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CHAPTER I

INTRODUCTION

In an early study of the effects of electroconvulsive shock (ECS) on memory, Duncan (1949) demonstrated that rats showed learning deficits when subjected to ECS within 60 min. after each trial of a multiple-trial avoidance problem. It was assumed that ECS disrupted the memory trace laid down during training. Duncan's findings were consistent with clinical observations in which patients showed a retrograde amnesia (RA) for events just preceding electroconvulsive therapy (Zubin and Barrera, 1941) or severe head injury (Russell and Nathan, 1946).

Müller and Pilzecker (1900) found that a second list of nonsense syllables learned 17 sec. after a first list caused poorer retention of that first list than did interpolated rest. Retention deficits were attenuated when the second list was learned 6 min. rather than 17 sec. after the first. Müller and Pilzecker explained these results in terms of a perseveration-consolidation concept which posits that neural activity resulting from the learning of a task perseverates for a period of time and may be disrupted by interpolated material. Such findings have contributed to the current consolidation hypothesis elaborated by Hebb (1949) which holds that the memory trace is established in two stages: an initial, short stage corresponding to reverberating electrical activity in neural circuits and a second stage corresponding to permanent neuronal changes. Only the first stage may be disrupted by ECS or other amnestic agents.

The consolidation interpretation of Duncan's results has not gone unchallenged. Coons and Miller (1960) argued that his results could be explained by a "conflict hypothesis" emphasizing the punishing effects of ECS rather than its memory-disruptive effects. They were able to show enhanced learning when ECS was appropriately combined with foot shock. Their objections have been met, however, by procedures which provide for learning of a passive avoidance response in one trial follwed by a single ECS. In one such procedure, the step-down, S is placed on a safe platform from which it readily descends onto a shock grid (Jarvik and Essman, 1960). In the step-through procedure (Essman and Alpern, 1964), S is placed in the small, light compartement of a two-compartment apparatus. S readily steps through a hole into a large, dark, grid-floored chamber. A single foot shock is sufficient to produce passive avoidance as evidenced by significantly increased step-down or step-through latencies when S is tested 24 hrs. after training. If a single ECS is administered shortly after training, Ss enter upon the grid floor on the second (test) day with latencies comparable to those seen on the training day. Thus, in order to demonstrate amnesia, S must work against the punishment gradient. S must perform the very response that just preceded ECS during training.

While these one-trial results cannot be explained in terms of Coons and Miller's punishment hypothesis, Hudspeth et al. (1964) have indicated that multiple administrations of ECS produce a measureable punishing effect. The following treatment will therefore be confined to studies employing single ECS procedures.

The Conditioned Inhibition Hypothesis

While the passive avoidance procedures outlined above seem to obviate a punishment interpretation, attempts have been made to explain passive avoidance results in terms of mechanisms other than disruption of the memory trace. Lewis and Maher (1965, 1966) have carefully differentiated between the performance deficit seen after ECS administration and the underlying causes of that deficit. They conclude that: "These studies do demonstrate a performance impairment, but are not conclusive to the more precise issue of whether performance impairment (retrograde amnesia) is a consequence of disruption of the engram, or whether it is due to interference by some competing response." (Lewis and Maher, 1966). The nature of this competing response is most precisely summed up by McGaugh and Petrinovitch (1966):

> ECS is assumed to be an unconditioned stimulus which produces an unconditioned response consisting of loss of consciousness. A weak version of loss of consciousness, that is, relaxation, becomes conditioned to the cues present at the time of ECS administration. Apparent amnesia results, according to this view, because the relaxation response (or conditioned inhibition) competes with other responses

Lewis and Maher (1966) explain the behavioral consequences of conditioned inhibition:

> Inhibitory avoidance learning--of which freezing in the conditioned emotional response is the exemplar--involves the maintenance of a considerable amount of muscular tension in a highly aroused animal. Under conditions where fear is gradually reduced--as by normal extinction processes--the reduction in arousal is evidenced by an <u>increase</u> in the general locomotor activity. Thus, the animal that is in a lowered state of arousal should be less likely to "freeze"--more likely to move around--than one that is in a highly fearful state.

This is to say that the "lowered state of arousal" presumabley produced by ECS will result in the same passive avoidance behavior as disruption of the memory trace--shorter step-through/step-down latencies than those seen in non-convulsed controls. One is thus unable to separate the disruptive effects of ECS from its conditioned inhibition effects on the basis of passive avoidance behavior.

The following paragraphs constitute a review of relevant ECS-behavior variables in an attempt to determine the efficacy of the consolidation and conditioned inhibition hypotheses.

ECS and Activity Level

The conditioned inhibition hypothesis leads to the prediction that Ss will show more activity (less inhibition of movement) after ECS administration. Several studies have found results consonant with this prediction. Routtenberg and Kay (1965) report that pretreatment with ECS alone (without foot shock) decreases step-down latencies in rats. Decreased step-down latencies may indicate a high level of activity. Schneider et al. were unable to replicate this finding but did find an interaction between foot shock and ECS. Rats pretreated with noncontingent foot shock followed 0.5 sec. later by ECS showed decreased step-down latencies. Nielson (1966) reported increases in open-field ambulation when rats were tested 24 hrs. after foot shock-ECS treatment. Kopp et al. (1967) obtained decreased step-through latencies when ECS was administered 1 hr. before a training trial or 1-8 hrs. before a subsequent retest. No effect was observed if ECS was given 12 hrs. before retest and "...retest latencies were decreased significantly less by a single ECS administered 1 hr.

before retest (proactive disinhibitive effect) than by a single ECS administered 1 hr. after punishing shock (retroactive disruptive effect)." Zornetzer and Mcgaugh (1969) found significant increases in open-field activity at 48 and 72 hrs. after treatment with foot shock and ECS in the step-through procedure, but not after 24 hrs. Since the passive avoidance procedures usually test for RA 24 hrs. after ECS administration, the time course during which the ECS-foot shock combination increases activity becomes a critical consideration. Kopp et al. and Zornetzer and McGaugh seem to agree that no activity increases can be seen at 24 hrs. but the differences in their findings and the increses in open-field ambulation after 24 hrs. reported by Nielson cast doubt on the efficiency of the passive avoidance procedure for discriminating between RA and increased activity.

The Training-ECS Interval

Duncan (1949) reported that, as the time interval between each training trial and the administration of ECS increased, the amount of learning impairment decreased. These results are quite consistent with both the conditioned inhibition and consolidation hypotheses. There is, however, considerable disagreement as to the length of the effective training-ECS interval. Some studies report RA only when ECS is administered within a few seconds after training. Others have obtained RA effects when ECS was administered 1 or more hrs. after training.

Heriot and Coleman (1962) produced one-trial passive avoidance of an established bar pressing response by shocking rats at the bar. ECS eliminated the subsequent suppression of bar pressing when administered as much as 1 hr. after punishing shock. The amount of amnesia varied inversely with the length of the training-ECS interval. Kopp et al. (1966) obtained similar training-ECS interval gradients using the step-through procedure. Bures and Buresova (1963) found that rats which were allowed to explore a two-compartment box spent most of their time in the smaller compartment, but most Ss refused to re-enter the smaller compartment after receiving 1 min. of inescapable shock therin. ECS delivered more than 2 hrs. after foot shock attenuated avoidance of the smaller compartment. the amount of attenuation varied inversely with the interval between training and ECS.

In contradistinction to the relatively long training-ECS intervals reported above, Chorover and Schiller (1965) and Sprott (1966) were unable to show any impairment with intervals longer than 10 and 20 sec. respectively. In order to reconcile these disparate results, Chorover and Schiller (1966) performed a series of four experiments in which they 1) essentially replicated the results of Bures and Buresova 2) showed that rats receiving ECS 1 min. after training shock and confinement in the smaller compartment of the Bures-Buresova apparatus exhibited more locomoter activity than non-convulsed controls when allowed to explore the larger compartment on the following day 3) obtained no impairment with ECS administered 1 min. after shock training when Ss were allowed to escape the smaller compartment rather than being confined for the duration of shock and 4) obtained impairment at training-ECS intervals longer than 10 sec. in the step-down procedure when Ss were confined to the apparatus prior to ECS. They were thus able to increase of decrease the length of the effective training-ECS interval in both the step-down and two-compartment apparatus by confining or not confining Ss after foot shock. They concluded that confinement in the shock apparatus produced a generalized conditioned emotional response (CER) rather than a discriminated avoidance. The CER blocks exploratory behavior, i. e., causes freezing behavior which reduces the probability of entering the smaller compartment or stepping down onto the shock grid. "The effect of ECS is to suppress this inhibitory aspect of the CER and to thereby produce a relative increase in locomotor activity..." While they agree that ECS can interfere with the consolidation of the memory trace, they hypothesize that "...RA may be responsible for impaired retention test performance only when the convulsive treatment is given a few seconds after the learning trial." Robustelli and Jarvik (1968) also showed that detention in the apparatus (step-through) after foot shock lengthened the effective training-ECS gradient. However, detention alone, without ECS, caused decreased step-through latencies.

These studies all confirm that the degree of impairment caused by ECS does increase as the training-ECS interval decreases. But the length of time after training during which ECS is capable of causing amnesia is still in question. Chorover and Schiller have raised the possibility that impairment observed with short training-ECS intervals may be due to RA, but that impairment following long intervals may be due to a form of conditioned inhibition.

ECS Intensity, Duration, and Mode of Delivery

Some of the disparate results reported above might be reconciled if it could be shown that the effective training-ECS interval varies with intensity, duration, and mode of administration of ECS. In their 1965 experiment, Chorover and Schiller were unable to show impairment with training-ECS intervals longer than 10 sec. They used a transpinnate ECS of 30-50 ma intensity passed for 0.2 sec. A. J. Miller (1968) obtained significant impairment in the step-down up to 30 sec. after training with a 35 ma, transpinnate ECS. When intensity was increased to 100 ma, he obtained impairment up to 50 min. Ray and Barrett (1969) showed that the effective training-ECS interval increased when ECS intensity was varied between 5 and 80 ma. In fact, impairment was seen when ECS intensity was too low to cause convulsions. This latter finding was confirmed by McGaugh and Alpern (1966) and Essman (1968) who also produced impairment after blocking convulsions with ether and xylocaine respectively. Paolino et al., however, were unable to demonstrate increases in impairment when ECS intensity was increased from 100 to 200 ma. They used a transpinnate ECS administered 60 sec. after step-through training.

Alpern and McGaugh (1968) also found increases in impairment as a function of ECS duration. The effective training-ECS interval was only a few seconds for brief ECS durations (15 ma passed for 0.2 sec.) but increased to 3 hrs. with long ECS durations (8 ma passed for 0.8 sec.). However, Paolino et al. (1969) and A. J. Miller (1968) found no differences in impairment with increased ECS duration. They used 200-800 msec. and 200-500 msec. respectively. It should be noted that Alpern and McGaugh administered ECS transcorneally while the latter investigators used the transpinnate technique. Transcorneal ECS has been shown to cause greater impairment than the transpinnate technique (Ray and Barrett, 1969; Dorfman and Jarvik, 1968).

Although the literature appears somewhat equivocal, it is evident that statements concerning the effective training-ECS interval must take into account the variables of intensity, duration, and mode of administration.

The Reactivated Trace

The consolidation hypothesis stipulates that the memory trace is consolidated in at least two stages. The first stage is operationally defined by its susceptability to disruption by ECS and other amnestic agents. The second phase is defined by its resistance to such disruption. The consolidation hypothesis is therefore sharply questioned by studies which show apparent disruption of the memory trace long after the first stage should have been terminated as well as a return of a presumably disrupted trace.

Schneider and Sherman (1968) trained rats in a step-down procedure and delivered a second, non-contingent foot shock either 29.5 sec. or 6 hrs. after initial training. ECS administered 0.5 sec. after the second, non-contingent foot shock produced impairment in both groups although the second group had been trained more than 6 hrs. prior to ECS administration. Furthermore, subsequent retesting showed a return of avoidance behavior for the 29.5 sec. condition although no return was seen in the 6 hr. condition. The authors attribute the return of avoidance to an increase in conditioning strength caused by the non-contingent shock when administered at 29.5 sec. after training shock. They conclude that ECS does not disrupt the memory trace but that apparent amnesia results from some interaction between foot shock and ECS (perhaps the reduction of freezing behavior suggested by Lewis and Maher). Misanin et al. (1968) showed similar results when they suppressed lick rate in rats with a single foot shock paired with an 80-db white noise CS. When the CS was presented 24 hrs. later and followed immediately by ECS, Ss failed to show suppression of lick rate (amnesia). Thus an apparent amnesia was produced when ECS was administered many hours after the memory trace was presumed consolidated. Controls given ECS without the white noise CS showed lick rate suppression (retention). Apparently, CS presentation made the consolidated trace disruptable once again.

Attempts to replicate Schneider and Sherman (Banker et al., 1969) and Misanin et al. (Dawson and McGaugh, in press) have obtained negative results. Interestingly, Davis and Klinger (1969) also obtained negative results with goldfish. They were unable to obtain impairment when ECS was given after re-introduction into a shuttlebox environment in which the fish had previously been trained with shock. Yet, they were able to show impairment when re-introduction was followed by other amnestic agents (intracerebral injections of puromycin, acetoxycycloheximide, or KCl).

The ability to disrupt the memory trace long after the first, disruptible stage of consolidation should be over creates grave difficulties, particularly for those who hold that the second stage of consolidation represents real anatomical changes within the organism. At best, one must posit that the first stage has in some way been reactivated (the physical changes reversed?) by the experimental procedures thus exposing the previously impervious engram to disruption. The phenomenon might be readily explained by the conditioned inhibition hypothesis, but its very existence must be questioned in view of lack of replication.

The Permanence of Amnesia

The production of an incomplete amnesia (partial behavioral deficit) following ECS is not inconsistent with the consolidation hypothesis. However, the consolidation hypothesis would seem to require that any deficit which is produced by ECS must remain permanent in the absence of further training. Several studies, including Schneider and Sherman cited above, have indicated that deficits need not be permanent. The implication is that ECS does not destroy the memory trace (amnesia) but rather blocks its expression (retrieval).

Zinkin and Miller (1967) administered a 35 ma, 200 msec., transpinnate ECS to rats after step-down training. Impairment of retention was present when Ss were tested 24 hrs. later; but when Ss were tested again at 48 and 72 hrs., impairment disappeared. Lewis et al. (1968) reported that a "reminder shock" delivered outside the step-down apparatus after training therein greatly reduced impairment caused by ECS. They concluded that: "This indicates that at least part of the memory remained, but that its retrieval was prevented by ECS."

Other workers have been unable to demonstrate this return of the disrupted memory trace. Herz and Peeke (1967) found no diminution of impairment caused by transpinnate ECS delivered within 15 sec. after training. Ss were tested once at 24, 48, or 72 hrs. and repeatedly at 24, 48, and 72 hrs. They used a head-poke apparatus consisting of "...a chamber with a cul-de-sac into which a mouse could poke its head and receive either water or foot-shock from a cup." Luttges and McGaugh (1967) were unable to replicate Zinkin and Miller's results. Ss were given one retention trial each at 24, 48, or 72 hrs. after training. No diminution in impairment was observed. They speculate that Zinkin and Miller's results were contaminated by repeated exposure to the test apparatus.

These differences might be reconciled by Pagano et al. who report that the permanence of amnesia is a function of ECS intensity. Rats receiving a 95 ma, transpinnate ECS 0.5 sec. after foot shock training showed deficits when tested after 48 hrs.; but a 55 ma ECS produced deficits lasting less than 24 hrs. Unfortunately, ECS intensities in the above studies cannot be compared due to differences in species, training procedures, and mode of administration.

Other Factors

Besides the variables already mentioned, other factors have been shown to influence post-ECS behavior and may affect the interpretation of the studies above. The strain of animal, as well as the species may affect results. Bovet et al. and Wimer et al. report differences in active and passive learning performance among different strains of inbred mice. Sprott (1966), however, found no differences in the effective training-ECS interval when two inbred strains of mice were compared. The time of day during which Ss are trained or tested has been shown to affect the training-ECS interval (Stevens and McGaugh, 1968a, 1968b). Furthermore, some studies have obtained passive avoidance by interrupting an ongoing response such as bar pressing. Their results may not be comparable to those obtained by procedures such as the step-through which require only one exposure to the apparatus during training.

The Present Study

Both the consolidation and conditioned inhibition hypotheses have been invoked to explain the performance deficit seen after ECS in the passive avoidance procedures described above. Since both hypotheses predict the same behavior in the passive avoidance situation, it is impossible to differentiate between them on the basis of the passive avoidance paradigm. In the present study, a testing situation is employed wherein opposite behavioral outcomes may be predicted by the consolidation and conditioned inhibition hypotheses.

Maatsch (1959) reported that rats could be trained to jump from a shock box in order to avoid shock after only one training trial. While the present investigator has been unable to replicate these findings, he has shown (see below) that a single, inescapable foot shock delivered in the shock compartment of a step-through apparatus is sufficient to facilitate the acquisition of a multiple trial active avoidance response. This response consists of jump-out from that same shock compartment before shock onset. Thus a single foot shock can be used in the training of both a passive and active avoidance response in the same apparatus.

If ECS disrupts the memory trace left by training shock (consolidation hypothesis), convulsed S should not only show passive avoidance deficits when tested 24 hrs. after foot shock-ECS but should also show poorer acquisition of active avoidance when compared to non-convulsed controls. The conditioned inhibition hypothesis, however, holds that ECS does not disrupt the memory trace. The information consolidated after foot shock on the previous day should still be available to facilitate the acquisition of active avoidance 24 hrs. later. The conditioned inhibition hypothesis further posits that convulsed Ss will be more active in the apparatus during testing than non-convulsed controls. This greater activity may in fact potentiate the jump-out response so that convulsed Ss may learn the active avoidance response more readily than controls.

Thus, two opposite predictions are generated in regard to active avoidance performance. The consolidation hypothesis predicts a deficit in active avoidance performance after ECS. The conditioned inhibition hypothesis predicts no deficit, perhaps a potentiation of performance, after ECS.

CHAPTER II

EXPERIMENT I

Methods

Subjects. One hundred and twenty male, albino mice of the CF-1 strain, weighing 18-21 g., were obtained from Carworth Farms Laboratories (New City, N. Y.) several wks. before the experiment. *S*s were housed 10 per cage in standard, 12 x 12 x 30 cm clear plastic cages and given Purina Lab Chow and fresh water *ad libitum*.

Apparatus and procedures. The two-compartment step-through apparatus used in this study was similar to that described by Essman and Alpern (1964). On the first (training) day of the experiment, *S*s were housed 5 per cage and their tails marked for identification. Training was begun at approximately 10:00 AM by placing each *S* individually into the vestibule of the step-through, a 5 x 4 x 6.5 cm compartment constructed of clear plexiglass with a hardware cloth floor. Each *S* was placed with its head facing a 4 cm diameter hole which communicated with the shock box; a 20.5 x 22 x 10.5 cm, unlighted, black, plexiglass box with a hinged lid and grid floor. The floor was composed of 20 stainless steel rods approximately 2 mm in diameter which were wired in series through a cam operated grid scrambler to a 95 V power supply providing a current of approximately 3 ma when the circuit was completed by foot contact across the grids.

The time interval between placement in the vestibule and entry with all four feet into the shock compartment (step-through latency) was timed with a stopwatch; a guilletine door was closed behind *S*;

and an inescapable, 3ma, scrambled training shock was delivered to the feet. Shock duration was timed with an industrial timer.

Prior to the training procedure described above, Ss were divided into 4 groups of 30 each:

Group 1 (Training Only) was trained as above. Step-through latencies were taken, training shock was delivered, and Ss were returned to their home cages without receiving ECS.

Group 2 (Training + Immediate ECS) was trained as above and then given a 20 ma, 700 V, 200 msec., transcorneal ECS as soon after termination of foot shock as possible (usually 6 sec. but never more than 10 sec. after training shock) and returned to their home cages.

Group 3 (No Training, No ECS) was given no step-through training. Ss were marked for identification and returned to home cages.

Group 4 (ECS Only) was given no step-through training but received ECS outside the step-through apparatus.

On the second (testing) day, all groups were given identical treatment. Testing was begun 24 hrs. after training by placing each S individually into the vestibule as on day 1 and timing step-through latencies. Any S failing to step through within 180 sec. was forced into the shock compartment. After step-through, the guilletine door was lowered and the hinged lid of the shock compartment was raised. S then had 10 sec. to jump from the grid floor of the shock compartment onto the table upon which the apparatus rested. Failure to jump within 10 sec. was followed by a 3 ma, scrambled foot shock which was delivered until the jump-out response was performed. Most Ss jumped out before 1 min. of continuous foot shock was delivered on the first trial. After jump-out, S was allowed to explore the table top for 20 sec. (rest period) and was then placed directly onto the grid floor of the shock apparatus through the open lid. Again, foot shock could be avoided by performing the jump-out response within 10 sec. after placement on the grid floor. This procedure was repeated for 10 massed trials. Jump-out occuring less than 10 sec. after placement on the shock grid (before shock onset) was scored as an active avoidance.

Results

Passive avoidance. Table 1 indicates the number of Ss in each group of 30 which showed passive avoidance on the second (testing) day. Passive avoidance is defined as failure to step through into the shock compartment within 180 sec. after placement in the vestibule. The chi-square test indicated that Group 1 (Training Only) showed significantly more passive avoidance than the other groups ($P < .001$) while these latter groups did not differ.

Active avoidance. Table 2 indicates the results obtained from a Kruskal-Wallis one-way analysis of variance based on the number of active avoidances in each group of 30. Active avoidance is defined as a jump-out latency of less than 10 sec. recorded for any of the 10 jump-out trials administered to each S. Each S thus had a possible maximum of 10 active avoidances. Each group of 30 had a possible maximum of 300 active avoidances. Group 1 (Training Only) showed significantly more active avoidance than the other groups ($P < .01$). Group 2 (Training + Immediate ECS) did not differ from Group 3 (No Training, No ECS) but showed more active avoidance than Group 4 (ECS Only.

Discussion

Passive avoidance. The superior passive avoidance preformance of Group 1 (Training Only) compared to the other groups is consistent with results obtained in previous experiments and may be explained by both the consolidation and conditioned inhibition hypotheses. The consolidation hypothesis posits that ECS administered to Group 2 (Training + Immediate ECS) eradicated the memory trace left by foot shock training. The conditioned inhibition hypothesis that ECS is a US for unconsciousness. The apparatus cues then become a CS for relaxation (a weak form of unconsciousness) which disinhibits the freezing response in the vestibule.

Active avoidance. It is apparent from a comparison of Group 1 (Training Only) with Group 3 (No Training, No ECS) that a single foot shock delivered on the first (training) day significantly facilitates the aquisition of active avoidance on the second (testing) day. Since Group 2 (Training + Immediate ECS) received foot shock training, any active avoidance deficit in Group 2, when compared to Group 1 (Training Only) should be due to the effects of ECS. The consolidation hypothesis would posit that the inferior active avoidance performance of Group 2 is due to ECS-induced disruption of the memory trace left by foot shock on the first (training) day.

The conditioned inhibition hypothesis, on the other hand, holds that the memory trace left by training foot shock is not eradicated by ECS. Since a single foot shock does facilitate the acquisition of active avoidance 24 hrs. after training, proponents of the conditioned inhibition hypothesis must explain why the trace left by training shock did not facilitate the performance of Group 2 (Training + Immediate ECS).

These results can be interpreted in a manner consistent with the conditioned inhibition hypothesis. It has been assumed that ECS causes relaxation in convulsed Ss. Apparatus cues become a CS for relaxation. It is therefore possible that convulsed Ss are so relaxed in the shock box that they are not amenable to further training in that box. In other words, ECS (or the combination of foot shock and ECS) has produced a depressive effect which inhibits the jump-out response or, in effect, raises shock threshold in convulsed Ss. This relaxation may overcome increases in activity when an effortful active avoidance response is required. Experiment II will explore these possibilities.

CHAPTER III

EXPERIMENT II

Experiment II explores the nature of the effective training-ECS interval, particularly in regard to the active avoidance response. Evidence cited above indicates that passive avoidance performance deficits in convulsed Ss are attenuated as the training-ECS interval is increased. Experiment I showed that ECS administered immediately after training caused deficits in active avoidance acquisition. Active avoidance deficits should also be attenuated in a systematic fashion paralleling passive avoidance as the training-ECS interval is increased.

Chorover and Schiller (1966) concluded that performance deficits induced by short training-ECS intervals (10 sec. or less) were probably due to RA while these seen after long training-ECS intervals were due to some other mechanism. Lengthening the training-ECS interval and comparing the results of active and passive avoidance may throw some light upon this contention.

The consolidation-conditioned inhibition controversy will also be subjected to further study. The predictions of the consolidation hypothesis need no elaboration here. The predictions of the conditioned inhibition hypothesis, however, are more complex. It might be assumed that ECS induces conditioned relaxation--conditioned increases in activity level. As the training-ECS interval is increased, the CS-US interval is also increased. The amount of activity should then decrease as the training-ECS interval is increased. Ss trained with long training-ECS intervals might actually do more poorly in active avoidance than those trained with short intervals. The results of Experiment I indicate that this is an unlikely outcome since it would require that Group 2 (Training + Immediate ECS) would show superior active avoidance performance.

Alternatively, it might be assumed that ECS causes so much relaxation in the shock box that convulsed Ss are not amenable to further learning there. In that case, as the training-ECS (CS-US) interval is increased, there should be less conditioned relaxation and better learning. Performance should improve in a systematic fashion paralleling that of passive avoidance as the interval is increased. Again, there would be no way to discriminate between the consolidation and conditioned inhibition hypotheses.

Experiment II will show that, contrary to both hypotheses, increases in the training-ECS interval does not result in systematic improvement in active avoidance performance paralleling that seen in passive avoidance.

Methods

Subjects. Two hundred and ten of the mice described in Experiment I were obtained several weeks before the experiment and housed in the same manner.

Apparatus and procedure. The same apparatus was used as that described in Experiment I except that a hole was cut in the table top upon which the step-through rested and the entire apparatus was lowered so that the top of the shock compartment became level with the table top. The lid of the shock compartment was unhinged so that the entire lid could be removed from the apparatus. These modifications were introduced in order to reduce variability in jump-out performance.

The general procedure was the same as that described in Experiment I. Ss were introduced into the vestibule on the first (training) day and step-through latencies were recorded. After 24 hrs., step-through latencies were again recorded (testing) and Ss were given 10 jump-out trials as described above.

Prior to this general training procedure, Ss were divided into 7 groups of 30 each:

Group 1 (Training Only) was a replication of Group 1 in Experiment I. Ss were removed to their home cages immediately after training shock without receiving ECS.

Group 2 (Training + Immediate ECS) was a replication of Group 2 in Experiment I. Ss were given ECS immediately after foot shock training.

Group 3 (No Training, No ECS) was a replication of Group 3 in Experiment I. Ss were given no ECS or step-through experience.

Group 4 (ECS Only) was a replication of Group 4 in Experiment I. Ss received ECS outside the apparatus but no step-through experience.

Group 5 (Proactive) was designed to test whether the foot shock-ECS combination could affect learning or performance without causing RA and without causing conditioning. ECS was therefore delivered outside the apparatus 15 min. before foot shock training in the step-through.

Kruskal-Wallis one-way analysis of variance based on the number of active avoidances (jump-out latencies of less than 10 sec.) in each group. Group 1 (Training Only), Group 5 (Proactive), Group 6 (20 sec. ECS), and Group 7 (1 min. ECS) did not differ significantly although the performance of Groups 5, 6, and 7 was numerically superior to that of Group 1. These 4 groups did show significantly more active avoidance than Group 2 (Training + Immediate ECS), Group 3 (No Training, No ECS), and Group 4 (ECS Only) with p < .02.

Discussion

Passive avoidance. Both groups which received no step-through training (Group 3, No Training, No ECS and Group 4, ECS Only) showed significantly poor passive avoidance performance. All groups which received foot shock training followed by ECS showed significantly impaired passive avoidance performance. The groups which were trained with a long training-ECS interval showed intermediate amounts of passive avoidance performance impairment (Group 6, 20 sec. ECS and Group 7, 1 min. ECS). They showed significantly less passive avoidance than Group 1 which received training but no ECS and significantly more passive avoidance than Group 2 which received ECS immediately after shock training. These results are predicted by both the conditioned inhibition and consolidation hypotheses.

Group 5 (Proactive) also showed an intermediate amount of impairment in passive avoidance (Groups 5, 6, and 7 did not differ statistically) despite the fact that ECS was administered 15 min. before foot shock. Since ECS preceded foot shock, this performance cannot be explained in terms of RA nor in terms of conditioned inhibition. The conditioning interpretation would require a backward conditioning paradigm with a US-CS interval of 15 min. This indicates that ECS, or the combination of ECS-foot shock, is capable of impairing passive avoidance by mechanisms other than those proposed by either the consolidation or conditioned inhibition hypotheses.

Active avoidance. The results of Experiment I were successfully replicated. A single foot shock was shown to facilitate active avoidance acquisition 24 hrs. later as evidenced by the significant difference between Group 1 which received foot shock but no ECS and Group 3 which received no foot shock nor ECS. Again, a single ECS administered immediately after foot shock (Group 2) was shown to significantly impair the acquisition of active avoidance.

The results of Group 5 (Proactive), Group 6 (20 sec. ECS), and Group 7 (1 min. ECS) require careful consideration. Each of these groups received both foot shock and ECS. ECS impaired the passive avoidance performance of each of these groups. However, ECS had no significant effect on active avoidance performance in any of these groups. They did not differ statistically from Group 1 (Training Only). This suggests that the intermediate amount of passive avoidance impairment seen in these groups was not due to RA but to some other mechanism. Furthermore, the greater (though nonsignificant) number of active avoidances found in these groups as compared to Group 1 (Training Only) might be explained if it were assumed that the Ss in Groups 5, 6, and 7 were more active than normal--more ready to jump in response to foot shock. It would then appear that ECS has a dual action. At short training-ECS intervals as in Group 2 (Training + Immediate ECS), ECS disrupts the memory trace. At longer intervals, ECS, or the ECS-foot shock combination, simply increases activity levels. The impaired passive avoidance performance in Groups 5, 6, and 7 would then be due to increased activity. This interpretation is quite similar to that put forward by Chorover and Schiller cited above.

CHAPTER IV

CONCLUSIONS

A single foot shock administered in the shock compartment of the step-through apparatus facilitates the acquisition of a multiple-trial active avoidance response trained 24 hrs. later in the same apparatus. A single ECS administered immediately after the single foot shock significantly impairs the acquisition of active avoidance 24 hrs. later. A single ECS administered more than 10 sec. after training foot shock or 15 min. before training foot shock significantly impairs passive avoidance in the step-through but does not impair active avoidance acquisition. A conditioning interpretation of these results seems most unlikely for several reasons. ECS was always administered outside the apparatus for all groups. This would seem to reduce the possibility that cues from the apparatus could become a CS for conditioned relaxation as required by the conditioned inhibition hypothesis. Furthermore, a backward conditioning paradigm must be invoked to explain how ECS could impair passive avoidance performance when it is delivered 15 min. before foot shock as in group 5 (Pro-active).

A more reasonable interpretation entails the supposition that ECS causes RA at short training-ECS intervals, but simply contributes to increases in activity level at longer intervals and in the pro-active condition. Such an interpretation would be consistent with the results obtained by Chorover and Schiller (1965, 1966), which indicated