholding a response. The present results could be attributed to the fact that they were less able than the other animals to wait long enough to see both stimuli before responding. Although this possibility was not systematically pursued in this study, we have on other occasions found such an explanation wanting. The imposition of a transparent screen which allows the Ss sufficient time adequately to explore the stimulus panel visually, does not overcome discrimination difficulties when these seem to be due to "impulsive" behavior. Rather, we venture to suggest that the failures to withhold response, just as are position habits, are derived from impairments in other processes and are thus not primarily responsible for the observed aberrations in behavior.

9. EXPERIMENT V: REVERSAL

The model demands that animals with hippocampal lesions be slow to reverse responses in a discrimination situation when rewards are reversed. The model does however, predict that hippocampectomized Ss should eventually reverse as long as there is a finite probability that the reversal-response will be emitted. Reversal-responses will then accrue by the action of the reinforce-register system alone. This process could, however, especially on a 70–30 task, take more trials than the patience of the experimenter would allow. Through the use of gating however, the reversal would be greatly speeded up. On any reversal task, sham-operated and amygdalectomized Ss would be expected to outperform, by using their error-evaluating process, the hippocampally lesioned Ss, but this difference should be greatest when differential partial reinforcement is used. In order to test this hypothesis, all Ss were given reversal training after acquiring both a 70–30 and a 100–0 task.

9.1. *Method*

In the 70–30 reversal problem the same stimuli were used as in the preceding experiment, except that they were always presented paired. All Ss were trained to criterion, if they had not already reached it on the previous task, and were given an additional eighty "overtraining" trials. Immediately after these extra trials had been run, the reward schedule was reversed from what it had been during learning so that the formerly least rewarded stimulus was now rewarded 70 percent of the time, if pressed. The formerly most rewarded stimulus was now rewarded only 30 percent of the time. The switch was made during

a single testing day, so there was no time break between conditions. All Ss were trained until a criterion of 36/40 responses to the newly most rewarded stimulus had been achieved.

In the 100–0 reversal problem Ss were reversed after learning the 100–0 task reported in Experiment I. One day elapsed between the achievement of the learning criterion and the start of the reversal test.

9.2. *Results*

Reversal performance on the 70–30 task is shown in Fig. 6.

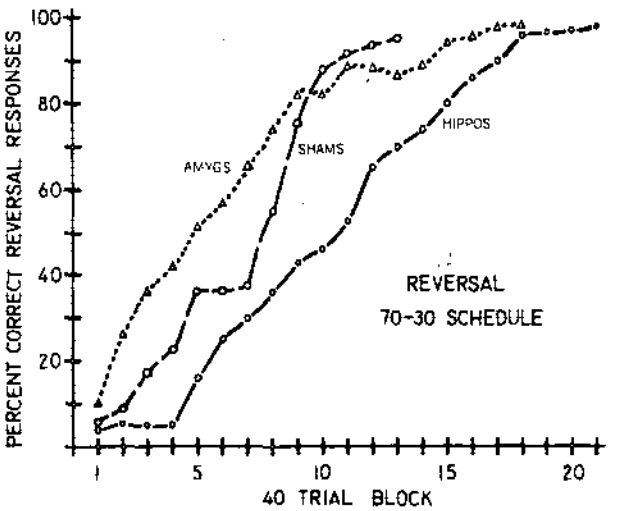

FIG. 6. Reversal with 70–30 schedule.

These curves demonstrate that reversal was initiated and complete in fewer trials in both the sham-operated and amygdalectomized groups than in the hippocampal. Because of the variability in performance among Ss, however, the result does not reach statistical significance on a measure of trials to a reversal criterion of 36/40 (90 percent). If, however, one does not insist that the reversal problem be completely learned, and uses a criterion of 28/40 (70 percent) then both the sham-operated and amygdalectomized groups reached this level in significantly fewer trials than did the hippocampectomized Ss ($t=2.1, P<0.05$) for hippocampals vs. shams; ($t=2.3$, $P<0.05$) for hippocampals vs. amygdalas. The amygdalectomized and sham-operated groups did not significantly differ from each other using this criterion.

Reversal on the 100–0 schedule can be seen in Fig. 1. In terms of trials to a 36/40 criterion, the hippocampectomized Ss required significantly more trials than the sham-operated Ss ($t=2.5$, $P<0.05$). The amygdalectomized group did not differ reliably from either group, mainly because of high variability.

9.3. *Discussion*

It is clear from the reversal curves presented here that the reversal process can be broken down into two stages, loss of the old response and acquisition of the new. The amygdalectomized group had no difficulty in the first stage, and may even be superior to the sham-operated group in eliminating the original response. Their chief difficulty was

in acquiring the new response with consistency. This explanation is consistent with the results of the extinction experiment. However, the inability of the amygdalectomized Ss to perform at a high rate on the 70–30 task is unexpected, since they did not show such a deficit in Experiment I.

Another result that had not been anticipated was the considerably greater rapidity of reversal shown by the hippocampal group than was predicted. As can be seen in Fig. 6, the curve is much steeper than one would expect on the basis of equal increments of change in impellance. Perhaps in this case impellance increments were not equal. This is supported by common sense: it is easy to imagine that a peanut acquired after a long period of fruitless panel-pushing could have much greater significance than a similar peanut delivered with recurrent regularity 8 sec after a previous one. It is thus the shape of the reversal curve upon which the statement is based that the size of the impellance increment produced by a reward of constant magnitude is directly proportional to its primacy in a sequence, the interval between rewards, and to the amount of work necessary to obtain the reward. Such a statement is certainly in accord with others that deal with the effects of reinforcement more descriptively. In the following experiment this idea was found necessary to an adequate analysis of the data.

When Figs. 1 and 6 are compared, the amygdalectomized Ss can be seen to be relatively worse in reversing on the 100–0 task than the 70–30, as compared to the other groups. This is probably related to the finding that they are slightly slower in acquiring a 100–0 task. Also the type of reward schedule makes much more difference to the reversal performance of the sham-operated than of the amygdalectomized Ss. A possible explanation for this is that the amygdala group learned both tasks exclusively by means of the error-evaluate system, i.e. by gating out the negative stimulus. In that case, reversal would involve the removal of this "gate" as well as the building of a new gate, so that the formerly positive stimulus could now be ignored. The slightly faster reversal on the 100–0 problem could then be explained in terms of the greater orienting, i.e. ungating, produced by the marked disparity between the reversal and initial conditions. The sham-operated Ss on the other hand, needed only to "ungate" in the 70–30 problem and not in the 100–0, as the error-evaluate process was not necessary for the solution of that problem, or at least was used to a lesser degree. Thus, the sham-operated Ss are assumed not to have required the extra step of "ungating" in the 100–0 reversal problem, and so that they could proceed directly to gating out the new negative stimulus. If this explanation were true, then it could also be used to account for other results in the literature such as the "overlearning reversal effect" (see THEIOS and BLOSSER [18]). If overlearning should be found to result in the relaxation (i.e. progressive modulation) of gating processes, then overlearning should produce faster reversal by eliminating the extra step of ungating. Of course, this effect would not be found in tasks which require continued gating nor in tasks which do not involve the error-evaluate process at all, which might explain the puzzling fact that the overlearning reversal effect is not always found.

In summary, hippocampectomized Ss were found to be generally poor in reversal performance, both in dropping the earlier learned response and in acquiring the new. The amygdalectomized Ss were equal or superior to the sham-operated group in losing the earlier response, but were defective in achieving a high level of performance to the reversed stimulus. Deficits in reversal have also been reported in hippocampectomized rats (KIMBLE and KIMBLE [19], and in cats (TEITELBAUM [20]), lending credence to the view that these effects are generally applicable across species.

10. EXPERIMENT VI: MASSED vs. DISTRIBUTED TRIALS

This experiment involved a test of one of the statements discussed in the previous section, that the impellance increment due to a reward is proportional to the time interval between trials. If this were true, then it could account for the often observed fact that it takes fewer trials for an animal to learn when trials are spaced than when they are massed. If the spaced vs. massed trials effect is due to the impellance-time factor, then it necessarily follows that this effect would be observed to occur only in animals possessing intact reinforcement-register processes. Of the present three groups, only the amygdalectomized Ss would be expected to fail to show an improvement with longer intertrial intervals. In fact, these animals would be expected to do worse at the longer interval, if one takes into account the possibility of decaying memory traces, interference effects, etc.

It has already been shown in this laboratory that hippocampectomized and normal monkeys show a steady and equal improvement in performance as the intertrial interval is lengthened up to 6 min (KIMBLE and PRIBRAM [9]). Since that study involved the same surgeon and testing apparatus as the present one, and since the results were so unequivocal, the present hippocampal and sham-operated groups were not run in the present experiment. The amygdalectomized Ss trained on two different tasks using a long and a short intertrial interval, with the expectation that they would learn quicker when given the short interval.

10.1. *Method*

Half of the Ss in the amygdalectomized group were trained on a new 70–30 problem using an intertrial interval of 5 sec, while the other half were trained to the same cues (X vs. N), but with an intertrial interval of 20 sec. Due to time and scheduling problems, all Ss were trained for only three hundred and twenty trials, with training being terminated at that point whether a criterion of 36/40 had been reached or not. Following this, all Ss were trained on a new 70–30 problem (green cross vs. blue square), with each S having the opposite intertrial interval to that on the first problem. As far as possible, the two half-groups were equated for learning ability, as had been demonstrated on the previous tests.

10.2. *Results*

The performance of the amygdalectomized Ss was worse at every stage of the learning process at the longer interval, as compared to the shorter. When performance is broken down by 20-trial blocks, performance on the 20-sec interval is inferior at every last point. A binomial test revealed that this difference was unlikely to be a chance occurrence ($P<0.01$). In terms of trials to a criterion of over 70 percent correct in a block of forty trials (some animals did not reach 90 percent) learning was significantly slower at the longer interval ($t=2.5$, $P<0.05$).

10.3. *Discussion*

We can think of no other explanation for the present results than the one used here: that the amygdala mediates impellance increases, and that the massed vs. spaced trials effect is due to larger impellance increments at the longer intervals. If these results are attributed to poor memory or to a lack of "recent memory", then it is difficult to explain why the amygdalectomized Ss were relatively worse in learning the 100–0 problem than the 70–30, as compared to the sham-operated Ss, since the flow of information is slower in the latter task. If it is assumed that the amygdala deficit on the 100–0 task is due to an inability to process information at a fast rate, then one would expect these animals to perform better when a longer intertrial interval is used.

Additional evidence implicating a deficit in the reinforce-register process in amygdalectomized monkeys is provided by the work of SCHWARTZBAUM [21]. In that study he found that the amygdalectomized Ss were much less responsive to the differences in the magnitude of the reward than were normal monkeys. This result can be applied to the model more directly. The model clearly predicts that amygdalectomized monkeys should have extreme difficulty in learning a discrimination task when both stimuli are rewarded equally often, but with rewards of different magnitude. Hippocampectomized and normal Ss should have much less difficulty in mastering such a problem. This possibility remains to be tested however, but it is another instance of the model's propensity for making statements which can be disproven, a feature which is of no small importance.

11. GENERAL DISCUSSION

11.1. *The data*

While many faults can be found with the procedures used in some of these tests, no individual experiment is claimed to represent the final answer to any of the questions raised by the model. Some of the results may have been influenced by the specific order in which the various tests were given or by the slightly different histories of the different groups, or perhaps the present groups are really biased samples of the population they are presumed to represent. It is difficult to believe, however, that these factors could result in groups which behave so consistently in the ways specified by the model. The possibility does exist however, that even though the groups as a whole behaved according to the model, the mean differences were occasioned in one task by one S and in another by another. For example, the hippocampal Ss which were most deficient in extinction may not have been those most prone to choose the least-rewarded stimulus on the novelty test, etc. This possibility was tested by means of a measure of group homogeneity. The homogeneity score of an individual consisted of the percent of the time that its score was closer to the mean of its own group than to the mean of the group to which it was compared. The hippocampectomized Ss were compared to the sham-operated monkeys on the nine measures which should have best discriminated these groups, hippocampectomized and amygdalectomized Ss were compared on ten measures, and amygdalectomized and sham-operated Ss on eight different measures. A score of 100 percent indicated that an individual always acted like a member of its group, and a score of 0 percent, of course, meant that the S actually behaved as if it were a member of the opposite group. Fortunately, no zeros were found. The results of this homogeneity test indicated that the groups were indeed composed of very similar individuals. The scores for the hippocampal and sham-operated Ss were 83.3 and 88.9 percent homogeneity, respectively. When hippocampal Ss were compared to the amygdalectomized group the scores were 80.0 and 78.3. The homogeneity of the amygdalectomized Ss when tested against the sham-operated animals was 68.8 percent, with 78.1 for the shams. The individuals within a given group had a remarkably small range in scores except for one of the hippocampectomized and one of the amygdalectomized monkeys. These two Ss appreciably lowered their group means and behaved much like the shams. These results suggest that two animals may not have as extensive a lesion as the other members of their group. This possibility has been checked in the case of the odd hippocampal monkey. This animal recently died from a respiratory ailment, and the brain had been prepared for histological examination. While the hippocampal formation was almost entirely absent on one side of the brain, the opposite hippocampus was found to be only slightly damaged. It had been noted during the operation

that the placement of this lesion was not certain, as the others had been. The deviant amygdalectomized S remains to be investigated, but the results above indicate that the homogeneity test may be an accurate indicator of the extent of the lesion.

The homogeneity scores discussed above can also be converted to yield a measure of overall group differences. In order to compare the difference between any two groups one has only to reverse (or take the complement of) the scores of Ss in one of the groups. This results in a measure of the degree to which the second group was like the first one. The results of these tests were: hippocampals vs. shams, $t=6.3$, $P<0.001$; hippocampals vs. amygdalas, $t=5.6$, $P<0.001$; and amygdalas vs. shams, $t=6.2$, $P<0.001$. Thus, even though the differences between groups on any one test were not always as pronounced as one might have hoped for, the overall group differences were great. The hippocampals and amygdalas differed from each other just about to the same degree that either one differed from the shams. This suggests that it is an oversimplification to consider the limbic system only in terms of some unitary behavioral process.

11.2. *The model*

Even though the results reported here are generally in confirmation of expectations based on the model, we feel that they constitute only preliminary proof of the model. Obviously, the original contention that problem solving is guided by two processes, one of which increments and the other decrements awareness, was never directly tested. If these two processes are unnamed but thought of as direct determinants of the probability of a response, the same results would have been anticipated. Nonetheless, we feel that the present form of the model has many advantages. For example, it is possible that impellance may prove to be directly assessable, as for instance in terms of pupil dilation (see HESS [22]), and that gating effects may be observable in terms of pupil constriction. Tests at the neurophysiological level may also prove to be useful in this respect. In addition, psychophysiological measures and measurements of eye movements during problem solving may eventually help to determine whether a pattern of "observing responses" can be found to support the model.

It is no accident that the model is contradicted by very few of the many limbic lesion studies which have appeared in the literature, as it was formulated with most of these reports in mind. It is, of course, impossible for any model to explain all of the extant data, as the data are often conflicting. The ability of the model to encompass many diverse findings is not due to vagueness or lack of specificity in the model, as the model generates many clear-cut predictions which could easily be disconfirmed, if untrue. Whether a given finding supports or disconfirms the model depends, however, on a careful analysis of the factors involved in performance of the task which was used. The expected and obtained results of experiments using hippocampectomized and amygdalectomized rats in active avoidance tasks can serve as an example.

The instrument most commonly used in tests of active avoidance is the double grill or shuttle box, which basically consists of two adjoining compartments with grid floors which can be individually electrified. The S is trained to run to the opposite compartment upon presentation of a signal (usually a buzzer) in order to avoid shock. If the S is trained to first run to one compartment on the first trial, and then back to the original one on the following trial, the device is termed a two-way shuttle box. If the S is always replaced in the original compartment, this is called a one-way shuttle box task. Predictions from the model suggest that correct performance in the two-way shuttle box involves mainly the

reinforce-register process, while in the one-way task both systems may contribute. If this analysis is correct, then normal animals would learn the one-way task faster than the two-way as has been shown by HORVATH [23]. Further, hippocampectomized animals should have no deficit in the two-way task, as compared to normals, but should be slightly worse on the one-way problem. These results have been found by ISAACSON *et al.* [13] and NIKI [14]. In addition, amygdalectomized animals should be deficient on both problems, with a relatively greater defect on the two-way problem. These results were obtained in HORVATH's [23] study. Unfortunately, there are some conflicting data on the effects of hippocampectomy on active avoidance behavior in different species*.

11.3. *Other explanations*

Finally, a word about the relationship between this model and other explanations that have been forwarded to account for the effects of limbic lesions. The model proposed here takes as its point of departure the concept "experience" and then delineates how experience may be experienced in awareness, registered in memory, and gated out of awareness. The assumed effect of these processes on behavior is then spelled out and the assumptions tested. Other points of departure are of course possible and equally useful. One can begin with observed behavior and make inferences from these observations about processes. There have been a considerable number of such efforts which include some of our own. The assumed effects of limbic lesions on response perseveration (KIMBLE and KIMBLE [19]), response inhibition (NIKI [24], MCCLEARY [25]), response programming (planning) (MILLER *et al.* [26]) are examples of this sort of theorizing. Such models and the one presented here are considered to be complimentary to each other, as would be any stimulus derivative vs. response derivative models (since stimulus and response mutually imply each other). However, we feel strongly that the validity of such complimentary models depends on the degree to which they converge on processes at the neuronal level which, if not identical, are at least compatible.

The relationship between the model proposed here and explanations of the effects of limbic lesions in terms of such global conceptions as "memory" and "emotion" or even such somewhat more restricted concepts as "drive" is a different one. The connection between the observed effects of the lesions and *these* explanations is a felt, tacit connection. The models, on the other hand, externalize in explicit, testable fashion at least some of the tacit knowledge gained through observation, experiment and perusal of relevant reports. The relationship between the models and the more global explanations would perhaps be most effectively served by using the models to model the more global conceptions and then spot convergences and divergences. The locations of divergence from the model-model would then indicate processes important to the conception which are not subsumed in the model-model. Thus the specific contribution of the limbic systems to memory, emotion and drive would be ascertained and the logical error of localizing such "functions" in a part of the brain avoided. Further, and perhaps even more important, such model-modelling would provide some explicit communication about these fascinating topics.

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* ISAACSON *et al.* [13] found hippocampectomized rats to be clearly superior to control animals in the two-way shuttle-box, and have twice replicated these findings. In contrast, PRIBRAM and WEISKRANTZ [5] found that their hippocampectomized monkeys required 3 times as many shocks to learn a similar task. The reason for the discrepancy may lie in the slight procedural differences which did exist. In the latter case animals were trained before the operation.

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Résumé—On présente un modèle identifiant l'amygdale et l'hippocampe avec deux processus distincts de direction de l'attention. Le processus auquel contribue l'amygdale est postulé augmenter à la conscience des expériences comme une fonction du renforcement antérieur tandis que la processus auquel contribue l'hippocampe agit en diminuant la conscience de l'expérience comme une fonction de la probabilité du non-renforcement. On esquisse la façon dont ces processus gouvernent le comportement de résolutions des problèmes et on formule un certain nombre d'hypothèses. Ces hypothèses sont testées chez des singes avec lésions hippocampiques et amygdaliennes. Les résultats de ces expériences s'accordent en général avec le modèle et les deux groupes de lésions apparaissent comme aussi distincts l'un de l'autre que chaque groupe d'un groupe de contrôle formé de sujets avec opération fictive.

Zusammenfassung—Ein Modell wird gezeigt, welches den Mandelkern und Hippocampus mit zwei, deutlich die Aufmerksamkeit lenkenden Prozessen, identificiert. Der Prozess, zu welchem der Mandelkern beiträgt, ist erforderlich, um die Wahrnehmung von Erfahrungen als eine Function vorausgegangener Verstärkung zu erhöhen, während der Prozess, zu welchem der Hippocampus beiträgt, zur Verringerung des Wahrnehmens von Erfahrung, als eine Function der Wahrscheinlichkeit von Nicht-Verstärkung dient. Die Art, in welcher diese Prozesse Problem-lösendes Benehmen beherrschen, wird beschrieben und eine Anzahl von Hypothesen geschaffen. Diese Hypothesen wurden an Affen mit Hippocampus- und Mandelkern-Schädigung geprüft. Die Ergebnisse dieser Experimente waren allgemein in Übereinstimmung mit dem Modell, und die beiden Läsions-Gruppen wurden ebenso verschieden von einander befunden wie jede Einzelgruppe verschieden war von schein-behandelten Kontroll-Tieren.