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THE NATURE OF NONLIMBIC LEARNING[1]

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Naive monkeys with combined hippocampal-amygdaloid lesions and a group of unoperated controls were trained postoperatively on a visual discrimination problem, tested for retention after 3 wk., and trained on a reversal task. The subjects with lesions were profoundly deficient on all but the retention test. The difference between groups in the original learning task was entirely due, however, to the fact that monkeys with lesions were very slow to begin learning. Learning curves between 60% and 90% were identical. On the reversal task the monkeys with lesions, unlike the control monkeys, generated a long plateau around the 50% level of performance. Comparison with similar results on human retardates suggests that ordinarily these limbic structures function to maintain an hypothesis in the face of distraction or disconfirmation.

In a previously published model of limbic system function (Douglas & Pribram, 1966), the amygdala was postulated to play a key role in a system in which attention was locked on to a stimulus as a function of reinforcement, whether positive or negative. The hippocampus, in contrast, was suggested to be crucially involved in a mechanism in which attention was directed away from a once salient stimulus as a function of nonreinforcement. These ideas have had some success in accounting for a wide variety of experimental results (e.g., Douglas, 1967; Douglas & Pribram, 1969; Pribram, 1969).

This success has continually tempted others, as well as occasionally ourselves, to consider the amygdala-hippocampus system as sole repositors of the learning process. To dramatize the absurdity of such a position we initiated the present experiments to test the learning capacities of monkeys deprived of both amygdala and hippocampus.

More important, such experiments would allow us to ask whether one aspect of the learning mechanism was dependent on limbic, another on isocortical function. We were interested, for instance, to find out whether discontinuities described by multiple stage models of discrimination learning curves (Blehert, 1966; Zeaman & House, 1963) would, in limbic-lesioned monkeys, turn into more continuously incremental slopes.

Several studies had already been done in which the behavior of subjects with the combined lesion had been investigated (e.g., Correll & Scoville, 1965; Mishkin & Pribram, 1954; Orbach, Milner, & Rasmussen, 1960). It was obvious that such extensive damage produced learning deficits, and equally obvious that it did not prevent eventual learning. The object of the present study was not, however, merely to demonstrate once again that animals with large brain lesions are stupid. Instead, the present experiment was designed so that the learning process could be analyzed in detail. The question: What are the characteristics of nonlimbic learning?

In order to answer this question it was deemed desirable to investigate the learning process in animals which had not been formally trained while possessing intact limbic structures. This would, as much as possible, eliminate the probability that abilities or knowledge acquired with the help of limbic structures might be carried over after the lesion had been made. The present subjects were newly captured rhesus monkeys, and lesions were made prior to any formal training. Several weeks after the operation, monkeys were first trained

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on a discrimination problem involving class-subclass distinctions (Dewson, 1967). That is, there were two major classes of stimuli presented: lit and unlit panels. The unlit were "irrelevant," while among the two lit panels was one which was rewarded and another which was not. Following acquisition, subjects were tested for retention after a 3-wk. interval, trained on a new discrimination problem, and finally given a reversal task.

Method

Subjects and Lesions

A total of 11 rhesus monkeys weighing 2–2.5 kg. served as subjects. All were experimentally naive, and all were housed individually with free access to water. Four monkeys were in the lesion group while seven served as normal unoperated controls. Three of the latter were used in a different experiment following the original learning and retention tests, and so the reversal task involved only four normal subjects.

Four animals were subjected to bilateral suction removal of amygdala and hippocampus through an opening made in the medial aspect of the ventral surface of the temporal lobe. The details of the surgical procedure are similar to those reported by Douglas and Pribram (1966). The monkeys with the surgical resection will be referred to as the "hip-am" group. The remaining subjects were surgically untouched, as extensive research in this laboratory has revealed that "sham" lesions produce no detectable effect.

Since the brain-operated monkeys are scheduled for an extensive series of tests over the next 2 yr., there is no histological verification of the lesions at this time.

Apparatus

All testing was done with the use of a computerized version of the DADTA apparatus described in Pribram, Gardner, Pressman, and Bagshaw (1962). This consists of a small enclosure in which the subject is effectively sealed off from the external environment. A 4 × 4 array of 16 depressible panels is placed against one of the walls, with a food cup located at bottom center of the array. A one-way viewing glass makes up most of another wall. Various stimuli are projected onto the panels from the rear, and appear as white patterns on a dark background. Stimulus location is varied in an unsystematic fashion so that the same stimulus rarely appears on the same panel twice in a row, and it appears on all panels over any extended number of trials. Stimulus presentation, recording of responses, and delivery of food rewards to the food cup are carried out automatically by a specially programmed PDP-8 computer. Rewards were 190-mg. Noyes banana pellets.

Procedures

Beginning 3 wk. after surgery, the following training sequence was administered to all animals: Shaping, pretraining, first discrimination problem, 3-wk. rest, retention test, 2-wk. rest, second discrimination problem, and finally, the reversal problem.

Subjects were deprived only during shaping, during which monkeys were accustomed to banana pellets, but the food ration was then gradually built up to normal by the beginning of the first discrimination problem. All feeding, other than rewards, took place after completion of daily testing. This procedure has been found to produce high motivation without weakening the subject.

Shaping, using the technique of successive approximation, was carried out under manual control in 20-min. sessions. The shaping cue was the numeral "1" displayed simultaneously on 12 of the panels (the other 4 were unlit). Shaping was terminated when the monkey had made about a dozen short-latency responses to any of the panels. This was followed by five pretraining sessions, given one per day, each of which terminated when the subject had received 50 pellets. During such a pretraining session a press of an unlit panel produced no visible effect but was recorded by the DADTA apparatus. A press of a panel containing a "1" resulted in delivery of one pellet and the onset of a new trial marked by the resetting of the machine. The reset consisted of offset of all stimuli for 3 sec., followed by reappearance at randomly determined locations. On all later problems the offset time (intertrial interval) was 5 sec. After shaping and pretraining the first discrimination task was begun.

On the first discrimination problem two lit stimuli were used, the numerals "2" and "4," with the remaining 14 panels unlit. On any trial a press of an unlit panel was unrewarded and did not reset the display. A press of the numeral "4" was also unrewarded, but resulted in resetting of the display, with the stimuli disappearing for 5-sec. and reappearing at different locations. A press of stimulus "2" resulted in the delivery of one banana pellet and a reset of the display as above. Daily sessions lasted until the subject had made 50 total presses of either of the lit panels. Training continued with daily sessions until a criterion of 90 correct responses had been made in a total of 100 presses of lit panels. At this point each monkey received another 100 overtraining trials and was then given 3 wk. off from testing.

After the rest, all monkeys were given a retention test consisting of 100 trials using the same procedure as in learning. The test was given in two sessions and followed by a 2-wk. rest.

After the second rest, training began on a new discrimination problem using the same procedures as on the first. This time the numeral "0" was rewarded or positive, while "5" was unrewarded or negative. No overtraining trials were given, however. Instead, as soon as a 90/100 criterion was reached each animal was started on a reversal

problem. The reversal task began on the day following attainment of criterion and involved similar procedures except that "5" was now rewarded while "0" was unrewarded.

Results

The combined hippocampal-amygdala lesion produced some of the classic Klüver-Bucy (1939) effects. The monkeys with lesions were very tame, in marked contrast to the savage normals, although they could display apparent fear and anger when thoroughly provoked. The taming and other dramatic effects were most likely due to the inclusion of the amygdala in the lesion (Mishkin & Pribram, 1954; Pribram & Bagshaw, 1953).

Shaping and Pretraining

All monkeys with lesions shaped within the first session, while all normals required between two and three sessions. The difference was probably entirely due to the tendency of the normal subjects to huddle at the rear of the cage and thus emit few rewardable responses.

On the five pretraining sessions the normal group averaged 78.8% correct responses (presses of "1") while the hip-am group had a mean of 73.9. Neither figure deviates significantly from a nominal chance rate of 75%. There was no sign that either group was improving in efficiency, as these conditions apparently provide little incentive for eliminating the infrequent unrewarded responses.

First Discrimination Problem

The gross results of the first discrimination problem can be seen in Table 1. As expected, the group with lesions had a profound learning deficit. In reaching criterion performance they required over twice as many responses to the rewarded stimulus as did the controls, made more than 3½ times as many responses to the unrewarded lit panel, and roughly five times as many presses of unlit panels. All differences were highly reliable statistically (see Table 1).

A closer analysis of the data revealed, however, that removal of these two limbic structures did not merely slow up learning "in general." Instead, the hip-am monkeys

TABLE 1

Responses to Criterion in Normal and "Hippocampal-Amygdaloid" Monkeys

Stimulus	Group		t	p
	Normal	Lesion		
Positive (2)	258.2	529.75	3.1	.02
Negative (4)	109.8	380.25	5.3	.01
Unlit panels	250.0	1,208.50	3.9	.01
Total	632.0	2,118.50	4.3	.01

appeared to go for very long periods with no improvement in success. This would then be followed by a period of very rapid improvement. In order to demonstrate this effect, two aspects of the present problem will be considered separately. Previous research using similar procedures indicated that there are two stages in the solution of this problem (see Dewson, 1967). First, the subject learns to press lit, rather than unlit, panels. Secondly, it differentiates between the positive and negative lit panels or stimuli. Both processes were investigated using the method of successive criteria. Figure 1 shows the number of trials required for successive 5% reductions in unlit panel pressing. It can be seen that most of the difference between groups is prior to the 70% level, and that after this point the curves for the two groups are nearly equal in slope. This is illustrated by the dotted line, which is the normal curve superimposed over that of the monkeys with lesions. The hip-am group made a mean of 1,240 total presses before falling below 70%, while the normal group averaged a reliably lower 164 ($t = 4.6, p < .01$). The drop from 70% to 10% in the hip-am group required only another 485 trials, however, and this is not significantly different from the normal group mean of 340 ($t < 1.0$). Thus, the difference between groups was almost entirely due to a sluggishness in the initiation of learning in the hip-am monkeys.

A similar analysis was made of the course of differentiation between lit panels, disregarding presses of unlit panels, and the results can be seen in Figure 2. Once again the difference between groups was mainly in the initial stages. After a mean of 84 presses of lit stimuli the normals reached

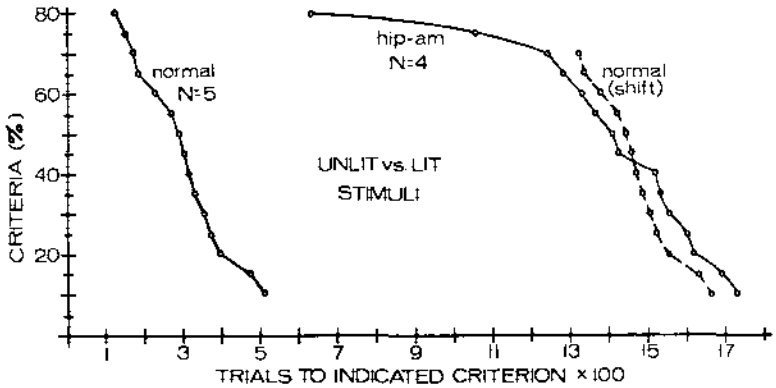

FIG. 1. Reduction of presses of unlit panels. (Y axis shows criteria decreasing by 5% intervals, and X axis shows mean trials to each criterion. Dotted line is the curve for normal animals superimposed on lesion-group curve.)

the 60% criterion point, but the hip-am monkeys had a reliably higher mean of 690 ($t = 5.3$, $p < .01$). Between 60% and 90%, however, the animals with lesions actually had a lower mean (220) than did the normal animals (284), although the difference did not approach reliability ($t < 1.0$). Thus, both analyses revealed the same thing. Hippocampal-amygdaloid lesions appear to retard the commencement of learning, but they have little or no effect on the progress of learning once the learning is underway.

Another difference between the groups was that the normal animals appeared to begin to differentiate between lit stimuli

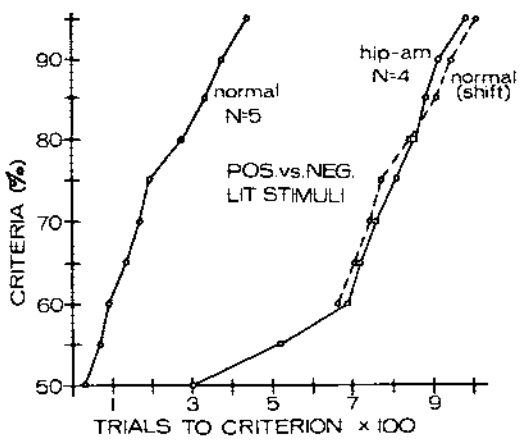

FIG. 2. Differentiation between positive and negative lit stimuli. (Y axis shows 5% criteria points, and X axis the mean trials to each criterion. Dotted line is the curve for normal animals superimposed on lesion group curve.)

even before they had finished reducing their unlit panel pressing. For example, during the period in which these irrelevant responses were being reduced from 60% to 10%, the normal monkeys were pressing the positive stimulus almost twice as often as the negative lit stimulus. During this period in the hip-am group the ratio of positive to negative lit panel presses was 1.1. The difference is reliable ($t = 3.4$, $p < .02$). A related finding was that the groups differed in the maximal number of responses to the negative lit stimulus during any 100-trial block (including all types of responses). The mean peak for the hip-am group was 45.5 such responses, while in the normal group the average peak was reliably lower (30.0, $t = 5.1$, $p < .01$). Both results suggest that the normal monkeys learned both cue categorizations in an overlapping fashion (or in very rapid succession), while those with lesions, in contrast, began the differentiation between lit panels only after unlit panel pressing was reduced to a secure and very low level.

Retention

After the 3-wk. layoff the hip-am subjects were found to have retained the problem to a remarkable degree. This group averaged 99.25% correct responses on the retention test, while the controls had a mean of 95.4. Most of this difference came on the first 20 trials, where the normals fell below 90%. Thus, there can be little doubt that monkeys with hippocampal-amygdaloid lesions

retain what they learn. This does not contradict the finding of recent memory loss in humans with essentially the same lesion (e.g., Scoville & Milner, 1957), because human patients in that study were not trained to perfection on a task. Also, the verbal element of the task as presented to humans makes the procedure considerably different (Rosenberger, Mohr, Stoddard, & Sidman, 1968; Stepien & Sierpinski, 1964).

Second Discrimination Problem

The second problem was given both as a test for learning set and as a preliminary for reversal training. Since the hip-am monkeys did not begin to press unlit panels once again, they obviously carried over some of their experience from one problem to the next. It was unfortunately impossible, however, to evaluate speed of learning on the second problem. The reason was that two hip-am subjects began the second problem with a bias for the positive stimulus (all others began at about 50%-success rates). Their behavior, however, tended to confirm the conclusions of the first study. The two biased monkeys (65–70% on first 100-trial block) behaved as if they had begun at that given level on a learning curve, and they had a mean of 120 trials to criterion. The other two monkeys behaved much as they had on the first problem, with some floundering following by rapid improvement in success, and they averaged 620 trials to criterion. Since even these animals were faster to learn than the mean for this group on the first problem, some learning-to-learn must have occurred, but it is impossible to say how much. The normal subjects required only 145 trials to criterion of the second problem, as compared with 368 on the first. These figures do not, of course, include presses of unlit panels. The difference between groups on the problem did not reach significance, but this was undoubtedly an accident due to the bias, as the latter produced extreme variance.

Reversal Task

Since both amygdaloid and hippocampal lesions have been found to result in reversal deficits (e.g., Douglas & Pribram, 1966) it was expected that the combined lesion would do as much. Expectations were borne out, as the hip-am group averaged 790 trials to reversal criterion while the normals had a mean of only 175. There was no overlap, and this difference (with the small n) is significant at the 5% level (rank-sums test). Figure 3 shows the total number of trials required to achieve successively higher criteria. It can be seen that the monkeys with lesions were deficient at all stages from first to last. They took somewhat longer than the normals to rise (or fall) to 50%, but the curve breaks sharply at that point. There is

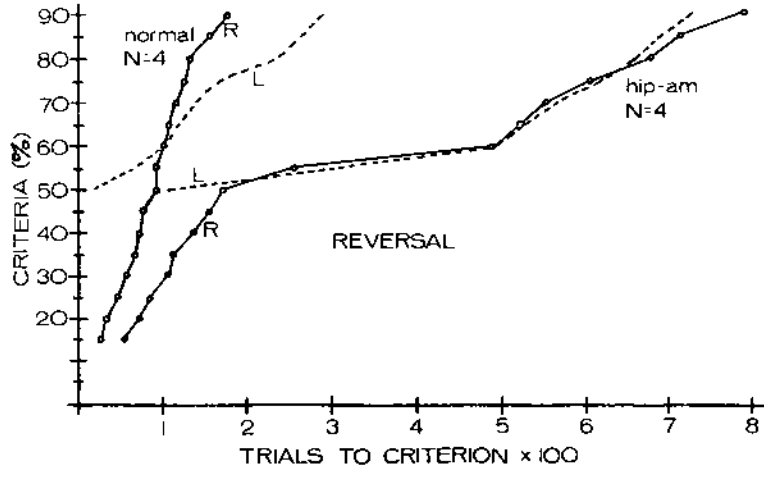

FIG. 3. Successive criterion curves for reversal training (R) with lit-panel differentiation curves from first problem indicated by dotted lines (L).

no corresponding break in the normals' curve at the 50% level. The normals' curve has an almost constant slope from 20% up to 90%, with a slight acceleration near the 50% mark. This suggests that the actual learning may well have taken place at a point where success was very infrequent (20% or even lower). In the hip-am group, however, the course of learning between the 50% and 90% levels bears a startling resemblance to original learning in the first problem. The original curve is shown in Figure 3 as a dotted line superimposed over the reversal curve. Thus, reversal learning in the hip-am group appears to consist of three distinct stages which suggest *qualitative* differences between the normal and the lesion groups.

Discussion

As indicated in the Introduction, we were especially interested in investigating whether the multiple-stage learning-curve discontinuity would be altered by resection of the limbic mechanisms. Clearly, the answer is contrary to our expectations that such a change in the direction of greater continuity would be produced. The slope of the discrimination learning curves was largely unaltered by limbic lesions. The discrimination aspects of discrimination learning (in retrospect, and not surprisingly) are apparently a function of isocortex.

Limbic lesions did markedly slow learning, nonetheless. The impairment seems to be localized to those parts of the learning curve which are relatively flat or stationary, before the inflection or take-off point occurs. The same sort of a "hang up" is shown during reversal performance. Normal monkeys quickly change from making most of their responses to the previously rewarded cue to making most responses to the presently rewarded cue without any inflection in their performance curve. Monkeys with limbic lesions, by contrast, show a long plateau after their performance reaches a chance 50% level. Despite this, the slopes of the curves on either side of this plateau are comparable to those in original learning. It is difficult to explain these results in terms of an inability to discriminate between stimuli.

One way of considering the plateau is to suggest that the situation does not furnish sufficient incentive to alter the behavior of monkeys with lesions. This would conform to ideas that the limbic system is a substrate of motivation. However, no simple reference to the stimulus situation per se will provide a sufficient explanation. The cues remain the same; the overall probabilities of their being reinforced have not changed. What has changed during reversal is the short-term probability that the monkey's response will be reinforced. Apparently, when the probability reaches 50%, a disposition or state is induced in the hip-am monkeys, but not the normals, which leads to a continuation of this relatively ineffective performance. The question is how best to characterize this state.

The data and theories of Zeaman and House (1963) would appear to be relevant to present findings, and to offer a possible answer. Through the use of backwards learning curves these investigators have found results much like ours in human retardate learning. That is, discrimination learning appears to involve a stationary period of varying length in which the subject is correct at only a chance probability. This is followed by an abruptly rising curve of relatively constant slope from one person or problem to the next. Their data, and our findings with many normal monkeys, indicate that the difference between a difficult and an easy problem or a fast and a slow learner is in the length of the stationary period prior to the take-off point. Furthermore, a stationary period is often found at the 50% point in reversal learning in human retardates. Zeaman and House (1963) suggest that the stationary period in a discrimination-learning problem is one in which the subject learns an observing response or, in other words, learns which aspects of the compound stimuli to attend to.

A long flat period might then indicate that the instrumental and observing responses have come under the control of different aspects of the reinforcement schedule. The stationary period during reversal training thus represents an extinction of the previously appropriate observing responses,

while the instrumental responses are maintained by the 50% schedule. Apparently, the animal or retarded human is unable to keep his attention fixed on the relevant stimuli long enough to switch positive and negative signs. Instead, he gives up observing (but not responding) and later learns as if a new problem had been presented. It is in this sense perhaps that the limbic system can be said to play a role in incentive motivation.

The results can also be interpreted in "hypothesis formation" terms if observing responses are taken as indicators of hypothesis testing. When an organism's observing of a distinctive feature or stimulus dimension is reinforced, an hypothesis (attentive state) may be induced which increases the likelihood that this feature will be observed again. This hypothesis will be either confirmed or disconfirmed on subsequent trials. As already noted, two separate factors seem to be responsible for learning in the discrimination situation. One is likely to be related to the stimulus dimensions per se and, thus, probabilistically will distribute observing among dimensions. With no other mechanism than this, any subject could learn any problem in which the correct stimulus dimension has a finite probability of being observed. With low probability stimulus dimensions, however, this learning could be extremely protracted.

The second factor is likely to be related to the duration over which any hypothesis is held in the face of disconfirmation. It is this factor (a criterion for relinquishing the hypothesis) which is most likely to be regulated by limbic structures. The hippocampus could thus be conceived to be involved in setting the criterion for dropping unsuccessful hypotheses (as a function of errors), while the amygdala sets the criterion for keeping a likely hypothesis from slipping away through distraction or being replaced by others—possibly as a function of the first few rewards subsequent to hypothesis adoption. Both systems would ordinarily act to increase greatly the probability of actively observing the relevant stimulus dimension for enough trials so that the correct hypothesis becomes established. With respect to the reversal plateau, this explanation reads as follows: With the same stimulus characteristics relevant all during reversal training, the hip-am monkeys had obviously learned that "0" was crucially different from "5," yet they could not maintain this as an active hypothesis in the face of a 50-50 schedule of reinforcement.

This study and earlier ones thus emphasize the fact that the distribution of attention is determined by at least two factors. One is directly related to stimulus dimensions; the other to the temporal organization of the learning situation. Only the second of these, the duration over which an hypothesis is held in the face of distraction and disconfirmation, is critically affected by amygdalectomy and hippocampectomy. This may explain why animals with hippocampal and amygdala lesion (or both) have their characteristic learning difficulties, and perhaps equally important, why they can readily solve most discrimination problems in which the reinforcing contingencies are not varied.

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