Runway performance of amygdalectomized rats: Magnitude of reinforcement and delay of food reward

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In an alleyway, running speeds for amygdalectomized rats given large (12 pellets) or small (2 pellets) magnitude of reinforcement were initially faster for the large-magnitude group, but the small-magnitude group caught up (termed the collapse effect) in 20 trials. This is in contrast to intact animals, which typically exhibit the collapse effect anywhere between 50 and 100 trials. As such, control animals administered 20 trials showed the familiar form of responding—the speeds of the large-magnitude animals were reliably faster than those of the small-magnitude animals. With periodic increases in delay of food reward, from 0 to 45 sec in 15-sec increments, lesioned animals ran reliably faster than control animals, more so for the 30- and 45-sec delays. These results were viewed as supporting Richardson's (1973) theory.

According to Richardson (1973), ablation of the amygdala produces many behavioral deficits that center around the inability of the organism to integrate changes in reinforcement contingencies into new behavior patterns. There is considerably less responsiveness, though by no means insensitivity, in operant and runway response rates with amygdalectomized rats (Henke, Allen, & Davison, 1972; Kemble & Beckman, 1970) and monkeys (Schwartzbaum, 1960a, 1960b) following increased or decreased amounts of quantitative reinforcement.

The present experiment was designed to further explore the role of the amygdaloid complex in food-motivated behavior. Its design features magnitude of reinforcement used as a between-subjects factor and delay of food reward used as a within-subjects factor.

Briefly, magnitude of reinforcement data obtained in the runway with the between-subjects methodology indicate that intact subjects given a moderate amount of training typically have higher performance levels the larger the magnitude of reinforcement (Pubols, 1960); however, these performance differences may dissipate following extended training (termed the collapse effect). Several investigators (Black, 1969; Campbell, Batsche, & Batsche, 1972; McCain, Dyleski, & McElvain, 1971) have reported that, with two groups of rats consuming either large or small magnitude of reinforcement, faster speeds were exhibited early in training by the large-magnitude group, but late in training these differences disappeared; the small-magnitude group caught up.

Data obtained in the runway with delay of food reward used as a within-subjects factor indicate that intact subjects exhibit lower performance levels as delay of food reward increases. For example, D'Amato and Cox (1976) reported that when monkeys experienced delay of food reward as short as only 8 sec, performance declined from a virtual 100% to chance responding in a simple two-stimulus discrimination problem. Similarly in pigeons, the effect of delay of food reward on a variable-interval schedule of reinforcement caused a decline in the rate of pecking to a value near zero (Ferster, 1953).

This experiment's design, then, involves comparing running speeds of amygdalectomized rats to those of control (nonlesioned) rats given either large or small magnitude of reinforcement, 12 and 2 pellets, respectively, with periodic increases in delay of food reward, 0- to 15- to 30- to 45-sec goalbox delay.

Two hypotheses are presented. The first is concerned with the timing of the collapse effect, while the second is concerned with responding following periodic increases in delay of food reward.

The first phase of this experiment, 0-sec delay, contains the basic components for studying the collapse effect, that is, groups receive either large or small magnitude of reinforcement. Much of the earlier work on the collapse effect (e.g., McCain et al., 1971) indicates that the collapse effect generally occurs anywhere between 50 and 100 trials.

There are no data on the collapse effect with amygdalectomized animals. Yet, the facts that one of the functions of the amygdala is to interpret and integrate reinforcing stimuli (Richardson, 1973) and that intact animals show the collapse effect between 50 and 100 trials suggest that amygdalectomized animals should exhibit the collapse effect earlier than

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control animals. If this assumption is true, then the following testable hypothesis may be postulated. While the collapse effect occurs anywhere between 50 and 100 trials with intact rats, amygdalectomized rats should exhibit the collapse effect with only a moderate amount of training, that is, in less than 50 trials.

Another reinforcement parameter not yet studied with amygdalectomized animals is that of delay of food reward. The second, third, and fourth stages of this experiment were designed to examine such a parameter. In situations other than food-motivated behavior, researchers have found less responsiveness to aversive stimuli on the part of lesioned animals (Fuller, Rosvold, & Pribram 1957; Pribram & Bagshaw, 1953; Schreiner & Kling, 1953, 1956). With food-motivated behavior, then, assuming that periodic increases in delay of food reward are aversive, it is hypothesized that amygdalectomized animals should be less responsive to the debilitating effects of periodic increases in delay of food reward than control animals; thus, the running speeds of lesioned animals should be faster than control animals following these increases in delay.

METHOD

Subjects

Twenty-seven 90-day-old, 200- to 225-g female rats of Holtzman strain were used. The subjects were housed in separate cages with free access to water upon arrival from the supplier. A 12-h light-dark cycle was maintained throughout the experimental proceedings, with the subjects being tested during the light cycle.

Surgery and Histology

Following sodium amobarbital anesthesia (3 ml/kg), bilateral electrolytic amygdaloid lesions were performed on 16 animals. Electrode placement was 2.0 mm posterior to bregma, 4.25 mm lateral to midline, and 8.25 mm ventral to the surface of the cortex, with the skull level between bregma and lambda. A 2.0-mA direct current of 25-sec duration was passed through the uninsulated tip of a stainless steel wire electrode. Eleven control animals received identical surgery except that no electrode was inserted.

At the conclusion of testing, lesioned animals were deeply anesthetized, then perfused with isotonic saline and 10% Formalin. The brains were removed and hardened in 10% Formalin for 48 h. The brains were then sectioned at 50- to 100-micron thickness in the frontal plane. All sections were mounted and stained with cresyl violet for viewing.

Apparatus

The L-shaped gray plywood alleyway consisted of a startbox (30.2 × 10.4 × 13.5 cm), a runway (114.0 × 10.4 × 13.5 cm), a goalbox (65.2 × 10.4 × 13.5 cm), and an extension (15.3 × 10.4 × 13.5 cm) set at a right angle to the goalbox. Hinged hardware cloth covered the entire length of the apparatus. Guillotine doors separated the startbox from the runway and the runway from the goalbox.

Interrupting a photobeam 5.0 cm beyond the start door activated the first of three .01-sec clocks. Interrupting a second photobeam 25.0 cm beyond the start door stopped the first clock (start time) and started the second clock. The interruption of a third photobeam, located 70.0 cm beyond the second photobeam,

stopped the second clock (run time) and started the third clock. The interruption of a fourth photobeam, located 32.5 cm beyond the third photobeam and approximately 5.0 cm into the goalbox, stopped the third clock (goal time).

At the distal end of the goalbox was the attached extension housing a metal cylindrical wooden-lidded food cup (diameter = 8.0 cm; height = 3.1 cm). The manually operated wooden lid covered the food cup on all reinforced trials preventing access to the pellets until the appropriate delay interval had elapsed. The second guillotine door served to confine the rat to the goalbox.

Pretraining

Animals were assigned randomly to lesioned and nonlesioned (sham surgery) groups prior to surgery. Pretraining was initiated 10 days postsurgery. Five days prior to the first training day, subjects were placed on an 85% food-deprivation schedule, maintained throughout the experiment. On Days 1 and 2, each subject was handled for approximately 2 min. On Days 3 to 5, each subject received, in addition to handling, 3 min exploration and was fed 10 45-mg Noyes pellets in the home cage. Home-cage feeding was adjusted in all stages of the experiment to maintain 85% deprivation levels.

Training

Following pretraining, a 2 by 2 between-subjects by 4 within-subjects design was used combining lesioned (L) and nonlesioned (NL) animals randomly assigned to either large (LR) or small reinforcement magnitude (SR) given on all trials with periodic increases in delay of food reward: 0 to 15 to 30 to 45 sec.

The lesioned/large-reinforcement (L-LR; $n=8$) and the nonlesioned/large-reinforcement (NL-LR; $n=6$) groups were reinforced with 12 pellets on all trials; the lesioned/small-reinforcement (L-SR; $n=8$) and the nonlesioned/small-reinforcement (NL-SR; $n=5$) groups were reinforced with 2 pellets on all trials. A trial was initiated by placing the subject in the startbox. After a 3-sec orientation, the experimenter raised the start door to allow the animal to enter the runway. The animal, confined in the goal area, was allowed access to the pellets, with the lid of the food cup being raised after the appropriate delay. The subject was removed from the goal area immediately after consuming the reinforcement.

Each subject received four trials per day for 17 days. Shifts in delay of food reward occurred on Days 6, 10, and 14 for 16 trials each.

The subjects were run in squads of four animals. Each subject received trial n before any subject received trial $n+1$, resulting in an intertrial interval of approximately 5 min. The subjects were tested in the same sequence every day.

RESULTS

Histology

Figure 1 illustrates maximal cross-sectional damage of representative amygdala lesions. All lesions included bilateral damage to the amygdaloid complex and ventral pyriform cortex. Total cross-sectional damage extended, in most cases, throughout the entire anterior-posterior extent of the cortical, basal, and central, but not medial, nuclei. The damage corresponded to areas represented on Plates 30 to 35 of the stereotaxic atlas by König and Klippel (1963).

Run Speeds

All of the results are based on transformation of $\log_{10} (1/\bar{x} + 1)$ of the times recorded in the run segment of the alleyway, since differences were gen-

Figure 1. Camera lucida drawing of representative amygdala lesions.

erally larger and more reliable in the run measure. The results will be discussed in relation to the two hypotheses.

The first hypothesis was that the collapse effect would occur earlier in time for animals with amygdala lesions than for animals without. That is, with only moderate training (less than 50 trials), the differences in speeds between large and small magnitude of reinforcement for lesioned animals would be less than the differences in speeds between similar magnitudes for nonlesioned animals.

In Figure 2, by the 5th day of training (i.e., 20 trials), the differences between L-LR and L-SR were negligible ($t = .56$, $df = 14$, $p > .05$); however, the differences between NL-LR and NL-SR were reliable ($t = 2.37$, $df = 9$, $p < .05$). An analysis of variance for the 5th day of training indicated that the effects of lesion, magnitude, and their interaction were reliable (smallest $F = 4.68$, $df = 1,23$, $p < .05$). Subsequent Newman-Keuls tests indicated that Group NL-SR differed reliably from all other groups ($p < .05$); Groups L-LR, NL-LR, and L-SR did not differ reliably among themselves.

The second hypothesis was that amygdalectomized animals would be less responsive to the periodic increases in delay of food reward than nonlesioned animals. That is, running speeds of amygdalectomized animals would be faster than those of nonlesioned animals following these increases in delay.

At the 15-sec interval, lesioned rats ran faster than nonlesioned rats for the small magnitude of reinforcement (L-SR vs. NL-SR) but not the large magnitude of reinforcement (L-LR vs. NL-LR); the performance of Group NL-LR was not affected by the shorter, 15-sec delay. With longer delays, however, both lesion groups were clearly running faster than the nonlesion groups (see Figure 2).

With 15-sec delay of food reward, the effects of lesion, magnitude, and their interaction were reliable

(smallest $F = 5.12$, $df = 1,23$, $p < .05$). The interaction was due to Group NL-SR showing a large drop in running speeds following the shift; however, this decrement began to dissipate somewhat later during the same shift, as evidenced by a reliable days by lesion effect ($F = 4.69$, $df = 3,69$, $p < .05$). No other within-subjects differences were reliable.

For the 30- and 45-sec delay intervals, the effect of lesion was reliable (the smaller of both lesion F s = 5.79, $df = 1,23$, $p < .05$); magnitude and its interaction with lesion were not reliable. The running speeds of both lesion groups combined, L-LR and L-SR, were faster than their nonlesioned counterparts, NL-LR and NL-SR. A reliable days effect occurred in both shifts (the smaller of both F s = 3.16, $df = 3,69$, $p < .05$), meaning all groups generally slowed down following these longer delays. No other within-subjects differences were reliable.

DISCUSSION

In a number of studies examining the food-motivated behavior of amygdalectomized animals in the alleyway, an emerging empirical base is beginning to develop. The results of this experiment indicated that animals with amygdala lesions exhibited the collapse effect when given only a moderate amount of training; likewise, their running speeds were faster than those of nonlesioned animals following periodic increases in the delay of food reward, more so for the longer (30- and 45-sec) delay intervals. Other researchers have reported that amygdalectomized rats have failed to alter running speeds in an alleyway when magnitude of reinforcement was either decreased or increased (Kemble & Beckman, 1970), or when reinforcement was omitted from the first goal-

Figure 2. Run speeds of groups given 0-, 15-, 30-, and 45-sec goalbox delay (L = lesion, NL = nonlesion, LR = large reinforcement, SR = small reinforcement).

box of a double alleyway (Henke & Maxwell, 1973), and that resistance to extinction was greater for amygdalotomized than for control animals (Kemble & Beckman, 1970). These results, then, suggest that amygdalotomized animals are less responsive to *original* contingencies of reinforcement than non-lesioned animals. This is a new finding insofar as food reinforcement is concerned. The lesioned animals were also less responsive to *changes* in contingencies of reinforcement, a finding which confirms antecedent theory (Richardson, 1973). The results obtained with original contingencies of reinforcement, however, are viewed as being predominantly consonant with Richardson's theory.

The findings of the lesion experiments discussed above suggest that the normal function of the amygdala is involved in the interpretation and integration of reinforcing stimuli. The involvement of the amygdala in the processing of information associated with reinforcement was demonstrated electrophysiologically by Norton (1970), who reported an increase in the amplitude of the electrical activity of the amygdala following reinforced barpresses by rats but not following nonreinforced responses. Lesse (1957) found that the electrical activity of the amygdalas of rats assumed a high-amplitude 40- to 45-Hz waveform following electric shock. In fact, the shift to 40 and 45 Hz could be classically conditioned to a buzzer by pairing the buzzer with electric shock. Thus, it appears that the amygdaloid complex is involved with the successful behavioral adaptation to the positive and negative emotional aspects of various reinforcement contingencies.

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