Absence of latent learning in rats with hippocampal lesions¹

DANIEL PORTER KIMBLE AND ERNEST G. GREENE,
DEPARTMENT OF PSYCHOLOGY, UNIVERSITY OF
OREGON, Eugene, Ore. 97403

Albino rats with either bilateral hippocampal lesions, neocortical lesions, or no lesions were allowed to explore either a Hebb-Williams open field or a Lashley III maze. All animals subsequently learned the maze for a water reward. Normal and neocortically-lesioned rats allowed to explore the maze before training made fewer errors than those allowed to explore the open field. Rats with hippocampal lesions did not display such latent learning, despite greater than normal exploratory behavior.

Of the several hypotheses developed in recent years regarding the functions of the mammalian hippocampus, at least two would seem to indicate that rats with hippocampal lesions should not show any particular benefit from free exploration on subsequent maze learning, that is, that hippocampal lesions should reduce "latent" learning. Several studies indicate that hippocampal lesions distort the normal orienting or exploratory behavior of rats (Wickelgren & Isaacson, 1963; Raphelson et al, 1965; Leaton, 1965; Kirkby et al, 1967) although an actual deficit in the orienting response itself can be ruled out (Hendrickson & Kimble, 1967). If these findings are interpreted to mean that hippocampal lesions are incompatible with normal attentional processes, latent learning might presumably suffer (Tolman, 1948).

If hippocampal lesions disrupt a neural system which is important for the selection, emission, and inhibition of responses (McCleary, 1966) then exploratory behavior patterns might be more difficult for the hippocampally-lesioned animal to eliminate from its repertoire. Thus both hypotheses would seem to predict reduced latent learning as a result of hippocampal damage despite the well-documented fact that rats with such lesions show increased "exploratory" behavior in either open field or maze situations (Kimble, 1963; Stein & Kimble, 1966; Kirkby et al, 1967). The present experiment thus undertook an examination of latent learning in normal, neocortically-lesioned or hippocampally-lesioned rats.

Method. The Ss were 24 male Sprague-Dawley albino rats, 90 days old at the time of surgery and approximately 150 days old at the time of testing. All Ss had previously been trained in a brightness discrimination and reversal. Eight of the rats sustained bilateral hippocampal lesions, eight had received lesions of the neocortex overlying the hippocampus, and eight rats served as unoperated controls. The surgical technique has been published previously (Kimble, 1963).

Our procedure most closely resembles the Type 2 design (of five latent learning paradigms) as identified by MacCorquodale & Meehl (1954), consisting of free exploration followed by introduction of reward. Half of the rats in each group were allowed free exploration of the Lashley III maze for 5 min each day for four days. No food or water was available in the maze, nor were the animals deprived of either food or water. The remaining half of each group was allowed an equivalent time to explore a 30 in. square Hebb-Williams open field. On the last day of training all Ss were water-deprived. For the duration of testing Ss received water only in the maze and for 5 min each day in their home cage. On the first training day, a water bottle was available in the goal box of the Lashley III maze. Each S was placed in the goal box and allowed 15 sec drinking. It was then placed in the start box and allowed to run the maze. Entering a cul de sac and backtracking through a gate were counted as errors. Repeating the same error within a given trial was not counted. To minimize backtracking, a gate was lowered after the rat had passed through the subsequent gate. Each S was allowed 15 sec drinking in the start box. Five massed trials were run each day for four days.

Results. It was possible to formulate a simple model of the expected results, and therefore use linear contrast to evaluate the data. The contrast weights were generated from the following predictions: (1) The rats with hippocampal lesions would make more errors than would those with neocortical lesions, which in turn would make more errors than normal Ss. (2) The cortically-lesioned and normal rats allowed to explore the maze would

produce fewer errors than those allowed to explore the open field; but this would not be true for the hippocampally-lesioned rats. Since there was no basis for predicting whether pre-exposure to the maze or lesion treatment would have the strongest effect, an equal effect was assumed. It was also assumed that the spacing among groups was equal. Table 1 summarizes the results. It shows that the results were consistent with the predictions. The result of particular interest is that pre-exposure to the Lashley III maze did not reduce the error scores of the rats with hippocampal lesions whereas it did for the other animals. The pre-exposure benefit was particularly evident during the first 10 trials and diminished after that among the normal and cortically-lesioned rats. The data were evaluated using an F test of the linear contrast specified a priori (Guenther, 1964). The computed F was 41.5 ($p < .001$). The degree to which the data departed from prediction was evaluated by subtracting the sum of squares of the linear contrast from the sum of squares for the treatment conditions. The residual F was 5.27 ($p < .01$). A post hoc procedure was used to suggest the source of the departure from prediction. If the difference in error rate between rats with hippocampal lesions and those with neocortical lesions is assumed to be three times larger than the difference between the normals and cortically-lesioned Ss (where previously these differences were assumed to be equal), the new linear contrast accounts for 97% of the total treatment variance.

The effect of the neocortical lesions was evident only during the first 10 trials. Likewise, pre-exposure differences were seen only during that same period. A 2 by 2 analysis of variance of these 10 trials showed both cortical lesion and pre-exposure differences to have significant influence on error levels ($F = 6.4$, $p < .05$ and $F = 4.78$, $p < .05$ respectively). The general histological procedure has been published previously (Kimble et al, 1966) and Fig. 1 shows representative frontal sections from a hippocampally-lesioned and neocortically-lesioned brain.

Discussion. The result of primary interest is the lack of "latent learning" among the rats with hippocampal lesions. Latent learning, while relatively modest in magnitude, was displayed by both the normal and neocortically-lesioned rats. The difference in degree of influence of pre-training experience cannot be due to hypoactivity in the pre-exposure condition by the hippocampally-lesioned rats, for just the opposite was the case; they consistently entered more squares in the open field than Ss from the other groups and were much more active in the maze as well. It is interesting that both of the hypotheses of hippocampal function cited earlier could generate the present results, but for different reasons. Thus, if there is an abnormality

Table 1
Errors on Day 1 and Total Errors on Maze Learning for Normals (N),
Cortically-lesioned (CC) and Hippocampally-lesioned (H) rats

Rat No.	Maze pre-exposure		Rat No.	Open-field pre-exposure	
	Day 1	Total		Day 1	Total
N 20	22	57	N 15	17	62
N 32	14	56	N 12	20	59
N 13	12	16	N 4	17	48
N 31	12	50	N 23	22	57
Mean	15.0	44.7		19.0	56.5
CC 6	18	41	CC 8	31	59
CC 21	17	50	CC 17	24	58
CC 5	20	67	CC 27	26	74
CC 11	20	62	CC 2	26	63
Mean	18.7	55.0		26.7	63.5
H 29	34	97	H 24	26	95
H 3	37	107	H 7	32	96
H 10	27	98	H 1	27	94
H 18	24	83	H 30	24	80
Mean	30.5	96.2		27.2	91.2

Fig. 1. Representative frontal sections of a brain from a hippocampally-lesioned rat (top) and from a neocortically-lesioned rat (bottom).

of attentional processes, then despite greater activity in the pre-exposure period the rats with hippocampal damage would actually have attended to fewer relevant aspects of the maze, built a poorer "cognitive map," and therefore not display any benefit (i.e., latent learning) of this pre-exposure.

The "inhibition of erroneous response" hypothesis could also account for the present results since it might be predicted that pre-exposure experience

would allow the expression of responses which would later, in the training phase of the experiment, be regarded as errors (i.e., exploration of cul de sacs). If hippocampal lesions impair the rat's ability to later inhibit these responses, this impairment could tend to cancel out any benefit of pre-exposure.

It seems that a test of these two notions could be devised by simply extending the duration of the pre-exposure period. Presumably, given many hours of pre-exposure, the hippocampectomized rat could partially compensate for an attentional deficit and thus these Ss would display more latent learning with longer pre-exposure periods. However, no such improvement would follow if the lesion impaired a response inhibition process. One might actually predict increasingly poorer maze performance as a function of increased duration of pre-exposure period, since the to-be-erroneous exploratory responses might be expected to be strengthened with repetition.

The present discussion obviously does not exhaust the possible interpretations of the primary finding of this experiment—the apparent lack of latent learning in rats with hippocampal lesions.

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NOTE

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