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Stimulus Change Influences Escape Performance: Deficits Induced by Uncontrollable Stress and by Haloperidol[1]

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ANISMAN, H. AND R. M. ZACHARKO. *Stimulus change influences escape performance: Deficits induced by uncontrollable stress and by haloperidol.* PHARMAC. BIOCHEM. BEHAV. 17(2) 263–269, 1982.—Exposure to uncontrollable foot-shock or treatment with haloperidol was found to disrupt subsequent escape behavior. Performance among naive mice, as well as mice that had been exposed to inescapable shock or treated with haloperidol could be enhanced by either interrupting the shock train during escape testing or by presentation of a novel stimulus. The effectiveness of these treatments were dependent on the time at which the change in stimulation occurred. That is, shock interruption or cue presentation just prior to escape being possible enhanced performance, but the same manipulation several seconds prior to escape being possible had only a limited effect. In addition, the time of cue termination also influenced escape behavior. When cue offset coincided with or followed successful escape a performance enhancement was evident, but when cue offset occurred several seconds prior to escape, performance was not affected. It was suggested that inescapable shock and haloperidol treatment hinder performance by disrupting response maintenance. Shock interruption and novel cue presentation minimize disturbances of escape performance by altering the course of the decline of shock-elicited activity.

Escape performance Stress Haloperidol Foot-shock

EXPOSURE to uncontrollable shock has repeatedly been shown to induce pronounced deficits of later escape behavior [2, 10, 17]. Whereas some investigators have attributed the performance disruption to cognitive changes (i.e., learned helplessness) provoked by the uncontrollable stress [10], others have contended that difficulties in response initiation and maintenance are responsible for the behavioral disturbance [2, 5, 17]. These motor disturbances are thought to reflect either learned competing response tendencies [2, 7, 8] or are a consequence of the depletion of brain catecholamines engendered by the inescapable shock [4, 14, 16, 17].

Discrete analyses of the behavior of animals during exposure to inescapable shock have revealed a characteristic profile of shock-elicited activity, i.e., activity measured during shock itself [2]. Upon shock inception mice exhibited a transient (2–3 sec) period of vigorous responding, followed by a period of limited active responding. Among mice that had previously been exposed to inescapable shock the period of excitation was truncated, and response immobility became particularly pronounced. The transient excitation seen upon shock inception is thought to favor adequate escape behavior in a task where escape was possible soon after shock onset. However, if the response could not be completed quickly, the depression of motor activity would favor poor escape behavior, particularly among animals that had previously been exposed to inescapable shock.

The pattern of shock-elicited activity, and hence escape behavior, could be modified through a manipulation as simple as briefly interrupting the shock train [2]. Moreover, casual observation of mice in this laboratory has revealed that any number of extraneous cues would disrupt the stereotyped immobility evident during long-duration shock presentations and would provoke efficient escape behavior. One purpose of the present investigation was to document the effects of a novel cue on escape behavior among experimentally naive mice and among mice that had been exposed to uncontrollable shock. The second purpose of the current investigation was to determine whether shock interruption or presentation of a novel stimulus would alter the escape deficits introduced by the dopamine receptor blocker, haloperidol. It has been suggested [5] that the deficits in response initiation and maintenance induced by uncontrollable shock are reminiscent of the motoric effects induced by haloperidol. As such, manipulations that alter the effects of inescapable shock would be expected to alter the behavioral consequences produced by treatment with haloperidol.

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It might be added that the results of the present investigation have direct bearing on a second area of research. In particular, it has been proposed that DA neuronal activity may be one factor that mediates reward processes [18,19]. Indeed, it has been shown that the DA receptor blocker, pimozide, will produce behavioral changes in an appetitive task that parallel those seen during extinction of an operant response in the absence of any drug treatment [19]. However, it is possible that the disturbances of performance induced by DA receptor blockers are a result of deficits in response initiation and maintenance [15] or effects on a sensory motor interface [13]. It was shown, for example, that the presentation of noise that accompanied the insertion of a lever in a food motivated operant task largely attenuated the disruptive effects produced by DA receptor blockade [15]. The present investigation served to determine whether the effects of haloperidol on an aversively motivated behavior are modifiable by alterations of sensory stimulation during the course of shock presentations.

EXPERIMENTS 1 AND 2

As indicated earlier both inescapable shock and treatment with haloperidol will disrupt performance in an escape task, provided that the response required for successful performance entails vigorous or protracted responding [2]. We previously reported if shock was briefly interrupted (1 sec), the pattern of shock-elicited activity was altered and escape behavior was enhanced among mice that had previously been exposed to uncontrollable shock. Experiments 1 and 2 were undertaken to confirm and extend our previous findings, and to determine whether shock interruption would influence the performance of mice that had received treatment with haloperidol.

METHOD

Subjects

Experiments 1 and 2 involved 80 and 120 CD-1 mice obtained from the Canadian Breeding Farms at 55–60 days of age. Mice were housed in groups of 5 in standard polypropylene cages, and were acclimatized to the laboratory for 10–17 days prior to being used for experimental purposes. Mice were permitted ad lib access to food and water at all times.

Apparatus

The apparatus was the same as that described previously [5]. Inescapable shock was delivered in four identical black Plexiglas chambers which measured 30.0×14.0×15.0 cm. The grid floor of each chamber consisted of 0.32-cm stainless-steel rods spaced 1.0 cm apart, connected in series with neon bulbs, through which shock (150 μA, AC, 60 Hz) could be delivered from a 3000-V source. The walls of the chamber were lined with stainless-steel plates and connected in series with the grid floor.

Escape training was conducted in four identical Plexiglas shuttle-boxes (26.4×9.0×15.5 cm) whose grid floor, wiring, and shock sources were the same as those of the preshock boxes. An opaque disc (2 cm diameter), situated on each end wall 12 cm above the grid floor, could be illuminated by a 24 W lamp situated behind each disc. A speaker mounted in the center of the roof of each chamber permitted presentation of an auditory stimulus. Each shuttle-box was divided into two compartments by a stainless-steel wall, partially made up of a solenoid-controlled horizontally movable stainless-steel gate. In the open gate position a stainless-steel hurdle 1.0 cm in height separated the compartments and a 7.0×7.7 cm space permitted access to the adjacent compartment. A photodetection system described previously [2] determined the position of animals in the shuttle-boxes. The shuttle-boxes were housed in sound-attenuated chambers.

Procedure

Mice of Experiment 1 were individually placed in the preshock chambers for a 1.1 hr period. During this time half the mice received 60 inescapable shock presentations (150 μA, 60 Hz, AC) of 6 sec duration at intervals of 1 min, while the remaining mice were not shocked. Following the initial session mice were individually housed until the time of testing which occurred 24 hr later. Mice of the preshock and nonpreshock groups were assigned to one of 4 treatment groups (n=10/group). All groups initially received 5 escape trials at intervals of 30 sec, in which escape was possible immediately upon presentation of the shock. A successful escape response simply entailed crossing the hurdle separating the two compartments of the test chamber. This was followed by 25 escape trials in which an escape delay procedure was employed (see [2]). For one group of mice escape was prevented for 6 sec after shock onset by keeping the gate separating the compartments closed. The gate was then opened permitting entry into the adjacent shock free compartment. Likewise, in the remaining groups escape was not possible immediately upon shock onset. For one of these groups shock was presented for 3 sec, interrupted for 1 sec, then presented for 3 sec, after which the gate separating the compartments of the shuttle-box was opened, permitting escape. Thus, as in the first group six seconds of shock was applied prior to gate opening, but the shock train was interrupted for a 1 sec period 3 sec after its initial onset. In another group shock was presented for 5 sec, interrupted for 1 sec, and then applied for 1 sec before the gate opened. Accordingly, in this group the amount of shock applied was the same as in the other groups, but shock interruption and gate opening were temporally closer to one another. In a final group shock was presented for 6 sec prior to a 1 sec interruption of shock. Gate opening in this group coincided with subsequent shock onset. In all conditions a trial was terminated and the gate closed when the animal crossed into the shock-free chamber. If an escape response was not completed within 24 sec of the gate being opened the trial was terminated.

In Experiment 2 mice were not exposed to inescapable shock, but rather received intraperitoneal (IP) injection of either haloperidol hydrochloride (0.0375 mg/kg or 0.075 mg/kg salt weight dissolved in 0.01 N HCl) or vehicle in a volume of 10 ml/kg. These doses were selected on the basis of previous experiments [3,5] which showed them to be the lowest doses that would reliably induce escape deficits using a 6 sec escape delay procedure. Following injection mice were placed individually in holding cages for 45 min after which they were tested in the escape task using one of the four procedures described in Experiment 1 (n=10/group).

RESULTS AND DISCUSSION

The escape latencies for each of the groups of Experiment 1 are shown in Fig. 1. Analysis of variance of the escape latencies revealed a significant interaction between Prior

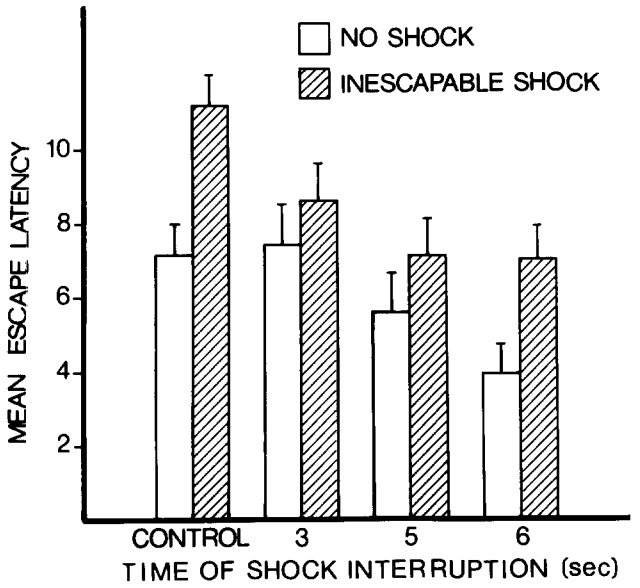

FIG. 1. Mean (±S.EM.) escape latencies among mice exposed to inescapable shock or no shock 24 hr earlier. During escape testing the shock train was either uninterrupted or was interrupted for 1 sec either 3, 5 or 6 sec after shock onset. (Latencies are calculated from the time of gate opening).

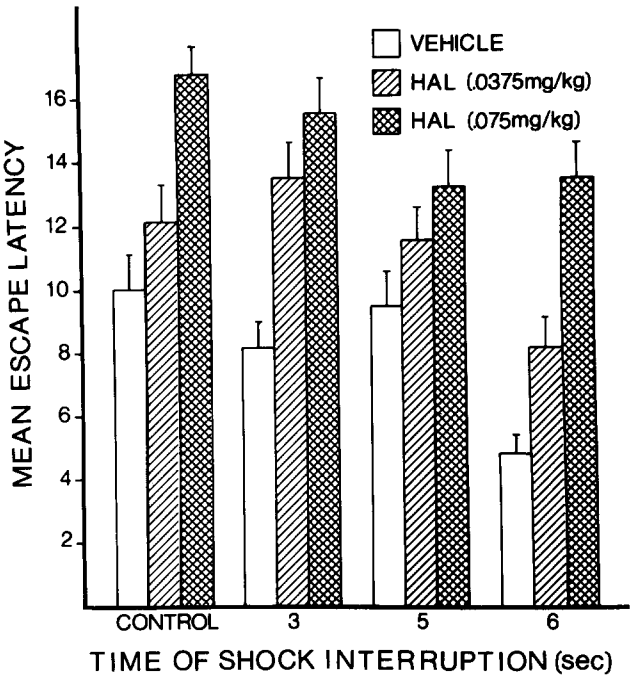

FIG. 2. Mean (±S.E.M.) escape latencies among mice treated with haloperidol (0.0375 or 0.075 mg/kg) or vehicle. During escape testing the shock train was either uninterrupted or was interrupted 3, 5 or 6 sec after shock onset. (Latencies are calculated from time of gate opening).

Shock Treatment and Blocks of Trials, $F(4,288)=5.40$, $p<0.01$. Pairwise comparisons between the treatment groups at each level of Blocks showed that the escape latencies of preshocked mice were longer than those of nonpreshocked mice throughout the last 3 trial blocks. The shock interruption procedure was also found to have a marginal effect on escape latencies, $F(3,72)=2.42$, $p=0.073$. Subsequent Newman-Keuls multiple comparisons revealed faster escape latencies among mice that had shock interrupted 6 sec after its initial onset than in the group in which shock had not been interrupted ($p<0.07$). Among animals that had been exposed to inescapable shock the escape latencies were shorter when shock was interrupted 5 or 6 sec after initial onset than in the noninterrupted condition ($p<0.05$).

As seen in Fig. 2, treatment with haloperidol influenced escape behavior in a task where escape was not possible at the time of shock onset, $F(2,108)=11.36$, $p<0.01$. Newman-Keuls multiple comparisons confirmed that both doses of haloperidol retarded escape latencies relative to saline treated animals. Moreover, latencies were longer among mice treated with the 0.075 mg/kg dose than among mice that received 0.0375 mg/kg of haloperidol. As in the case of Experiment 1, the shock interruption procedure modified escape performance, $F(3,108)=2.91$, $p<0.05$. Newman-Keuls multiple comparisons ($\alpha=0.05$) showed that only when shock was interrupted 6 sec after onset were escape latencies significantly shorter relative to mice that did not have shock interrupted.

Taken together, the results of Experiments 1 and 2 indicate that shock interruption will reduce the latency to escape from shock among naive mice, as well as mice previously exposed to inescapable shock or treated with haloperidol. Moreover, the effectiveness of this procedure was dependent on the time at which shock was interrupted. While marked response enhancements occurred when shock interruption coincided with gate opening, no detectable effects were evident when shock was interrupted several seconds prior to gate opening. Given that shock interruption results in a transient increase of shock-elicited activity [2], the results of the present investigation are consistent with the proposition that the motoric effects of the interruption procedure were responsible for the response enhancement. However, the possibility cannot be excluded that shock interruption acted as a signal for gate opening, thereby facilitating escape.

EXPERIMENTS 3—6

Having demonstrated that brief shock interruption would enhance escape performance in naive animals, as well as mice exposed to inescapable shock or treatment with haloperidol, Experiments 3 and 5 were undertaken to determine whether the presentation of novel stimulus would likewise alter escape behavior under these conditions. Maier *et al.* [11,12] reported that delaying offset of the CS used in avoidance/escape training would hinder escape behavior. Accordingly, Experiments 4 and 6 not only evaluated the effects of time of cue onset on escape performance, but also determined whether the time of cue offset would influence escape latencies.

METHOD

Experiments 3–6 involved 60, 80, 90 and 120 naive CD-1 mice, respectively. In Experiments 3 and 4 mice received either inescapable shock or no shock as described in Experiment 1 and were tested in the escape task 24 hr later using a 6 sec escape delay procedure. In one condition a compound cue (light plus buzzer) was presented at the time of shock

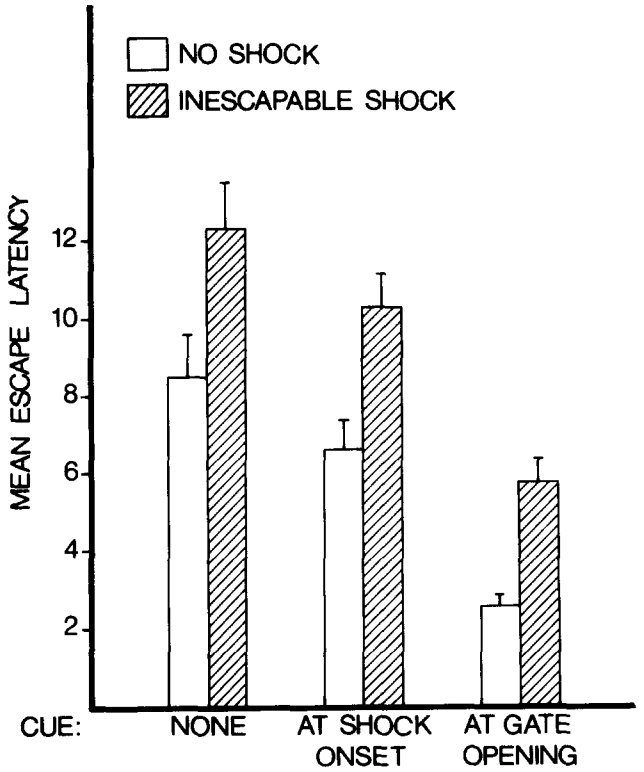

FIG. 3. Mean (±S.E.M.) escape latencies among mice exposed to inescapable shock or no shock 24 hr earlier. During escape testing a novel compound stimulus (light plus buzzer) was presented either at the time of shock onset, at the time of gate opening or not at all.

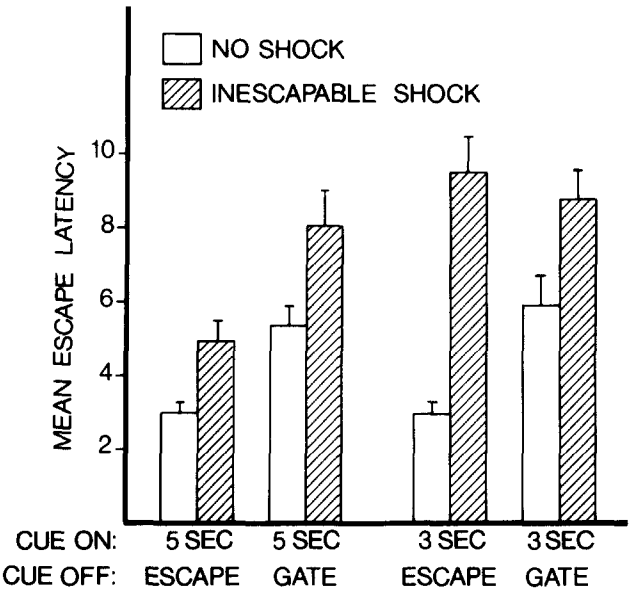

FIG. 4. Mean (±S.E.M.) escape latencies among mice exposed to inescapable shock or no shock 24 earlier. During escape testing a novel compound stimulus (light plus buzzer) was presented either 3 or 5 sec after shock onset, and terminated either with gate opening or escape.

onset and terminated with completion of an escape response. In a second condition the cue was presented at the time of gate opening and terminated with the escape response, while in the third condition the novel cue was not presented.

In Experiment 4 the novel cue was presented 3 sec after shock onset (3 sec before gate opening) for half the mice, while for the remaining mice the cue was presented 5 sec after shock onset (1 sec prior to gate opening). These groups were further subdivided, such that the cue either terminated with gate opening or with a successful escape response. If an escape response was not made within 24 sec the trial terminated.

In Experiments 5 and 6 mice did not receive the inescapable shock treatment, but rather received intraperitoneal injection of either haloperidol (0.0375 or 0.075 mg/kg) or vehicle in a volume of 10 ml/kg. Escape testing was conducted 45 min afterward. The escape testing procedure of Experiment 5 was the same as that of Experiment 3, whereas the escape procedure of Experiment 6 was identical to that of Experiment 4.

RESULTS AND DISCUSSION

The mean escape latencies for each of the groups of Experiment 3 are shown in Fig. 3. Analysis of variance of the escape latencies revealed that inescapable shock significantly retarded escape performance, $F(1,54)=5.84$, $p<0.05$, and that performance varied as a function of Cue Presentation, $F(2,54)=8.82$, $p<0.01$. Newman-Keuls multiple comparisons ($\alpha=0.05$) of the means involved in the Cue Presentation main effect revealed that relative to the no-cue group, presentation of the cue at the time of shock onset significantly reduced the escape latency. Moreover, cue presentation at the time of gate opening resulted in significantly shorter escape latencies than those observed in the remaining two groups.

As seen in Fig. 4 latencies to escape in Experiment 4 were retarded among mice that had previously been exposed to inescapable shock $F(1,72)=8.71$, $p<0.01$. The effectiveness of the cue in modifying escape performance varied as a function of the interaction between Time of Cue Presentation × Time of Cue Offset × Blocks of Trials, $F(4,288)=3.01$, $p<0.05$. Newman-Keuls multiple comparisons of the simple main effects ($\alpha=0.05$) conducted at each level of Blocks revealed that escape latencies were shorter when the cue was presented 5 sec after shock onset (1 sec prior to gate opening) and terminated with an escape response, than when the cue was presented 3 sec prior to gate opening and terminated with the response. When the cue was presented 5 sec after shock onset and terminated with gate opening, the reduced escape latencies were not evident relative to mice that had the cue presented at 3 sec after shock onset and terminated with gate opening. Indeed, the escape latencies of the former group were retarded relative to mice that had the cue presented 5 sec after shock onset and terminated with an escape response.

Analysis of variance of the escape latencies of Experiment 5 revealed that performance was influenced by the Haloperidol Treatment, $F(2,81)=3.36$, $p<0.05$ and by Cue Presentation $F(2,81)=9.65$, $p<0.01$ (see Fig. 5). Newman-Keuls multiple comparisons ($\alpha=0.05$) of these main effects confirmed that the 0.075 mg/kg dose of haloperidol disrupted escape behvior relative to vehicle treated mice, whereas the

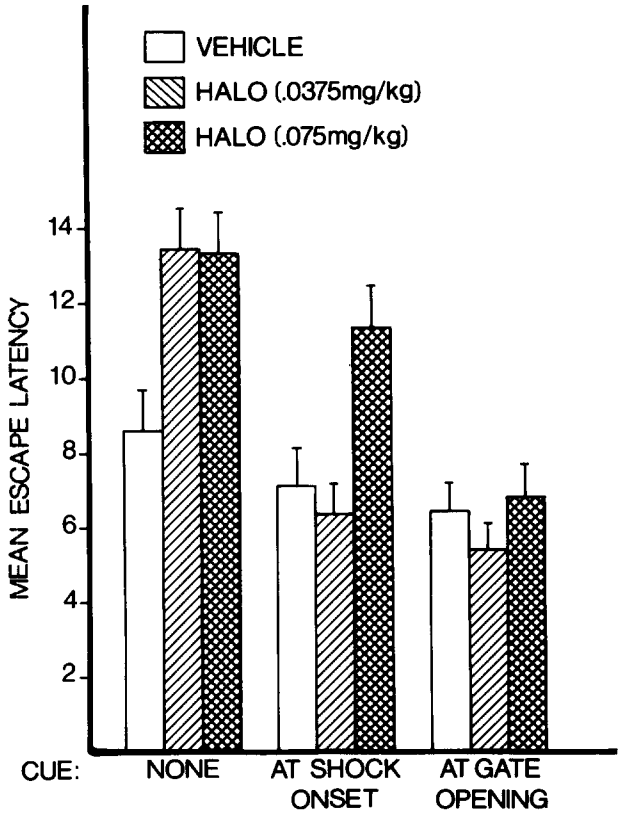

FIG. 5. Mean (±S.E.M.) escape latencies among mice treated with haloperidol (0.0375 or 0.075) or vehicle. During escape testing a novel compound stimulus (light plus buzzer) was presented either at the time of shock onset, at the time of gate opening or not at all.

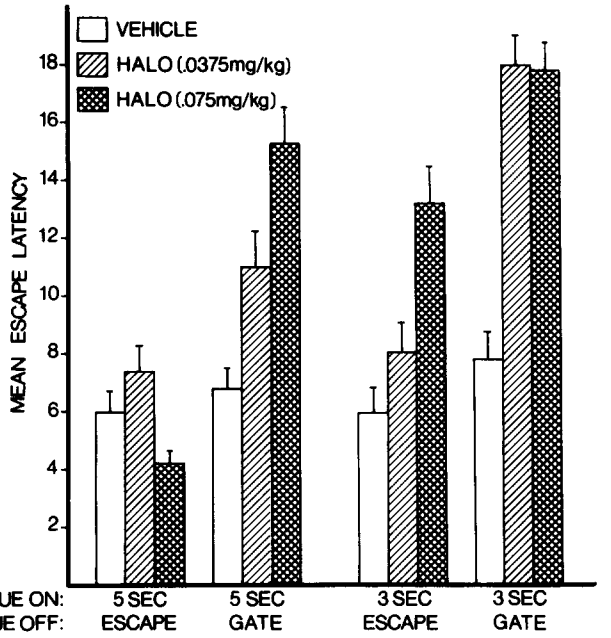

FIG. 6. Mean (±S.E.M.) escape latencies among mice treated with haloperidol (0.0375 or 0.075 mg/kg) or vehicle. During escape testing a novel compound stimulus (light plus tone) was presented either 3 or 5 sec after shock onset, and terminated either with gate opening or escape.

effect of the 0.0375 mg/kg dose was ineffective in this respect. Presentation of the cue either at the time of gate opening or upon shock presentation enhanced performance relative to mice that did not have the cue presented.

Although the Drug × Cue interraction did not reach satistical significance it is clear from Fig. 5, and confirmed by multiple comparisons that cue onset at the time of gate opening enhanced performance of mice treated with either dosage of haloperidol. In contrast, cue presentation at the time of shock onset only eliminated the disruptive influence of the lower haloperidol dose. Thus these data indicate that cue onset at the time of gate opening was, in fact, more effective in enhancing performance than cue presentation at the time of shock onset.

Analysis of variance of the escape latencies of Experiment 6 yielded a significant Drug × Cue Offset interaction, $F(2,08)=4.39$, $p<0.05$ (see Fig. 6). Newman-Keuls multiple comparisons ($\alpha=0.05$) were conducted between the means of the simple effects comprising this interaction. These comparisons revealed that the 0.075 mg/kg dose of haloperidol disrupted escape performance in all conditions except when cue onset occurred 5 sec after shock presentation and terminated with escape. The disruptive effect of the 0.0375 mg/kg dose was eliminated in those groups where cue offset accompanied the escape response.

Taken together, the results of Experiments 3–6 indicate that escape performance could be modified by the introduction of a novel cue. The effectiveness of this manipulation varied as a function of the time at which the cue was presented and terminated. In the case of the inescapable shock or treatment with haloperidol, the greatest performance enhancement was observed when cue onset immediately preceded gate opening, and this effect was further augmented when cue termination coincided with a successful escape response.

EXPERIMENT 7

The results of Experiments 3–6 indicated that the presentation of a novel cue during escape testing will effectively enhance escape performance among naive animals and among animals that had previously been exposed to inescapable shock. It seemed, however, that the effectiveness of the cue in enhancing performance varied as a function of the time at which the cue was presented, as well as when the cue was terminated. The briefer the interval between cue onset and gate opening, the more effective the cue was in provoking the response enhancement, provided that the cue terminated with an escape response. However, if the cue terminated at the time of gate opening, then no such enhancement was evident. It might be argued that cue offset provided information (feedback) as to the appropriateness of a response, thereby facilitating performance [12]. Alternatively, it is possible that the cue provoked response excitation, and the extent of the excitation was related to the duration of cue presentation. When the cue terminated at the time of gate opening the response enhancement diminished and consequently performance was not influenced as greatly as it was when cue offset occurred with an escape response.

If cue offset acted as a feedback stimulus for appropriate responding, then maximal benefit should be derived when the escape response and cue were contiguous. Delaying cue offset for several seconds after the escape response is completed should reduce the effectiveness of the feedback stimulus and hence minimize the performance facilitation. In contrast, if the effectiveness of the cue was derived from its capacity of enhancing active responding, then delaying cue offset should not limit the effects of the cue on escape performance. Experiment 7 assessed the effects of response-contingent and delayed cue offset on escape behavior of mice previously exposed to inescapable shock.

METHOD

Thirty naive male CD-1 mice were exposed to 60 inescapable shocks as described in Experiment 1. Twenty-four hours afterward mice were tested in the shuttle escape task using a 6 sec delay procedure. In one condition (n=10) the compound cue (light and buzzer) was not presented. In the second condition the cue was presented 5 sec after shock onset and terminated with a successful escape response, while in the third condition the cue was presented 5 sec after shock onset and terminated 3 sec after a successful escape response was completed. Thus, cue onset occurred at the same time in the latter two groups, but in one case cue offset was contiguous with an escape response, but in the other group cue offset was delayed.

RESULTS

Escape performance was found to vary as a function of the Cue Manipulation, $F(2,27)=5.54$, $p<0.01$. Newman-Keuls multiple comparisons ($\alpha=0.05$) revealed that the mean escape latency among mice in the no-cue condition (mean=10.84 sec ± 1.07) was significantly longer than in the condition where the cue terminated with the escape response (mean=6.48 sec ± 0.82) or 3 sec after successful escape (mean=3.33 sec ± 0.37). The difference between the latter two groups was not statistically significant, although it should be noted that delaying cue offset actually reduced the escape latency to some extent. Thus it appears that the effectiveness of a cue offset in modifying escape performance was not derived just from the information value provided by the stimulus.

GENERAL DISCUSSION

It has been our contention that the interference of escape performance induced by inescapable shock is due, at least in part, to disturbances in the maintenance of shock-elicited activity. The high levels of activity that occur soon after shock onset favor proficient escape behavior irrespective of the organism's prior stress history. In contrast, if the task is one that does not permit immediate escape, the reduction of shock-elicited activity favors poor escape behavior, particularly if the animal's prior treatment provoked rapid and marked reductions of shock-elicited activity (e.g., inescapable shock or haloperidol treatment). This does not imply that naive animals are unaffected by the decay of shock-elicited activity. Almost invariably the performance of mice tested with a 6 sec escape delay procedure is inferior to that of mice tested with brief delays or no delay at all (see [2,5]). Indeed, it is not uncommon to find frequent escape failures among a small proportion of naive mice tested with a long escape delay procedure, but it is rare for this ever to occur among naive mice tested under conditions where escape is possible immediately after shock onset.

The results of the present series of experiments indicated that interruption of the shock train, a treatment previously shown to attenuate the decline of shock elicited activity [2], or presentation of a novel cue, enhanced performance of naive mice, as well as mice that had received prior exposure to inescapable shock or treatment with haloperidol. The effectiveness of these procedures was dependent on the time at which shock interruption or stimulus presentation occurred. Maximal performance enhancement was observed when the stimulus change occurred at the time that escape could be accomplished. When stimulus change occurred several seconds prior to escape being possible, a negligible change of response latencies was observed. Such a finding was not unexpected since the enhancement of shock elicited activity that accompanies stimulus change occurs for only a brief period of time, and thus the beneficial effects on escape behavior should be limited to this brief period. The fact that escape performance was enhanced among naive animals is not surprising, since the escape delay and the consequent decline of shock-elicited activity will ordinarily limit performance in naive animals, albeit to a lesser extent than among mice that had been exposed to inescapable shock or treated with haloperidol. An alternative to the preceding formulation is that presentation of the compound stimulus served to signal imminent gate opening, thereby facilitating escape performance. However, it should be noted that the enhancement of shock elicited activity produced by shock interruption will be evident even when shock is inescapable [2]. Thus, the motoric effects of this treatment are probably unrelated to the signal value of the cue.

In the present investigation it was found that the time of cue termination was, in fact, an essential feature in determining whether or not facilitated escape performance would be evident. More specifically, when cue presentation preceded gate opening by 1 sec and terminated with gate opening performance was unaffected, whereas enhanced escape behavior was evident when cue termination coincided with a successful escape response. Contrary to previous assertions [11,12] this effect did not appear to be due to feedback for appropriate responding, since the performance enhancement was evident even when cue termination occurred several seconds after an escape response was completed. It would appear that cue presentation enhanced performance through its capacity of provoking motor excitation or minimizing the reduction of shock-elicited activity that ordinarily occurs with long shock presentations. Moreover, given that escape is possible, the effectiveness of the cue in provoking successful performance is directly related to the duration of cue presentation.

The fact that both inescapable shock and treatment with haloperidol (as well as more specific DA receptor blockers, such as pimozide) provoke similar effects on shock-elicited activity [5], disrupt escape performance under similar condition [5], are modifiable by comparable pharmacological interventions [3] and are similarly influenced by changes in stimulation, suggests that the two manipulations influence performance through similar mechanisms. Indeed, the finding that uncontrollable stress would result in DA depletion in some brain regions, notably the arcuate nucleus [9] and frontal mesolimbic cortex [14] prompted the suggestion that in addition to NE, alterations of DA neuronal activity subserve the disruptive effects of inescapable shock on subsequent escape behavior [1,5].

It will be recalled that Wise and his associates [18,19] argued that DA neuronal activity subserves reward processes and incentive motivation, at least in so far as appetitive responding is concerned. If one were to extrapolate to aversively-motivated behaviors, the possibility might be entertained that the disruptive effects of haloperidol on escape behavior (the serial or parallel action of the drug on nondopaminergic systems notwithstanding) might be due to reduced motivation to respond or to diminished reinforcement derived from shock termination (negative reinforcement). Such an explanation for the haloperidol effects, however, does not readily provide an accounting for the effects of shock interruption or novel cue presentation on the behavioral deficits engendered by haloperidol treatment. Rather, the results of the present investigation are more comfortably interpreted in terms of the effects of haloperidol on response initiation/maintenance [5,15], or on a sensory-motor interface [13]. Indeed, it has previously been reported that change in stimulation will influence motor deficits associated with reduced DA receptor activity [15] and will influence alimentary behaviors and performance in tasks involving appetitive motivation [15], as well as avoidance of impending shock [6]. It would appear that haloperidol, like inescapable shock, results in deficits in response initiation to weak stimuli and hinders response maintenance in the face of strong stimuli. The superimposition of a novel stimulus may briefly attenuate the difficulties of response maintenance, thereby permitting expression of learned responses or the acquisition of new responses.

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