Fig. 1. The lower curve represents the mean average discrimination scores as a function of the age of S at test. The upper curve represents the same scores when these are combined (made absolute) for both hen and age-mate. The independent scores of 32 Ss are included in each point; N = 256.

heredity and evolution for a theoretical framework. Let us consider them.

One, since none of the chicks in this experiment was given food or water until after the test, couldn't the demonstrated behavior be due, first, to activity initiated by tissue-need, and second, to malnutrition and loss of motor energy? Answer: The chick's internal source of food, as psychologists have known since Spalding's time, is adequate for 4 or even 5 days after hatching (Gray, 1967). The behavioral signs of malnutrition are listlessness and drooping wings. None of the chicks showed such symptoms and one and all ate normally after their testing; none appeared ill and none died.

Two, could it not be that what we have in Fig. 1 is a progressive preference for the age-mate as a result of CNS arousal, but that, after 36 h of age, there is less and less responsiveness because of the damping of the arousal system? Answer: To ascertain if such an objection had merit, we added time spent before the age-mate to time spent before the hen. This arrangement of absolute scores is plotted (arbitrarily) on the age-mate side of Fig. 1. We see that the total scores are about the same for the various age groups. Analysis of variance yielded no significant differences.

Therefore, the preference curve at the bottom of the figure is truly a discrimination curve, not an energy curve or an "arousal-damping" curve. This will be easier to understand when we say that the older chicks' lack of preference is indicated not by failure to respond but by *indiscriminate* responding. Descriptively, a chick older than about 36 h runs first to the hen and then to the age-mate, back and forth, accumulating about equal scores for each but losing a certain amount of total time because it often crosses the neutral area of the tilt-platform.

Taken at face value, the experiment reported here shows a biphasic familial preference that undoubtedly predicts some of the events that would occur when the hen incubates and mothers her young. The hens used as stimulus objects in this experiment were, of course, not mothers, and it's quite possible that they conveyed to the Ss some of the hostility that nonbroody hens typically exhibit toward young chicks.

It is suggested that the innate factors of imprinting should be isolated and accounted for before differential responsiveness can be ascribed to learning.

Memory loss following posttrial electrical stimulation of the hippocampus¹

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Retention of a one-trial passive-avoidance response in rats was found to be disrupted by subconvulsive electrical stimulation of the hippocampus, which was administered immediately following footshock. Lesions of the hippocampus or stimulation of anatomically related areas did not impair performance of the avoidance response significantly.

Although electroconvulsive shock (ECS) may have anterograde effects on retention (Hudspeth & Gerbrandt, 1965), amnesic changes are probably responsible for interference with performance in one-trial passive-avoidance situations. While it is often assumed that ECS disrupts a neural consolidation process underlying the formation of memory traces (McGaugh, 1966), the locus of action of ECS has received only limited experimental attention (Kesner & Doty, 1967; Hostetter, 1968). A considerable amount of research has accumulated indicating that the brain

- REFERENCES
- EDWARDS, A. L. *Experimental design in psychological research*. (2nd ed.) New York: Holt, Rinehart & Winston, 1960.
- GRAY, P. H. The releasers of imprinting: Differential reactions to color as a function of maturation. *Journal of Comparative & Physiological Psychology*, 1961, 54, 597-601.
- GRAY, P. H. Is the imprinting critical period an artifact of a biological clock? *Perceptual & Motor Skills*, 1962, 14, 70.
- GRAY, P. H. Interactions of temporal and releasing factors in familial recognition of own and ancestral species. *Perceptual & Motor Skills*, 1964, 18, 445-448.
- GRAY, P. H. Spalding and his influence on research in developmental behavior. *Journal of the History of the Behavioral Sciences*, 1967, 3, 168-179.
- GRAY, P. H., YATES, A. T., DAVIS, G. T., & MODE, C. J. Some aspects of the genetics of imprinting. *American Zoologist*, 1966, 6, 568.
- HESS, E. H., & HESS, D. B. Innate factors in imprinting. *Psychonomic Science*, 1969, 14, 129-130.

NOTES

1. The experiment reported here was supported by U.S. Public Health Service Grants MH-05766 and HD-00877 and was conducted in the senior author's Imprinting Laboratory at Montana State University.

2. Stella J. Sallee is now at Willamette University and Allen T. Yates is at Bowling Green State University.

hippocampi are involved in the integration of information to be stored elsewhere (Penfield & Milner, 1958; Flexner, Flexner, & Roberts, 1967; Deutsch, Hamburg, & Dahl, 1966; Uretsky & McCleary, 1969). Among recent hypotheses are several that propose that findings of retrograde amnesia produced by ECS depend on disruption of electrical activity in the hippocampus (Hudspeth & Gerbrandt, 1965; Douglas, 1967; Kimble, 1968; Routtenberg, 1968). Several recent studies (Stein & Chorover, 1968; Brunner & Rossi, 1969; Erickson & Patel, 1969) may be interpreted as showing that localized low-level electrical stimulation delivered to the hippocampus disrupts its functioning. Unlike the earlier experiments, low-level electrical stimulation of short duration was administered following a learning procedure typical of those used in ECS studies of memory.

SUBJECTS, SURGERY, AND HISTOLOGY

Forty-five male hooded rats (Long-Evans) served as Ss in the experiment. Thirty-two of these were implanted with a bipolar stimulating electrode (Stutz, 1968). Eleven Ss received

electrodes in the dorsal hippocampus (HpESB), 12 electrodes were aimed at the cortex overlying the hippocampus (CxESB), and 9 were aimed at the septal region (SpESB). Five Ss sustained large bilateral electrolytic lesions of the hippocampus (HpL) and eight Ss served as unoperated controls (C). Surgery was performed under Sedax anesthesia, and Ss were atropinized. The Ss were allowed about a month to recover from surgery during which time gentling procedures were initiated. At the conclusion of the experiment, the electrode placements and lesions were confirmed by microscopic examination of cresyl violet stained sections.

APPARATUS

The apparatus and basic procedure relies on the observation that rats will spontaneously step off of a slightly elevated platform and explore the test box. The apparatus consisted of a 50-cm-square box constructed of plywood, with gray walls and a grid floor. Located against the rear wall of the box was a 12.5-cm-square platform, 5 cm high. The platform was connected to microswitches that operated a timer and scrambled shock source that was set to deliver a .5-mA footshock for .5 sec.

PROCEDURE

The Ss were given three habituation trials (one per day), in which they were placed on the platform and, after stepping down, permitted to explore the test box for 10 sec. Step-down latencies were recorded. On Day 4, all Ss were given footshock immediately upon stepping completely off of the platform. Brain-stimulation groups received a 1.5-sec train of 60-cycle ac shock (25-50 microamperes) following footshock and an identical train 10 sec later. The ESB was triggered by E and delivered through a flexible lead that was attached to S during all sessions. Motor convulsions were not observed in any S. Other Ss (C and HpL) remained on the grid floor for 10 sec following footshock before being returned to their home cages. Twenty-four and 48 h later, Ss were tested for retention. Retention trials (Days 5 and 6) consisted of the same procedure as that employed for habituation trials unless the rat remained on the platform for 100 sec, in which case the trial was terminated.

RESULTS AND DISCUSSION

Table 1 presents median step-down latencies on the third day (preshock) and the two postshock retention days. Separate rank-order statistical comparisons (two-tailed Mann-Whitney U tests) were made between the unoperated controls and each of the experimental groups. Group HpESB was found to have

Table 1
Median Step Down Latencies (in Sec) of Rats
Prior to Receiving Footshock,
24 h and 48 h After Shock

Group	Preshock	Postshock	
		24 h	48 h
HpL	3.9	72.8	73.0
Control	2.8	26.6	20.5
HpESB	3.5	9.3	8.0
CxESB	2.4	8.0	13.0
SpESB	2.5	11.9	24.0

significantly shorter latencies than unoperated controls 24 h ($z = 2.35$, $p < .05$) and 48 h ($z = 1.98$, $p < .05$) after training. The hippocampal-lesioned group (HpL) had postshock latencies significantly longer than did controls at 24 h ($z = 2.35$, $p < .05$). This difference was not reliable in the 48-h retention test ($z = 1.32$, $p > .10$). No statistically significant differences occurred between either CxESB Ss (24 h: $z = 1.77$, $p > .10$; 48 h: $z = .90$, $p > .10$) and controls or SpESB Ss (24 h: $z = 1.25$, $p > .10$; 48 h: $z = .20$, $p > .10$) and controls. Similarly, there were no significant differences between SpESB or CxESB groups and the hippocampally stimulated group. The data in Table 1 appear to show retention deficits produced by cortical and possibly septal stimulation as well as hippocampal stimulation. The statistical tests indicate, however, that retention of inhibition of the step-down response was more severely affected by stimulation of the hippocampus than of related areas.

The results reported here indicate that retrograde amnesia may be produced by short duration, posttrial electrical stimulation of the hippocampus but not necessarily by stimulation of anatomically related areas. These findings are in agreement with the postulated relationship between hippocampal activity and memory. In support of a recent experiment using a similar task (Winocur & Mills, 1969), it was found that hippocampally lesioned Ss readily retained the passive-avoidance response. While the present study was in preparation, there were several similar experiments reported (Avis, 1969; Barcik, 1969; Shinkman & Kaufman, 1969). Electrical stimulation of the hippocampus may induce greater dysfunction of related structures than does surgical hippocampectomy, which leaves some connections intact (Uretsky & McCleary, 1969). It would seem that arguments forwarded against the use of electroconvulsive shock (Hudspeth, McGaugh, & Thompson, 1964) and surgical ablation in studies of memory in situations involving prolonged training periods do not necessarily prohibit the use of low-level electrical stimulation of the brain.

REFERENCES

- AVIS, H. H. Retrograde amnesia produced by seizure activity in the hippocampus of the rat. Paper read at the Eastern Psychological Association convention, 1969.
- BARCIK, J. D. Hippocampal afterdischarges and memory disruption. *Proceedings, 77th Annual Convention, APA*, 1969, 185-186.
- BRUNNER, R. L., & ROSSI, R. R. Hippocampal disruption and passive avoidance behavior. *Psychonomic Science*, 1969, 15, 228-229.
- DEUTSCH, J. A., HAMBURG, M. D., & DAHL, M. Anticholinesterase-induced amnesia and its temporal aspects. *Science*, 1966, 151, 221-223.
- DOUGLAS, R. J. The hippocampus and behavior. *Psychological Bulletin*, 1967, 67, 416-442.
- ERICKSON, C. K., & PATEL, J. B. Facilitation of avoidance learning by posttrial hippocampal electrical stimulation. *Journal of Comparative & Physiological Psychology*, 1969, 68, 400-406.
- FLEXNER, L. B., FLEXNER, J. B., & ROBERTS, R. B. Memory in mice analyzed with antibiotics. *Science*, 1967, 155, 1377-1383.
- HOSTETTER, G. Hippocampal lesions in rats weaken the retrograde amnesic effect of ECS. *Journal of Comparative & Physiological Psychology*, 1968, 66, 349-353.
- HUDSPETH, W. J., & GERBRANDT, L. K. ECS conflict, competition, consolidation, and neuroanatomical functions. *Psychological Bulletin*, 1965, 63, 1-13.
- HUDSPETH, W. J., MCGAUGH, J. L., & THOMPSON, C. W. The aversive and amnesic effects of electroconvulsive shock. *Journal of Comparative & Physiological Psychology*, 1964, 57, 61-64.
- KESNER, R., & DOTY, R. W. Amnesic effects dependent upon locus of ECS stimulation. Paper presented at the meetings of the American Physiological Society and the Psychonomic Society, 1967.
- KIMBLE, D. P. The hippocampus and internal inhibition. *Psychological Bulletin*, 1968, 70, 285-295.
- MCGAUGH, J. L. Time dependent processes in memory storage. *Science*, 1966, 153, 1351-1358.
- PENFIELD, W., & MILNER, B. Memory deficit produced by bilateral lesions in the hippocampal zone. *AMA Archives of Neurology & Psychiatry*, 1958, 79, 475-497.
- ROUTTENBERG, A. The two arousal hypotheses: Reticular formation and limbic system. *Psychological Review*, 1968, 75, 51-80.
- SHINKMAN, P. G., & KAUFMAN, K. P. Effects of posttrial hippocampal stimulation on acquisition of a CER. Paper read at the Southeastern Psychological Association convention, 1969.
- STEIN, D. G., & CHOROVER, S. L. Effects of posttrial electrical stimulation of hippocampus and caudate nucleus on maze learning in the rat. *Physiology & Behavior*, 1968, 3, 787-791.
- STUTZ, R. M. Stimulus generalization within the limbic system. *Journal of Comparative & Physiological Psychology*, 1968, 65, 79-82.
- URETSKY, E., & MCCLEARY, R. A. Effect of hippocampal isolation on retention. *Journal of Comparative & Physiological Psychology*, 1969, 68, 1-8.
- WINOCUR, G., & MILLS, J. A. Hippocampus and septum in response inhibition. *Journal of Comparative & Physiological Psychology*, 1969, 67, 352-357.

NOTE

1. This work was aided by Research Grant GB 7629 from the National Science Foundation.