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Reactions to DRL schedule change in rats with septal damage*

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Normal rats and rats with septal lesions were given 45 days of DRL training on one of three different schedule sequences: DRL 10-20-10, 20-30-20, and 30-40-30, with 15 days of training on each schedule value. Interpolated training on a DRL schedule having a 10-sec longer delay requirement than the initial schedule did not result in further response suppression than that generated by the initial schedule in the case of rats with septal lesions. However, normal rats showed lower responding on the second exposure of DRL 10 and DRL 20 but not on DRL 30. In general, increasing the DRL delay requirement by 10 sec resulted in decreased responding. However, the magnitude of the decrease was a function of the relative difference in schedule requirement only in the case of rats with septal damage. Decreasing the delay by 10 sec led to increased responding in both normal and operated rats, with the magnitude of the increase being a function of the relative difference in delay requirement.

It was reported by Ellen and Aitken (1973) that the response output of normal rats and rats with septal lesions that were required to track a DRL schedule sequence became progressively lower as the delay requirement increased. This inverse relationship described, in general, the response output-DRL delay function of all animals and is consistent with the work of others (Staddon, 1965). However, on each of the five schedules, normal rats made fewer responses and obtained more reinforcements than did operated rats.

An interesting characteristic of the response output-DRL delay function was that a 10-sec difference in the DRL delay requirement did not produce equivalent changes in response output across the range of DRL values. In fact, a 10-sec difference in delay requirement resulted in a greater change in responding at short DRL delay values (i.e., 10 to 20) than at intermediate and long delay values (i.e., 20 to 30 and 40 to 50). This suggests that the magnitude of increase or decrease in responding which results from a change of 10 sec in the delay requirement may be a function of the responding generated by the preceding schedule value.

A methodological weakness in the experimental design used by Ellen and Aitken (1973) limits the generality of this conclusion. In their study, the five different schedule values were presented to all rats using a single random sequence. Since a counterbalanced design was not used, the uncontrolled factors of schedule order (the DRL schedule prior to change being short, intermediate, or long in delay requirement) confounded the obtained results. The present experiment counterbalanced the presentation of the different DRL schedule values so that the effects of schedule order and direction of schedule change could be determined.

METHOD

Subjects

The Ss were 30 male Long-Evans hooded rats that weighed approximately 260 g at surgery.

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Apparatus

Five Scientific Prototype operant chambers were linked to the IBM 1800 data acquisition and control system (Ellen, DeLoache, & Bonds, 1972). The computer was programmed to deliver the different schedule values and recorded responses and reinforcements.

Procedure

Before the start of training, all rats were randomly grouped into six squads of five rats each. The Ss were then trained to press a lever for a 45-mg Noyes food pellet. Pretraining continued until each rat received a minimum of 150 reinforcements. On the day following the completion of pretraining, the Ss were given 10 days of CRF. During this time, each rat was required to obtain 150 reinforcements in 50 min or less. At the end of each daily session, all rats were returned to the home cages and allowed access for 50 min to 20 g of ground laboratory chow, which was made into a wet mash by adding water. Since all Ss were only fed for 50 min immediately after each daily session, the rats were 22 h food-deprived at the start of each test session.

Following the completion of the first 5 days of CRF, the six squads were divided into three groups. Each group consisted of two squads of rats. One of the squads from each of the three groups became the operated squad, while the other remained the control squad (see Surgery). Beginning on the day following the operation, both normal rats and rats with septal lesions were given another 5 days of CRF. Then, each group was started on one of the three different sequences of DRL schedules. A schematic of the design used is shown in Table 1. Briefly, the sequence of DRL schedules for Delay Group I was DRL 10-20-10; for Delay Group II, 20-30-20; and for Delay Group III, 30-40-30.

Surgery

During the preoperative procedure, the 15 operated animals were anesthetized with an intraperitoneal injection of a solution which contained 40 mg/kg of sodium pentobarbital (Nembutal) and .5 cc (.2 mg) of atropine sulfate. Local bleeding was controlled by infiltrating the cut tissue with 2% lidocaine hydrochloride which contained epinephrine (Xylocaine). The head of each animal was held in place by using a Baltimore Instrument Co. stereotaxic instrument. After an incision was made to expose the skull, a hole was drilled through the bone so that an electrode could be lowered into the brain of the S. A midline septal lesion was placed 7.8 mm anterior to, and 6.5 mm above, the intraural line (Pellegrino & Cushman, 1967). An anodal electrolytic lesion (2.0 mA for 15 sec) was made by using a Stoelting 58040 electrolytic lesion maker. Following the operation, each operated animal received .2 cc of procaine

Table 1
Experimental Design

Delay Group*	DRL Schedule Value (15 Days Each)		
I	−10 (1)	−20	−10 (2)
II	−20 (1)	−30	−20 (2)
III	−30 (1)	−40	−30 (2)

**There are five normal and five operated rats in each delay group.*

penicillin G (60,000 units, Duracillin) intramuscularly.

Histology

At the end of the experiment, the rats with lesions were sacrificed with a lethal dose of Nembutal, perfused intracardially with Ringer's solution, and the brains were fixed in Formalin. Frozen sections 45 microns thick were cut, and photomicrographs were made from wet slides (Powell, 1964).

RESULTS

Anatomical Findings

Reconstructions of the lesions of the operated rats for each of the three delay groups are shown in Fig. 1. For the most part, destruction of brain tissue was localized to the septal area, with little or no damage occurring to bordering brain structures. For all brains, the greatest destruction was in medial septum, accompanied by a partial destruction of lateral septum. Additionally, the ventral extent of the lesion went from medial septum through the anterior commissure and bordered on the medial preoptic area, in that there was some destruction of these two brain structures. Finally, the range of destruction extended anteriorally to the medial parolfactorial area and posteriorly to the anterior commissure. In passing, it may be of interest to mention that there was no correlation between the amount of septal tissue ablated and the absence or presence and/or duration of a hyperreactivity to handling. In any event, all evidence of either emotionality or hyperreactivity to handling had disappeared by the fifth day on DRL (i.e., 10 days after surgery).

Behavioral Findings

A separate three-way analysis of variance was computed for each of the three DRL delay groups. Unless otherwise indicated, the following design was used: Lesion (normal and operated rats) by Schedule (10-20-10; 20-30-20; 30-40-30) by Days (15 days). In all cases, the Newman-Keuls procedure was used to make multiple comparisons of response and reinforcement means (Winer, 1962).

Response Data

Responses for normal and septal rats for Delay Group I are shown in Fig. 2A. The response output for normal rats was not significantly different from that of the operated rats ($F = 2.94$, $p > .05$). There was a significant schedule effect ($F = 124.89$, $p < .05$), in that there was a higher response output on DRL 10 than on DRL 20. The significant Lesion by Schedule interaction ($F = 26.81$, $p < .01$) suggests that the level of responding on each of the schedules was influenced by the lesion. Additionally, there was a significant days effect ($F = 3.67$, $p < .05$), with the normal rats showing a further decrease in responding on the interpolated schedule. Finally, only the normal rats showed a significant decrease in response rate ($p < .01$) when DRL 10 (1) was compared with DRL 10 (2), while the operated rats did not show any change ($p > .05$) between the first and second exposure to DRL 10.

The level of responding for Delay Group II rats is shown in Fig. 2B. Throughout training, there was a significant lesion effect ($F = 5.32$, $p < .05$), i.e., a lower response output for normal rats as compared with

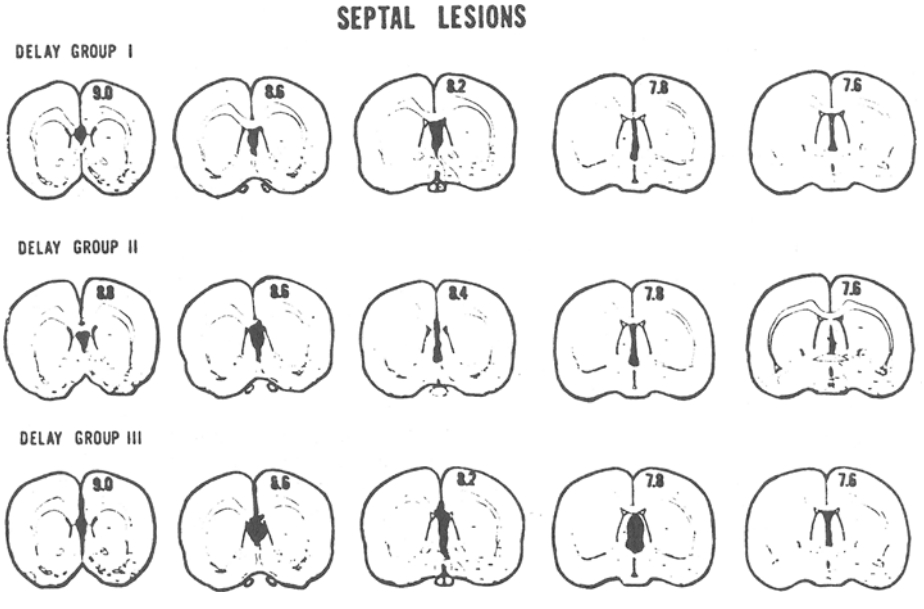

Fig. 1. Reconstructions of composite septal lesions for each of the three delay groups. Each section is identified with the appropriate AP stereotaxic coordinates taken from Pellegrino and Cushman (1967) atlas.

operated animals over the entire course of the experiment. Additionally, there was a significant schedule effect ($F = 129.58$, $p < .01$), i.e., response output on DRL 30 being lower than on DRL 20, and a significant Lesion by Schedule interaction ($F = 51.43$, $p < .01$). Again, only the normal rats showed a significant decrease in responding ($p < .01$) on the second exposure to DRL 20.

Although there was a significant lesion effect ($F = 7.92$, $p < .05$) in Delay Group III (see Fig. 2C), there was not a significant schedule effect ($F = 2.31$, $p > .05$). Two other second-order interactions were significant, Lesion by Schedule ($F = 9.24$, $p < .05$) and Days by Schedule ($F = 4.32$, $p < .05$). For neither normal nor operated rats did the interpolated training result in additional response suppression on DRL 30 (2) as compared to DRL 30 (1).

Reinforcement Data

It is clear from Fig. 3A that the normal rats of Group I obtained more reinforcements than did the septal rats ($F = 47.92$, $p < .01$). There was a significant schedule effect ($F = 130.27$, $p < .01$), as well as a significant Lesion by Schedule interaction ($F = 22.81$, $p < .01$), indicating that the number of reinforcements obtained depended on the lesion. In addition, there was a significant days effect ($F = 3.69$, $p < .01$), suggesting

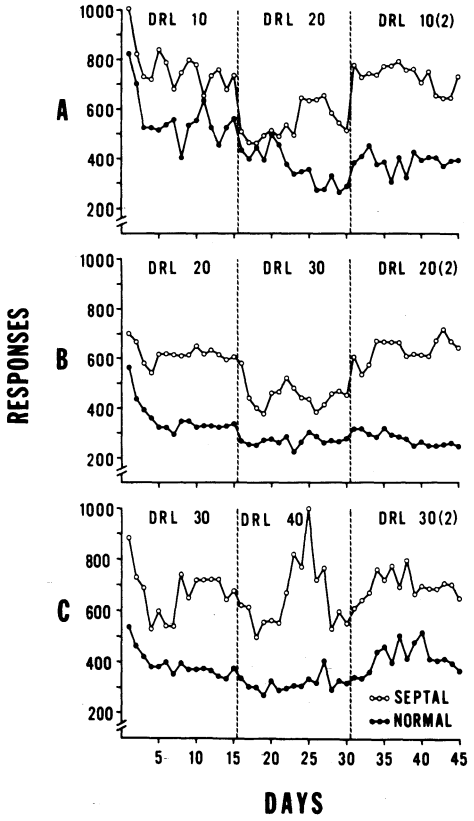

Fig. 2. Responses emitted by normal and operated rats of Delay Group I (A), Delay Group II (B), and Delay Group III (C).

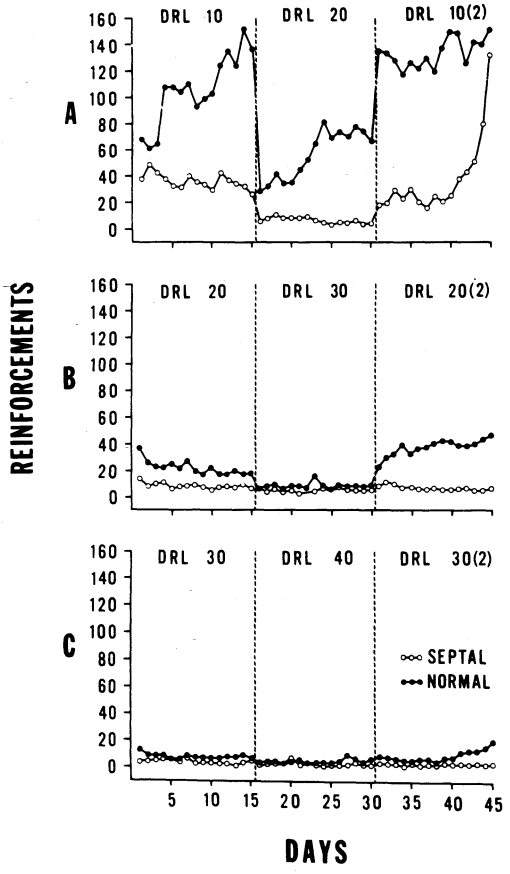

Fig. 3. Reinforcements obtained by normal and operated rats of Delay Group I (A), Delay Group II (B), and Delay Group III (C).

that more reinforcements were obtained at the end than at the start of each schedule. Additionally, normal rats obtained significantly more reinforcements ($p < .01$) on DRL 10 (2) than on DRL 10 (1), while operated animals did not show any change ($p > .05$).

Figure 3B shows the number of reinforcements obtained by Delay Group II rats. In addition to a significant lesion effect ($F = 6.49$, $p < .05$), there was a significant Lesion by Schedule interaction ($F = 333.46$, $p < .01$). As with the previous group, interpolated training resulted in an increase in reinforcements for normal ($p < .01$) but not for operated rats ($p > .05$).

Figure 3C shows the reinforcement data for Delay Group III. There were no significant differences between normal and operated animals with respect to either lesion ($F < 1$, $p > .05$), schedule ($F = 1.06$, $p > .05$), or days ($F < 1$, $p > .05$).

Reaction to Schedule Change

The percentage change in responding when there was a change of 10 sec at each of the starting values is shown in Fig. 4. These data compare the responding on the last 3 days on each schedule before the change with the first 3 days following a schedule change. An increase of

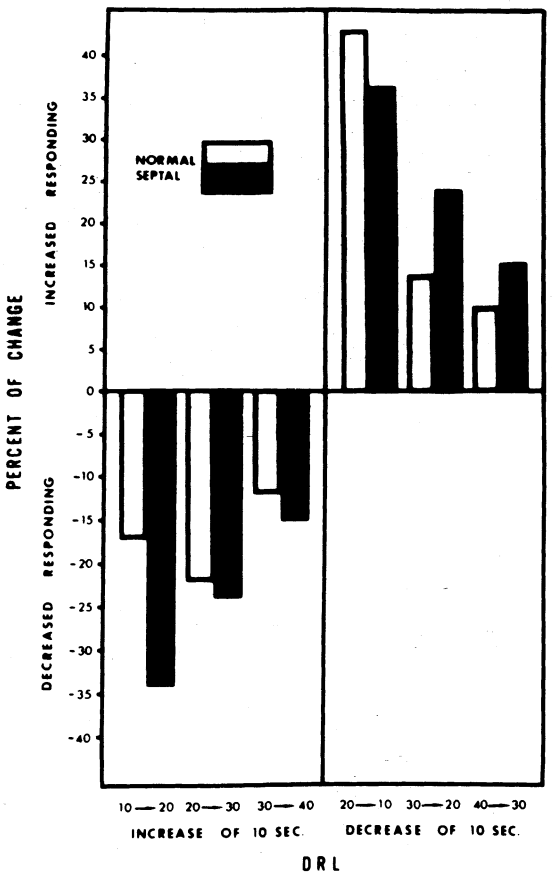

Fig. 4. Percentage of change in responding to increases and decreases of 10 sec in the delay requirement of the initial DRL schedule value.

10 sec in the delay requirement resulted in a decrease in response output for both normal and operated rats. A Friedman two-way analysis of variance by ranks showed that there was a significant difference with respect to the percentage of decrease in responding for the three starting values of DRL 10 to DRL 20, DRL 20 to DRL 30, and DRL 30 to DRL 40 for rats with septal damage ($X_r^2 = 7.80$, $p < .05$) but not for normal rats ($X_r^2 = 4.23$, $p > .05$). For the former group, the greatest percentage of decrease in responding occurred as a result of a 10-sec change from DRL 10 to DRL 20, and the smallest occurred when DRL 30 was increased to DRL 40. The change in responding for normal rats did not show a systematic difference as a function of schedule values. Additionally, when DRL 10 was the starting value, a 10-sec increase in delay requirement resulted in a significantly larger percentage decrease in responding for operated rats than for normal rats. When this comparison was made using the Mann-Whitney U test, it was significant beyond the level of chance ($U = 0$, $p < .05$). An increase of 10 sec in the delay requirement of the other two DRL schedule values of 20 and 30 did not result in a significant difference ($U = 10$, $p > .05$) between normal and operated animals (Siegel, 1956).

When there was a decrease of 10 sec in the DRL delay requirement, both normal and operated animals showed an increased responding to the same degree for each of the three schedule changes of DRL 20 to DRL 10, DRL 30 to DRL 20, and DRL 40 to DRL 30 (in all cases, $U > 1$, $p > .05$). It will be noted that in contrast to the previous data, now the extent of the percentage of increase in responding varies as a direct function of the initial DRL schedule value ($X_r^2 = 11.08$, $p < .05$) for both groups. In other words, the greatest percentage of increase in responding occurred when the schedule value was decreased from DRL 20 to DRL 10, and the smallest percentage of increase in responding occurred when DRL 40 was decreased to DRL 30.

DISCUSSION

A number of interesting observations stand out in this study. First, the interpolated training on a DRL schedule having a 10-sec longer delay requirement than the initial DRL schedule led to a lower response output on the initial schedule after it was reinstated in the case of normal rats, but not for rats with septal lesions. The interpolated training was only effective in facilitating response suppression for normal rats when the initial DRL schedule had a delay requirement of 10 or 20 sec, but not when it was 30 sec. That is, interpolated training on DRL 40 did not produce any additional response suppression on DRL 30. These observations lead to several conclusions. It would appear that there are no cumulative effects of training which facilitate response suppression within the 10- to 30-sec range for rats with septal lesions. Thus, these findings point to the relative insensitivity of septal rats to the influence of training effects or changes in schedule requirement which could draw attention to the relevant stimulus dimensions correlated with the reinforced delay and thus facilitate response suppression on a DRL schedule.

Additionally, these data also suggest that the level of responding is not determined simply by the loss of some hypothetical response suppression mechanism (McCleary, 1961). That is, although septal rats stabilize responding at a particular level on a given schedule, this level is not invariant from one DRL schedule to the next. In Delay Groups I and II, both septal and normal animals showed less responding on the second schedule, which had a longer delay requirement than did the initial schedule.

In interpreting the results of this experiment, it is important to consider the various aspects of DRL schedules which may play a role in determining response output. One such factor is simply the density of reinforcement which is generated by the schedule. With increases in DRL delay, there is a decrease in reinforcement density as well as a decrease in response rate. However, since both the level of responding and the reinforcement density that results is not under explicit experimental control, it is impossible to assess the effect

of one variable on the other in a DRL schedule. It should be noted, however, that this same relationship between delay requirement and reinforcement density and response rate is not unique to DRL schedules. A similar relationship is also found with VI schedules. However, because VI schedules typically produce a sufficiently high rate of response, all of the reinforcements that have been programmed are generally delivered. This means that density of reinforcement can be independently manipulated and resultant rate changes thereby observed. Catania and Reynolds (1968) have found that, as the VI interval is increased, thereby decreasing reinforcement density, there is in fact a resultant decrease in response rate. Thus, it is clear that reinforcement density in and of itself can affect response rate.

In addition to the effects of reinforcement density on the level of responding, there is still another factor which is related to the DRL contingency. One of Anger's (1956) original observations on the responding of normal rats on a DRL schedule was that the "low rates" are in part due to the fact that only a restricted class of responses that fulfill the DRL delay requirement are reinforced, while all others that do not satisfy the DRL contingency are not reinforced and subsequently become extinguished. In an unpublished study, Richardson (1972) has compared the DRL and VI performance of rats and pigeons under conditions of equal reinforcement density and interreinforcement times by using a yoked control procedure. He found that for all Ss, the level of responding was lower on DRL than on VI, thus supporting the conclusion that the DRL contingency (i.e., differential reinforcement of particular IRTs), quite apart from the reinforcement density, leads to response suppression.

These considerations thus clarify the observations of the DRL performances described in the present study. It will be recalled that when the delay requirement was increased by 10 sec from DRL 10 to DRL 20 for Delay Group I or from DRL 20 to DRL 30 for Delay Group II, rats with septal lesions showed an immediate decrease in responding with the magnitude of the decrease being a function of the relative difference in delay requirement. More importantly, the response rate for these rats did not change throughout the entire 15 days on the interpolated schedules. It will also be recalled that the interpolated training did not lead to further response suppression when the initial DRL 10 and DRL 20 schedules were reinitiated. Normal rats, in contrast, in addition to showing an immediate decrease in responding when there was a schedule change to a longer delay requirement, showed a further decrease in their level of responding during the interpolated DRL 20 schedule. In addition, they also showed further response suppression on DRL 10 and DRL 20 following the interpolated training. Thus, it would appear that for animals with septal damage, the DRL contingency itself has no response-suppressing effects on the rate generated by DRL 10 or 20. For normal rats, however, it would appear that the DRL contingency produces a decrease in responding quite apart from whatever decrease results from the schedule change. This latter decrease in responding most likely results from the difference in reinforcements generated by the different schedules. In addition, even for normal rats, there is an upper limit beyond which responding cannot be further suppressed. This upper limit would seem to be determined by the extent to which the change in reinforcement density resulting from a schedule change is discriminable, since normal rats did not show any further response suppression on DRL 30 after interpolated training on DRL 40 (Delay Group III), nor was there any decrease in responding during the interpolated DRL 30 training (Delay Group II).

The second set of observations that are of particular interest are those concerned with the reaction to schedule change itself. While the preceding discussion focused on the level of stabilized responding after extended training on a given schedule, the observations to be considered now deal with the behavior recorded within the first 3 days after the schedule change was initiated. Three important facts stand out: (1) Increasing the DRL requirement by 10 sec produces a decrease in responding, while decreasing the delay requirement by the same amount produces an increase in responding for both normal and operated rats. (2) The magnitude of the decrease in responding generated by an increase in delay varied as a function of the relative difference in schedule values *only* for operated rats. For normal rats, on the other hand, the magnitude of decrease was the same regardless of whether the change was from DRL 10 to DRL 20 or from DRL 30 to DRL 40. (3) When the schedule requirement was decreased by 10 sec, the increase in responding generated by the decrease in delay was a function of the relative difference in schedule value for *both* normal and septal rats.

These data can likewise be accounted for in terms of the differential influence of the DRL schedule contingency on normal and operated animals. To the extent that the schedule contingency has also produced a suppressive effect on responding, then the effects of reducing the reinforcement density, which as we have already seen decreases responding, will be less since the responding is already suppressed. In other words, on the schedules where the contingency has already produced a suppressive effect on responding, the decrease in reinforcement can only add little more to this suppressive effect. In the case of normal animals, the DRL contingency itself has had a suppressive influence on responding. A decrease in reinforcement adds little more suppressive influence than does the DRL contingency itself. However, in the case of septal animals, the contingency produces little or no suppression on responding, and hence any discriminable change in density has major effects. Conversely when there is a decrease in schedule requirement leading to an

increase in reinforcement density, responding is activated. The degree of activation of responding depends, however, on the performance generated by the difference in schedule value, with the larger relative difference being more effective.

In short, these data suggest that DRL responding by rats with septal lesions is controlled for the most part by the reinforcement density generated by the schedule, since effects due to the schedule contingency alone appear to be absent. In other words, performance of these animals is more like VI than DRL performance. This would suggest that the lesion has destroyed the suppressive control on behavior that results from the differential reinforcement of selected IRTs.

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