Single-alternation pattern running in amygdalectomized rats

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Amygdalectomized and nonlesioned rats were administered alternated reinforced and non-reinforced trials in an alleyway. Animals with amygdala lesions showed slower response speeds during earlier stages of training. As training continued, reinforced-trial speed, characteristically faster than nonreinforced-trial speed in normal animals, was delayed in the lesioned animals. Once the lesioned animals exhibited pattern running, there were no differences between them and the nonlesioned group. These results were discussed in terms of Richardson's (1973) theory.

The most common example of pattern running is the faster reinforced-trial than nonreinforced-trial speed that ultimately occurs when reinforced and nonreinforced trials are alternated (Bloom & Capaldi, 1961; Capaldi, 1967). Here, single-alternation patterning occurs because "a characteristic cue is produced by reinforcement (S^R), as is by nonreinforcement (S^N), and under a single-alternation schedule, S^R always occurs on nonreinforced trials and so is the $S-$ cue, while S^N always occurs on reinforced trials and so is the $S+$ cue" (Capaldi, 1979, p. 64). According to this analysis, pattern running may be described as discriminative running based on internal memory cues provided by a nonrandom schedule of reinforcement.

According to Richardson (1973), the normal function of the amygdala is to incorporate and integrate internally produced representations of cues and stimuli into new behavior patterns; lesioning the amygdala results in the disruption of these internally generated representations. Support for Richardson's theory is derived from studies in which the acquisition or retention of a task by lesioned subjects is dependent upon the utilization of either external environmental stimuli or internal cues. Consider the former case. Amygdaloid ablation does not interfere with rather complex tasks so long as the correct response is cued by an external environmental stimulus. For example, the acquisition of a go/no-go discrimination cued by a light and the reversal of this discrimination were not disrupted in lesioned rats (Pellegrino, 1968). On the other hand, for those tasks whose performance depends on internally formed cues for the correct response, a lesion deficit is observed. For example, Pellegrino (1968) re-

ported that lesions of the basolateral region of the nucleus impaired performance on a timing task (DRL-20) relative to performance by a control group of rats (see also Pubols, 1966). Likewise, Schartzbaum and Poulos (1965) reported that amygdalectomized monkeys did worse on a discrimination reversal task than did the control animals. It appears, then, that the amygdaloid lesion deficit is observed when internally generated representations of cues (e.g., timing task or discrimination reversal), rather than external stimuli (e.g., light), are being utilized for the correct response.

The purpose of this experiment was, first, to further extend those investigations that had incorporated internally produced cues into their manipulations in the study of the amygdala and, second, to test the lesioned rat on an instrumental running response rather than on the more commonly used leverpress task.

Since Capaldi's (1967) account suggests that single alternation utilizes cues that are internally produced, such as $S+$ and $S-$ cues, and given previous findings of a deficit in performance of those tasks utilizing internally formed cued responding, the hypothesis of this study is that single-alternation pattern running in amygdalectomized rats will be impaired.

METHOD

Subjects

Fifteen experimentally naive, 180- to 220-g male Holtzman rats were used. At the beginning of the study, their ages ranged from 90 to 100 days. The animals had ad-lib access to water.

Apparatus

The alleyway was similar to that used by Goomas and Steele (1980). Briefly, the gray plywood alleyway consisted of a startbox ($35.2 \times 10.4 \times 13.5$ cm), a runway ($121.5 \times 10.4 \times 13.5$ cm), and a goalbox ($30.2 \times 10.4 \times 13.5$ cm). Hinged hardware cloth covered the entire length of the apparatus. Guillotine doors separated the startbox from the runway and the runway from the goalbox. Raising the startdoor activated the first of two .01-sec clocks. Interrupting a photobeam located 96.0 cm beyond the

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startdoor stopped the first clock (run time) and started the second clock. The interruption of a second photobeam, located 32.5 cm beyond the first photobeam and approximately 5 cm inside the goalbox, stopped the second clock (goal time). At the distal end of the goalbox was an earthen foodcup (diameter = 8.0 cm; height = 3.1 cm). The second guillotine door served to confine the rat to the goal area.

Surgery

Under sodium pentathol (60-mg/kg) anesthesia, bilateral electrolytic lesions were placed in the amygdaloid complex of seven rats by passing a 2.0-mA direct current through the uninsulated tip of a 28-ga wire for 20 sec. The electrode was placed 2.0 mm posterior to bregma, 4.25 mm lateral to the midline, and 8.25 mm ventral to the surface of the cortex (head horizontal). Eight rats sustained identical control operations, whereby the electrode was lowered 6.0 mm and then withdrawn.

Training

After 14 days of postoperative recovery, each animal was placed on a 23-h food deprivation schedule for the remainder of the experiment. On Days 15 to 19, each subject was handled for 2 min, allowed 3 min of exploration, and fed eight 45-mg Noyes pellets in addition to its regular 1-h feeding.

Day 20 was the first day of training. All subjects received alternating reinforced and nonreinforced trials, with reinforcement occurring on all odd-numbered trials. A trial was initiated by placing the subject in the startbox. After 3 sec, the experimenter raised the startdoor to allow the animals to enter the runway. Once inside the goalbox, the subjects received eight 45-mg Noyes pellets on reinforced trials or 10 sec of confinement on nonreinforced trials. The subjects were given six trials per day for 18 days.

The rats were run in four squads (three squads of four rats and one squad of three rats). The selection was randomized daily. The intertrial interval was 20 sec, which the rats spent in a neutral holding cage.

Histology

At the conclusion of testing, the rats that had sustained amygdaloid lesions were placed under deep anesthesia and then perfused with isotonic saline followed by 10% formalin. The brains were removed, fixed in 10% formalin for 48 h, frozen, and, finally, sectioned at 20 to 45 μ in the frontal plane. Every fifth section was mounted and then stained with cresyl violet.

RESULTS

Damage extended generally throughout the entire anterior-posterior extent of the cortical and basal nuclei. In all cases, the medial and central nuclei

Figure 1. Damage to cortical and basal nuclei of the amygdaloid complex.

Figure 2. Run speeds of nonlesioned (top panel) and lesioned (bottom panel) animals to reinforced (R) and nonreinforced (N) trials.

were spared. The damage corresponded to areas represented on Plates 30 to 35 of the stereotaxic atlas by König and Klippel (1963). Figure 1 is representative of the extent of the lesion.¹

The results in Figure 2 are based on $\log_{10} (1/\bar{x} + 1)$ transformation speed scores of the daily mean run times on reinforced trials and nonreinforced trials. Generally, differences were larger in the run segment of the alleyway.

Inspection of Figure 2 suggests that (1) early in training, the running speeds of amygdalotomized rats were slower than those of the control animals, and (2) discriminative running occurred later in acquisition for the lesioned animals than for the nonlesioned group.

A two-factor mixed-design analysis of variance (lesion vs. nonlesion as the between-subjects factor; reinforcement vs. nonreinforcement as the within-subjects factor) of all days prior to pattern running by either group—in this case, Days 1 through 6—revealed a reliable difference between lesion vs. nonlesion over Days 5 and 6 [smaller $F(1,13) = 8.21$, $p < .05$]. Although the within-subjects factor was not significant, the curves of Figure 2 graphically depict faster nonreinforced-trial speeds than reinforced-trial speeds over most of these days of early learning.

A treatment $\times$ subjects analysis of variance indicated that pattern running appeared on Day 7 for the nonlesioned animals and continued thereafter [smallest $F(1,7) = 10.67$, $p < .05$]; patterned running for the lesioned animals, however, appeared on Day 14 and continued thereafter [smallest $F(1,6) = 14.96$, $p < .05$].

Once the groups exhibited pattern running, it was possible to make comparisons between the groups in the magnitude of pattern running. A two-factor mixed-design analysis of variance of Days 14 to 18, however, revealed no reliable differences for the

trial $\times$ condition interaction ($F_s < 1$). Although the effect for trial, that is, reinforcement vs. nonreinforcement, was reliable [smallest $F(1,13) = 15.55$, $p < .05$], there was little that would indicate that there were differences in the magnitude of pattern running between the two groups.

DISCUSSION

Amygdala lesions retarded the acquisition of the discrimination response but not final performance. That is, the amygdaloid deficit occurred temporarily on a task that utilized internally generated cues. Pattern running, according to a memory interpretation, utilizes internally produced S+ and S- cues. Whatever impairment of function might have occurred, then, was only temporary, since the lesioned animals eventually acquired pattern running too.

This result is incompatible with studies that report *terminal* acquisition differences between amygdalectomized animals and controls on such tasks as behavior contrast (e.g., Henke, 1972) or the frustration effect (e.g., Henke & Maxwell, 1973), both of which utilize internally produced representations of cues for responding. Perhaps what is being observed here is a recovery of function toward specific nuclei. More research addressing this issue is clearly needed.

The involvement of the amygdala in the internally generated representation and manipulation of stimuli, as suggested by the lesion experiments, is also supported by the findings on the monitoring of neural units of the intact amygdala. For example, the amygdaloid units monitored by O'Keefe and Bouma (1969) in cats remained active for a while longer, after a specific stimulus (a black mouse or the song of a bird) had been removed, thus suggesting that the amygdala maintains the neural response to a specific stimulus in the absence of the stimulus. The amygdaloid action was in response to specific stimuli, whereas no response was recorded with changes in alertness. Likewise, while recording EEG potential and amplitude changes in the amygdala, Norton (1970) found that evoked responses occurred only

when the rat was rewarded, not in the absence of reward. Other brain sites showed no such responses.

REFERENCES

- BLOOM, J. M., & CAPALDI, E. J. The behavior of rats in relation to complex patterns of partial reinforcement. *Journal of Comparative and Physiological Psychology*, 1961, **54**, 261-265.
- CAPALDI, E. J. A sequential hypothesis of instrumental learning. In K. W. Spence & J. T. Spence (Eds.), *The psychology of learning and motivation* (Vol. 1). New York: Academic Press, 1967.
- CAPALDI, E. J. Latent discrimination learning under a regular schedule of partial reinforcement. *Animal Learning & Behavior*, 1979, **7**, 63-68.
- GOOMAS, D. T., & STEELE, M. K. The collapse effect and delay of reinforcement with amygdalectomized rats. *Physiological Psychology*, 1980, **8**, 463-466.
- HENKE, P. G. Amygdalectomy and mixed reinforcement schedule contrast effects. *Psychonomic Science*, 1972, **28**, 301-302.
- HENKE, P. G., & MAXWELL, D. Lesions in the amygdala and the frustration effect. *Physiology & Behavior*, 1973, **10**, 647-650.
- KÖNIG, F. R., & KLIPPEL, R. A. (Eds.). *The rat brain: A stereotaxic atlas of the forebrain and lower parts of the brain stem*. Baltimore: Williams & Wilkins, 1963.
- NORTON, P. R. E. Differences in the electrophysiological correlates upon receipt of a food reward and nonreward in the rat. *Brain Research*, 1970, **24**, 134-138.
- O'KEEFE, J., & BOUMA, H. Complex sensory properties of certain amygdala units in the freely moving cat. *Experimental Neurology*, 1969, **49**, 281-285.
- PELLEGRINO, L. J. Amygdaloid lesions and behavioral inhibition in the rat. *Journal of Comparative and Physiological Psychology*, 1968, **65**, 483-491.
- PUBOLS, S. M. Changes in food-motivated behavior of rats as a function of septal and amygdaloid lesions. *Experimental Neurology*, 1966, **15**, 240-254.
- RICHARDSON, J. S. The amygdala: Historical and functional analysis. *Acta Neurobiologiae Experimentalis*, 1973, **33**, 623-644.
- SCHWARTZBAUM, J. S., & POULOS, D. A. Discrimination behavior after amygdalectomy in monkeys: Learning set and discrimination reversal. *Journal of Comparative and Physiological Psychology*, 1965, **60**, 320-328.

NOTE

1. For the cost of reproduction, histological data and raw data will be sent to the user upon request.

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