Retrograde amnesia produced by electroconvulsive shock after reactivation of a consolidated memory trace: A replication*

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Rats showed retrograde amnesia when electroconvulsive shock was administered 24 h after a fear conditioning trial when the shock was preceded by some of the specific conditioning cues but not others. This result replicates the findings of Misanin, Miller, & Lewis (1968) and suggests that ECS can act to produce retrograde amnesia by means other than disrupting memory consolidation.

The finding that retrograde amnesia (RA) results from an ECS administered shortly after a training trial is well documented (e.g., King, 1965; Young & Day, 1970), and this result has most often been interpreted as due to an action of ECS on memory storage processes (Glickman, 1961; McGaugh, 1966; McGaugh & Dawson, 1971). That memory storage disruption is a singular memory effect of ECS was recently brought into question by Misanin, Miller, & Lewis (1968), who reported that ECS administered 24 h after fear conditioning, when preceded by a brief "reminder" of that conditioning, resulted in an amnesia that was as complete as that produced when ECS immediately followed the fear conditioning trial. This finding suggests that ECS may modify memory processes other than memory storage. However, the impact of this report was tempered by the failure of Dawson & McGaugh (1969) to replicate this effect.

The present study attempted to replicate the general finding of Misanin et al that RA can be produced by pairing specific conditioning cues with ECS the day following fear conditioning.

SUBJECTS AND APPARATUS

The Ss were 95 male Long-Evans rats from the Central Washington State Psychology Department animal colony and ranged between 90 and 120 days of age at the beginning of the experiment. They were individually housed, maintained on Purina Lab Chow ad lib, and allowed only 15 min of water a day in the home cage for the duration of the experiment.

Drink training, fear conditioning,

trace reactivation, and testing took place in one of two identical chambers. The chambers consisted of a grid floor with the grids spaced 2 cm apart, center to center, and had Plexiglas walls 30 x 25 x 27 cm high. The Plexiglas ceiling had a small hole through which ECS leads could be attached to the Ss' ears via small flattened alligator clips. Each chamber was illuminated by a 25-W light and was enclosed in a fan-ventilated, sound-attenuated chamber. A drink tube protruded 2.54 cm into the chamber from the center of the front wall, with the drinking orifice located 5 cm above the grid floor. A drinkometer circuit automatically pulsed at a rate of 2/sec when the S was in contact with the drink tube. A 10-cm loudspeaker for delivery of the 1,200-Hz 15-sec tone conditioned stimulus (CS) from a Heathkit tone generator was positioned behind the rear wall of the chamber at floor level. During trace reactivation, the drink chamber was modified by taping cardboard, painted flat black, inside the Plexiglas walls. A piece of flat black painted cardboard was placed either over the grid floor or under it (see below), and illumination was provided by a 25-W red bulb. Additional apparatus consisted of an ECS source set to deliver a 200-msec 92-mA 60-Hz 1,840-V rms shock through modified alligator earclips attached to the ears of the S.

PROCEDURE

For the first 3 days of the experiment, the Ss were handled for 5 min/day, with tiny flattened alligator clips attached to their ears, and were watered after each handling session. On the 4th and 5th days, Ss were individually placed into one or the other of the two chambers and allowed 50 sec of contact with the drink tube before they were returned to their home cage, where they received their daily ration of water. Fear conditioning took place on Day 6. The Ss received their ration of water just prior to placement into the

chamber, where the drink tube was absent. Five minutes afterwards, the 15-sec tone CS was presented. A 1.0-mA 60-Hz 206-V rms polarized shock through the grid floor was presented during the last 2 sec of the CS. Reactivation of fear memory traces and ECS treatments were administered 24 h later (Day 7). An attempt was made to grade the extent of the memory reactivation by individually placing the animals in the modified drink chamber for 32.5 sec and giving different groups the four combinations of the factorially combined presence or absence of the grid floor with the presence or absence of a 2-sec presentation of the CS. Logically, the presence of both the grid floor and the CS should be more effective in reactivating fear memory traces than the absence of these cues. Presence of either cue could be expected to produce memory traces intermediate between the extremes produced by both cues or neither cue present. To better insure that the reactivation of memory traces was restricted to the manipulation of specific conditioning cues (grid and CS), the reactivation session was conducted by a different E. The daily water ration was administered just prior to trace reactivation. Again, the water tube was not present in the drink chamber. Ss in Group G-CS-ECS were earclipped and placed onto the grid floor of the modified drink chamber. Thirty seconds later, they were given a 2-sec presentation of the CS, followed .5 sec later by ECS. The presentation of both grid and CS prior to ECS essentially replicates the procedure of both Misanin et al (1968) and Dawson & McGaugh (1969). Ss in the G-NCS-ECS group were earclipped, placed onto the grid floor, did not receive the CS, but ECS was administered 32.5 sec after initiation into the chamber. Ss in the NG-CS-ECS group were earclipped, placed onto the black cardboard floor that covered the grid floor in this condition, presented the 2-sec CS, and ECS followed. Ss in the NG-NCS-ECS group were placed on the cardboard floor in the drink chamber and received an ECS. Eighteen Ss were included in each of the four ECS groups. Approximately equal numbers (Ns ranged from 5 to 7) of Ss were run in each of the four treatment combinations but did not receive ECS. Subsequent analysis of their subgroup test data indicated no reliable treatment effect or any obvious trends due to differential reactivation, so these data (N = 23) were pooled to form a control group with which to assess the effects of the ECS administered to the four experimental groups.

The Ss were returned to the

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Table 1
Test Performance (Log)

Group	N	50-Sec Drink Latency		5-Sec Drink Latency	
		Mean	SD	Mean	SD
Control-No ECS	23	2.62	.32	2.12	.45
NG-NCS-ECS	18	2.70	.28	2.05	.61
NG-CS-ECS	18	2.63	.42	1.91	.41
G-NCS-ECS	18	2.67	.26	1.69	.71
G-CS-ECS	18	2.76	.32	1.62	.53

unmodified drink chamber to test for retention of the fear conditioning 24 h (Day 8) after the trace reactivation procedure. At the test, the Ss were allowed to complete 50 sec of tube contact before the 15-sec CS was automatically presented. The CS was not followed by grid shock. Both the time (in seconds) for the Ss to initiate 50 sec of tube contact and to return and complete an additional 5 sec of contact following the CS presentation were recorded, transformed to logarithms, and analyzed separately with a simple one-way analysis of variance.

It should be noted that this procedure does not represent an exact replication of that used by Misanin et al (1968). Some of the notable differences between the two procedures were that, in the present study compared to Misanin et al, the conditioning footshock was polarized, less intense, and of shorter duration; the ECS was more intense and of shorter duration; and, at the test, the CS was presented for only 15 sec.

RESULTS

The mean log drink latencies and standard deviations for both the 50- and 5-sec measures are shown in Table 1. No group differences were noted in the analysis of the 50-sec drink latency data ($F < 1$, $df = 4/90$, $p > .05$).

Every S broke contact with the drinking tube upon the presentation of the CS. Analysis of the time for the Ss to resume drinking and complete an additional 5 sec of tube contact proved to be a reliable source of variance ($F = 3.01$, $df = 4/90$, $p < .025$). Comparisons of each ECS group to the control with t tests showed that both the G-CS-ECS and G-NCS-ECS groups returned to the drinking tube faster than the control group ($p < .01$ in both cases). However, neither the NG-CS-ECS nor the NG-NCS-ECS group differed from the control ($p > .05$ in both cases).

DISCUSSION

Despite several procedural differences, the findings of the present

experiment are in close agreement with those of Misanin et al (1968).

The failure of the initial drink latencies to differentiate between the treatment groups is consistent with the findings of both Misanin et al (1968) and Dawson & McGaugh (1969). Although both of these latter investigations showed that an ECS administered immediately after fear conditioning produced RA reflected by this measure, neither study found that an ECS given 24 h after conditioning modified initial drink latencies. Apparently, the initial drink latency is not sensitive to the effects of an ECS administered 24 h after conditioning.

In the present study, both the G-CS-ECS and G-NCS-ECS groups showed RA when their 5-sec drink latencies were compared to the control. This finding is in good agreement with Misanin et al (1968), who also reported the finding of RA in a group of Ss placed on the grid floor and presented the CS just prior to ECS and in a group simply placed on the grid floor and given ECS. The results of both the Misanin et al study and the present study are contrary to those of Dawson & McGaugh (1969). The factors responsible for this discrepancy are not clear at the present time.

Groups NG-CS-ECS and NG-NCS-ECS showed as much retention of the fear conditioning as the control. That RA was obtained from only the G-CS-ECS and G-NCS-ECS groups strongly suggests that the RA resulting from ECS given 24 h after conditioning was not due to the ECS per se but, rather, was due to the interaction between specific conditioning cues and ECS.

Neither Misanin et al (1968) nor Dawson & McGaugh (1969) included a group that received the CS prior to ECS while not positioned on the grid floor. However, the present data appear to indicate that the grid floor was a more salient conditioning cue than the specific CS (the tone) since the G-NCS-ECS group but not the NG-CS-ECS group showed RA.

Whether this finding actually reflects relative cue saliency is debatable. Following the procedure of Misanin et al (1968), the Ss in the present study were placed on the grid floor for 32 sec prior to ECS, whereas the CS was presented for only 2 sec prior to ECS. Since the duration of the two cues before ECS differed, it is difficult to conclude that the grid floor per se is the more important cue.

The results of the present study replicate the findings of Misanin et al (1968) and join a rapidly growing body of data demonstrating that ECS modifies memory processes other than storage (e.g., DeVietti & Larson, 1971; Lewis, Bregman, & Mahan, 1972; Nielson, 1968; Robbins & Meyer, 1970; Thompson & Grossman, 1972).

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