

DISTRACTION AND HABITUATION IN MONKEYS WITH LIMBIC LESIONS¹

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Monkeys with intact brains and with either hippocampal or amygdala lesions were trained on a sequential task and then given tests in which distraction, habituation, and behavioral disinhibition could be measured. The Ss with hippocampal lesions learned the sequential task sooner than did the other groups and were normal in distractibility measures. However, they failed to habituate to the distractor despite the fact that they were fully capable of ceasing overt responses to it. Amygdalotomized Ss showed abnormally high distractibility on all tests but habituated at a normal rate and to a normal extent. The relevance of these results to the "response-inhibition" and "attention" hypotheses of limbic function is discussed.

It has been reported in the literature that rats with hippocampal lesions are less than normally distracted by an irrelevant stimulus while performing an ongoing response (Hendrickson & Kimble, 1966; Raphelson, Isaacson, & Douglas, 1965; Wickelgren & Isaacson, 1963). These observations conform to the response-inhibition hypothesis of hippocampal functioning, although they do not necessarily exclude other possibilities. The studies above, however, did not begin to tap the full range of conditions under which distraction could be defined and measured. It is far from certain that such a thing as distraction "in general" is a meaningful concept. Whether an animal with a given lesion is more or less distractible than a normal subject may well depend upon the conditions of the test. In each of the above studies the distractor was a stimulus entirely unlike the stimuli guiding the ongoing response. The purpose of the present study was first to discover whether hippocampal lesions would also reduce distractibility when the distracting agent closely resembled the guiding cue. Monkeys with intact brains and groups with lesions of the hippocampus

or amygdala were included in the study. The latter group was included primarily because informal observation had suggested they might in some situations be more distractible than the normal subject. In this study distraction was defined as a slowing response speed in an ongoing sequential task. To this end the monkeys were first trained to perform the sequential response, and then the distractor was presented on interspersed trials over a 10-day period. This allowed an investigation of habituation, defined as a waning of distractibility with repeated exposure. Finally, the genuineness of the habituation was investigated through the use of disinhibitory procedures.

METHOD

Subjects, Surgery, and Histology

Subjects were 13 immature Rhesus monkeys weighing 3-4.5 kg. All were housed individually with free access to water, but were kept on a deprivation diet consisting of about two-thirds of their usual ration of monkey pellets. Feeding took place after completion of daily testing. Six Ss had previously been subjected to bilateral removal of the amygdala by aspiration, three to similar removal of the hippocampus, and four to sham operations involving skull but not brain damage. References to surgical details can be found in Douglas and Pribram (1966). Histological preparation of the brains was accomplished according to Sherer and Pribram (1962). The reconstructions of the amygdalotomized brains have appeared in Bagshaw and Benzies (1968). The reconstructions of the hippocampectomized brains can be seen in Figure 1. All resections were reasonably symmetrical bilaterally, and adequate in extent.

¹ This study was supported in part by National Institute of Mental Health Grant MH-12970, by National Institutes of Health Fellowship 2-F2-MH-23,382, and by Career Research Award 5-K6MH-15,214.

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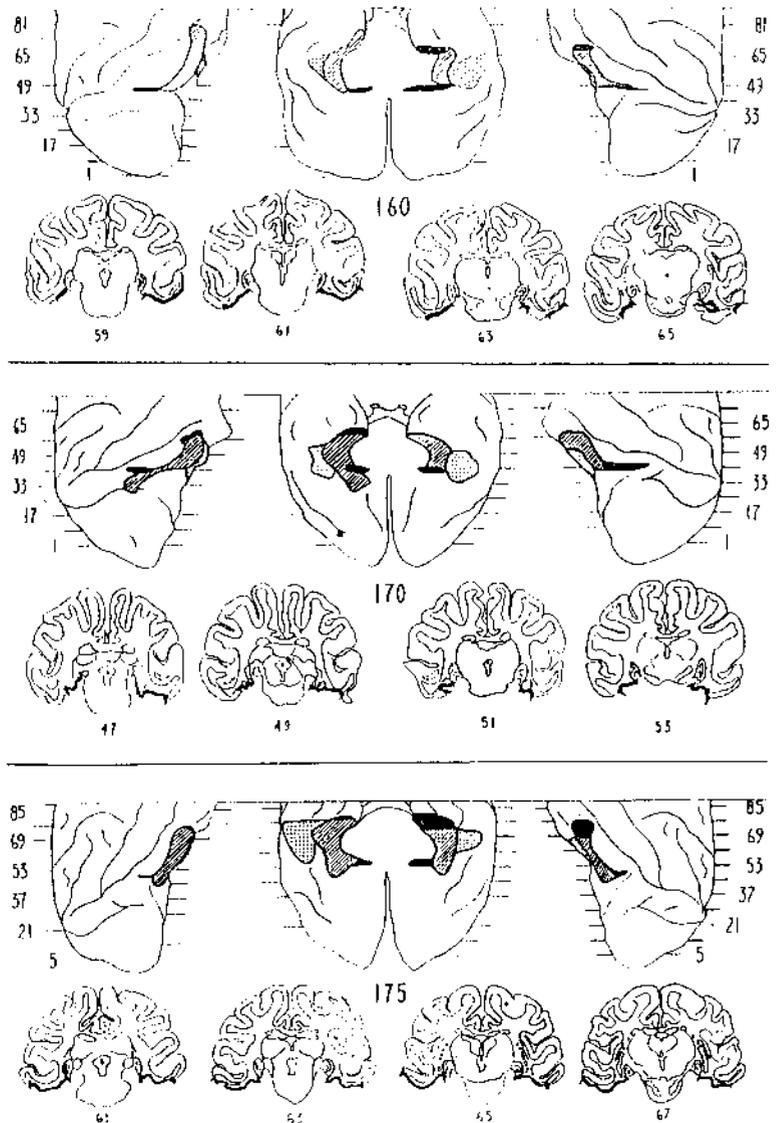


FIG. 1. Reconstructions of the brains of the hippocampectomized monkeys. (Stipple indicates superficial cortical damage; stripe indicates deep temporal lobe excision; black indicates spared hippocampus—Ammon's formation.)

Apparatus

Testing was carried out through the use of a computerized version of the DADTA machine (Pribam, Gardner, Pressman, & Bagshaw, 1963). This consists of an enclosure for *S* with a 4×4 array of 16 depressible clear plastic panels located on one wall. Stimuli are projected onto the panels from the rear, and appear as white patterns (numerals in this case) against a black background. A food cup is located at bottom center of the array. Stimulus presentation, reward delivery, and response recording are automatically carried

out by a specially programmed PDP-8 computer. For descriptive purposes each panel in the array has been assigned a letter of the alphabet as can be seen in Figure 2.

Procedure

At each session *S* was placed in the testing enclosure and allowed a few minutes for adjustment before testing began. A trial was initiated by the appearance of a stimulus (the numeral "5") on Panel P. When this panel was touched or pressed by *S*, the stimulus disappeared and

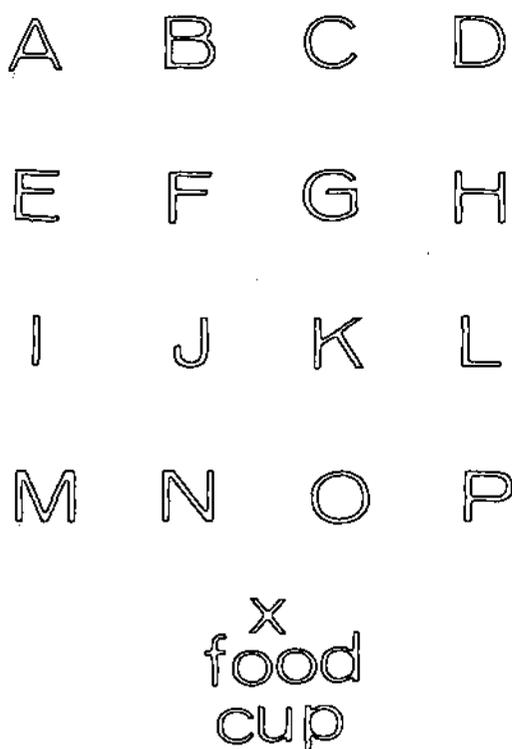


FIG. 2. Location and designation of panels.

different one (the numeral "0") appeared on Panel A. A press of this second stimulus resulted in its disappearance and a simultaneous delivery of a reward (one peanut) into the food cup which terminated the trial. After a lapse of 10 sec. a new trial began involving the same procedure as the first. For the initial training of the sequential response each *S* was given 50 such trials per session, with three daily sessions. Latencies were recorded (to 1,000/sec) both between the initial presentation and the first press of the sequence, and between the two responses. The former measure is used only in the evaluation of learning, as it was found to have no relation to distraction. The latter measure (interresponse latency) was converted into a speed score by adding 1.0 and then dividing the total into 1.0. The use of a reciprocal (or log) conversion is typically employed when dealing with such open-ended distributions, and the prior addition of 1.0 to the denominator was necessary in order to insure that the denominator always exceeded the numerator.

Distraction

At the end of the three training sessions all latency measures had become stable and distraction testing began. Each distraction session consisted of 50 trials, and there were 10 daily sessions. On each session, 46 of the 50 trials were

exactly like those used in original training, while the other 4 were distraction trials. A distraction trial was much like the others except that the press of the first stimulus ("5," at Location P) resulted not only in the presentation of "0" at Location A, but also in the simultaneous appearance of the numeral "1" at one of four locations: D, F, K, or M. Each location was used once per session, with the order randomized. The number of normal trials intervening between distraction trials was varied unsystematically between 5 and 10, and the first distraction trial occurred no earlier than the eleventh trial of the session. A distraction trial was terminated only when the second task response (a press of "0" at A) was made, and this response was rewarded as usual. Subjects were observed through a one-way glass, and overt pressings of the distractor panel were recorded.

Distraction was defined as a difference in interresponse speed when the speed on the distraction trial was compared to the median speed on the three preceding undistracted trials.

Habituation

Over the 10 sessions there was a marked reduction in distraction, as defined above. Habituation was defined as the percentage reduction in distraction on the last five sessions, compared to the first five. The number of overt responses to the distractor also declined, and this was measured as a percentage decline, comparing the number of presses made on the last five sessions with those made on the first five.

Disinhibition

Since both distraction, as presently defined, and overt pressing responses to the distractor declined with trials or exposure, attempts were made to disinhibit both measures. Two such tests were given: The first involved retaining the distracting stimulus "1" from the original distraction series, but presenting it at a new location, "G." This test consisted of 4 such trials interspersed between 46 normal sequential trials in a single session. This session was given on the day following the distraction test. Location "G" was used on all four disinhibition trials.

The second disinhibition test also consisted of one 50-trial session, and it was administered on the day following the test above. On this second test the location "G" was retained, but four new stimuli (the numerals "2," "4," "6," and "8") were presented there on four scattered trials.

RESULTS

Sequential Task

Original learning of the sequential problem was evaluated by dividing the 150 trials of the three sessions into 10-trial blocks. For each block the average speed

for the entire sequence, from presentation of the first stimulus to the press of the second, was computed. Learning was then defined as the number of blocks prior to the one on which mean speed was at least 90% of that on the fastest block. The hippocampally ablated Ss were found to have reached this high level after 6.3 blocks, while control Ss averaged 10.25 and the amygdala group 9.83. Each of the latter groups was reliably slower than the hippocampal group ($t = 3.4, p < .02$; $t = 3.0, p < .02$ respectively). This finding appears to contradict a report of slower than normal sequential learning after hippocampal lesions (Kimble & Pribram, 1963), but the procedures used in the two studies differed considerably, as is discussed below.

Distraction

Distraction was defined operationally as the difference in interresponse speed (the reciprocal of latency plus 1.0) on the distraction trial, as compared to the median of the three preceding trials. Mean distraction scores for the three groups on the first session (prior to habituation) were: control group, .119; hippocampal group, .129; and amygdalaectomized group, .192. The difference between control and hippocampal groups did not approach significance, while each was reliably less distracted than the amygdalaectomized group ($t = 3.1, p < .02$; $t = 2.7, p < .05$, respectively). In terms of real time, each group had stabilized at a latency of roughly 1 sec. The presence of the distractor typically added another second to the latency of the control and hippocampal groups, and about 2 sec. to that of amygdalaectomized Ss.

The relative distractibility of amygdalaectomized Ss confirmed informal observations made earlier, but the apparently normal behavior of hippocampally lesioned Ss is contradictory to the three reports mentioned earlier. The difference may be due to the fact that in the present experiment the distractor could conceivably be confused with the CS, a condition which did not obtain in the earlier studies.

With one exception the panel locations

were equipotential, the exception being Location M. This proved to be an awkward location with respect to S's position, as direct observation indicated that this site was often blocked from view by S's limbs or body. The groups did not differ reliably in this respect, and when the data are pooled, distraction at Site M is reliably less than that at the other three sites ($t = 4.0, p < .01$). The results were computed both with and without the inclusion of this location and in no case did it make a difference as far as relations between groups were concerned.

Habituation

Mean habituation scores for the three groups were: hippocampal, 12.7% reduction in distraction of the last five vs. the first five sessions; control group, 59.8%; and amygdalaectomized Ss, 42.4%. The difference between the latter two groups is largely due to one animal and does not approach significance. Both the control and amygdalaectomized groups displayed reliably greater habituation than did hippocampal Ss ($t = 2.7, p < .05$; $t = 2.8, p < .05$, respectively). Curves displaying the reduction in distraction over the 10 sessions can be seen in Figure 3.

It can be seen in Figure 3 that the amygdalaectomized monkeys appear to habituate

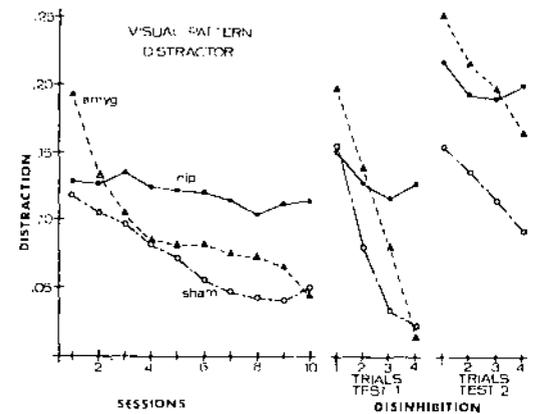


FIG. 3. Distraction with visual pattern stimulus as distractor. (Sham = sham-operated Ss; Amyg = amygdalaectomized monkeys; Hip = hippocampal animals. See method section for definition of distraction.)

more rapidly, and to a greater degree, than do the controls. Faster than normal habituation after amygdectomy can also be seen in curves presented in Schwartzbaum (1964). In contrast, the present hippocampally lesioned Ss habituated only to a modest extent, if at all. This agrees with reports of deficiencies in habituation after hippocampal ablations in rats (Douglas, 1967; Douglas & Isaacson, 1964; Roberts, Dember, & Brodwick, 1962; Teitelbaum & Milner, 1963).

Overt Responding to the Distractor and Its Cessation

For the most part a distracted monkey usually glanced at the distractor and then went on to make the rewarded task response. On other occasions, however, Ss actually made pressing responses to the distractor. The probability of such a response on any trial over all 10 sessions was .33 in the hippocampal group, .14 in the control group, and .19 in the amygdala group. The only difference which approached reliability was that between the hippocampal group and the other two groups combined ($t = 3.3, p < .01$). It can be seen in Figure 4 that most of these responses came in the first few sessions, and that in all groups they declined markedly with experience.

The mean percentage reduction in these responses on the last five sessions vs. the first five were: hippocampal group, 74.2%; control group, 72.2%; and amygdalotomized group, 51.6%. None of these differ-

ences approached reliability, as the apparently lower mean of the amygdala group was due to a single animal which began pressing in the middle sessions. Thus, even though the hippocampally ablated monkeys did not habituate according to the speed measure, they did reduce their initially high tendency to make distractor presses almost down to zero. Unfortunately, it is possible to refer to both types of decremental activity as "habituation." Since the present data indicate that these are two quite different processes, however, the present authors shall refer to the decline on the speed measure as habituation and the decline of distractor pressing as "response inhibition." It is granted that glancing at the distractor (or noticing it, or attending to it) can be said to be a response. It is becoming increasingly clear, however, that such attention or "observing responses" differ considerably from instrumental responses (Zeaman & House, 1963).

One would expect that more time would be required to make a pressing response to the distractor than to merely glance at it, and that the reduction in pressing responses in the hippocampal group should have resulted in a reduction on the speed measure. Perhaps it did, and this factor may account for the slight decline in this group on the speed measure. Surprisingly, however, there was only a slight and insignificant difference between speeds on distraction trials when a press was made and when it was not.

These results contradict the response-inhibition hypothesis of hippocampal function provided that it is understood that the "response" refers to instrumental responses. First, the hippocampally lesioned monkeys should not have pressed the distractor at all, being presumably unable to inhibit the well-practiced task response. Second, granted that they *are* making pressing responses to the distractor, they should not be able to cease making them. A similar finding of an apparently intact ability to cease making instrumental responses in hippocampectomized cats has recently been reported (Brown, Kauffman, & Marco, 1968).

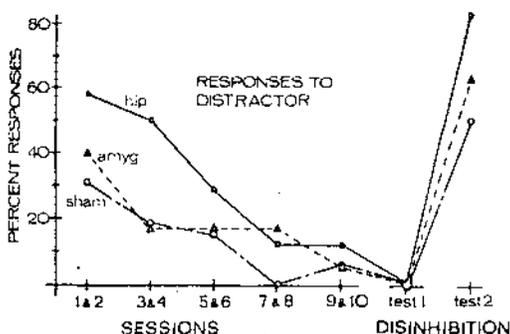


FIG. 4. Overt responses to the distracting stimulus.

Disinhibition

A great difference between observing and instrumental responses also showed up on the two disinhibition tests. In Test 1 the old stimulus "1" was retained as a distractor but presented on four separate occasions at a new location. As can be seen on the right of Figure 3, the change in location appeared to dishabituate the speed measure completely in both control and amygdalotomized Ss. The hippocampal group was little affected, presumably because it had not habituated to any appreciable extent in the first place. Mean distraction scores on the first trial of Test 1 were: hippocampal group, .153; control group, .155; and amygdala group, .198. These figures are very close to those obtained on the first session of the original distraction test, and even closer when the results of the ineffective Site M are excluded from the original test. These figures represent a rebound from the immediately preceding session of 350% in the amygdala group, 206.3% in the control group, and only 35.4% in the hippocampal group. The difference between the former two groups, though large, was not significant ($t = 1.1$). Both amygdala and control groups had larger rebounds than did the hippocampal group ($t = 3.7, p < .01$; $t = 2.9, p < .05$, respectively).

The disinhibition in the amygdala and control groups was followed by very rapid rehabituation, as can be seen in Figure 3. The stabilization of the distractor at one location over the four trials probably contributed to this effect.

While the change of location appeared to dishabituate two of the groups completely, not a single animal in any group made a single pressing response to the distractor on this test, as can be seen in Figure 4.

On Test 2, given on the day following Test 1, the location was retained but four new stimuli were presented there on 4 trials scattered throughout the 50-trial session. This procedure had a dramatic effect in restoring overt distractor presses. Since there was no change in this tendency over the four trials in any group, the re-

sults have been pooled. Mean distractor presses per opportunity were: hippocampal group, .83; control group, .50; and amygdala group, .40. These figures closely match those of the first session of the original distraction test if Site M is excluded (.78, .42, and .53, respectively). It can be seen in Figure 3 that the latencies also went up drastically under these conditions, and that habituation within the session was moderate in comparison to Test 1. Unfortunately, variability was high, and it could not be reliably demonstrated that the latencies of Test 2 were higher than those of Test 1. It seems clear that distraction trials which include a pressing response tend to have longer latencies than those where such a response is not made, but some animals at some times take very little time to make the press, while others gaze for relatively long periods without making an overt response.

DISCUSSION

The present data demonstrate in a number of ways that the decline in distractibility, as reflected in the speed measure, is a phenomenon which differs in many respects from the decline in overt pressing responses. This does not imply, however, that the two are entirely independent. For example, if the distractor is not "noticed", then it will not be pressed either. On the other hand, it was repeatedly demonstrated that some animals, fully capable of withholding an overt response, were unable (or unwilling) to stop being distracted. The decline in overt responding appeared to be related to the particular stimulus used as a distractor, while the decline in distraction (speed measure) could be disinhibited by either a change in stimulus or its location. The authors suggest that the declining speed measure primarily reflects a declining probability of attention being paid to the distractor, and that the deficit in the hippocampal group is an inability to cease attending to the distractor. The cessation of overt responding may, on the other hand, indicate the course of a conditional type of learning: The subject had no way of knowing that the distractor would

not be rewarded and, after a few unrewarded trials, learned that the distractor was crucially different from the CS. According to this line of analysis the hippocampectomized animal is fully capable of this learning, as the data would also indicate.

The relatively fast learning of the sequential problem by hippocampectomized Ss in this study appears to be at variance with an earlier report of defective sequential learning after this lesion (Kimble & Pribram, 1963). In the earlier study, however, the two stimuli were presented simultaneously. In order to perform correctly, S presumably had to refrain from pressing the stimulus of the pair which generated the reward. It was reported that both the control and hippocampectomized monkeys in that study rapidly developed a maladaptive tendency to make this error. The normal animals, however, soon learned to refrain from making this response, while hippocampally ablated Ss did not, or could not. Thus, the failure of the hippocampal group in the earlier study could be interpreted as being due to a lack of inhibition: Either the lack of response inhibition or an inhibitory attentional process would account for the results. In the present case no such "inhibitory" process was necessary, and indeed one might even expect an intact inhibitory tendency to hinder learning, since the first response was not overtly rewarded, and Ss with inhibitory abilities might shy away from the first stimulus after the first few presses. Such an effect did, in fact, seem to occur in both the normal and amygdalectomized animals of the present study. These Ss had a period varying roughly 10-40 trials in which speed of the initial response either remained constant or actually declined. This was then followed by a rapid increase. By contrast, in the hippocampal group the speed of the first response increased steadily from the first trial. The three groups were almost identical in inter-response speed at all points during learning. Unfortunately, there was so much variability in the length of the plateau or dip in the initial-response speed curve that

it proved to be impossible to establish a criterion which would summarize the effect and lead to reliable group differences.

The relatively high distractibility of amygdalectomized Ss and their subsequent rapid habituation also fits into the original Douglas-Pribram hypothesis that the amygdala is concerned with increasing attentiveness as a function of reinforcement. That is, these animals were relatively distractible because the attention-getting value of the CS was relatively low in comparison to that of the distractor. The pronounced habituation of distractibility in this group is consistent with other evidence indicating superior inhibitory ability in animals with amygdala lesions (Douglas & Pribram, 1966). According to the Douglas-Pribram model, the amygdalectomized animals are in full possession of an inhibitory attention-directing mechanism (akin to internal inhibition) which is more than normally able to control their behavior because of a lack of opposition from the lost system.

In summary, it appears that most of the present results conform to an attentional hypothesis of limbic functioning, while some of the results pose definite problems for a response-inhibition theory. It is unfortunate that the two ideas so often come up with the same prediction regarding overall behavior. This is primarily due to a tendency to evaluate behavior in terms of "correctness." Perhaps further progress in distinguishing between the two ideas will require the study of behavior more clearly related to attention, such as eye movements.

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(Received January 20, 1969)