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The effect of electroconvulsive shock on learned helplessness in the rat.

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THE EFFECT OF ELECTROCONVULSIVE SHOCK ON LEARNED HELPLESSNESS IN THE RAT

by

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Abstract

The effect of Electroconvulsive Shock (ECS) therapy on the retarded shock-escape acquisition following pretreatment with inescapable underwater exposure was examined. Three groups of 20 rats each served as subjects. The experimental animals (IW) were placed in a restraining device and given 40, 10 second, submersion trials of inescapable underwater exposure. A second group of animals (R) was simply restrained for the same time period as the IW group, but were not submerged. A third group of animals (N) was given no pretreatment. Twenty-four hours later (day 1) all animals were tested for shock escape in a two-way shuttle box for 25 trials. Immediately following test, half of the animals in each group received a single ECS treatment (ECS group) of 40 mA. for one second. The other half received no ECS treatment (NECS group). Twenty-four hours after treatment (day 2), the six groups (IW-ECS, IW-NECS, R-ECS, R-NECS, N-ECS, and N-NECS) were again tested for shock escape in the two-way shuttle box.

On day 1, the IW group was significantly slower

to escape shock when compared to the R and N groups. This interference effect is consistent with the learned helplessness hypothesis. The results of the test on day 2 (24 hours after treatment) indicated no effect of treatment, that is, the ECS groups (IW-ECS, R-ECS, and N-ECS) did not differ significantly in escape latency when compared to their corresponding NECS groups, although in each case, the ECS groups had lower response latencies than the NECS groups. This failure to find an ECS effect on learned helplessness in the rat was attributed to procedural measures which may have been insensitive to an ECS effect.

Overmier and Seligman demonstrated that dogs exposed to inescapable and unavoidable electric shock subsequently fail to learn a shock-escape task in a different situation (Overmier and Seligman, 1967; Seligman and Maier, 1967). Later studies (Overmier, 1968; Seligman, Maier, and Geer, 1968; Maier, 1970; Seligman and Groves, 1970) have provided additional evidence for this effect under a variety of other circumstances.

A "learned helplessness" hypothesis has been formulated to account for these interference effects (Overmier and Seligman, 1967; Maier, Seligman, and Solomon, 1969; Seligman, Maier, and Solomon, 1971). According to this hypothesis, animals which have received inescapable shock pretreatment learn of the independence between their responses and shock termination. This learning is retained when these animals are tested in a different situation where escape is possible. When given this escape task, pretreated animals fail to learn to escape, ultimately become inactive, and passively accept the shock, even though they may have occasionally successfully escaped the shock on previous trials.

This learned helplessness effect has been demonstrated in other species including the laboratory rat, however, Maier, Albin, and Testa (1973) and Seligman and Beagley (1975) have shown that it is more difficult to obtain interference effects in the rat using procedures similar to those used for the dog. Maier et.al. (1973) exposed rats to inescapable shock and found no failures to learn a shuttle-box shock escape task. The experimenters then varied the number, intensity, and interval between inescapable shocks and found the pretreated animals did not differ from controls in latency to escape shock in the shuttle box. Upon increasing the difficulty of the shock escape task from an FR-1 schedule (animal enters opposite compartment to escape shock) to an FR-2 schedule (animal escapes shock by entering opposite compartment then re-entering the original compartment). Maier et.al. (1973) found animals pretreated with inescapable shock to be retarded in acquiring the escape response, while the rats receiving no pretreatment, and the rats that were simply restrained, learned the task. The results were interpreted as supporting the

learned helplessness hypothesis. They also concluded that the interference effect could be demonstrated in rats providing the escape task was one which could be acquired more gradually. Since Maier et.al. (1973) have shown the lack of a learning curve for all tests using the FR-1 shock escape task, they concluded that this response was not learned by the animals. The response **latency** was very short and did not decrease over trials. Maier et.al. (1973) argue that the FR-1 response may simply be a reflexive response to shock. This suggests that the FR-1 response, in contrast to the FR-2 response, is one which is not acquired gradually. The FR-2 response, on the other hand, produced a learning curve for the control animals over trials, that is, the control animals gradually responded more quickly. This is an indication of learning. The inescapably shocked animals, however, did not show any learning curve which would suggest that they did not learn the task. Maier et.al. (1973) also demonstrated this interference effect using a wheel turning response to escape shock which was also shown to be acquired gradually. It was later demonstrated that an FR-3

bar press shock termination task also produced reliable interference effects in rats pretreated with inescapable shock (Seligman and Beagley, 1975). FR-1 and FR-2 schedules produced no such interference effect. Seligman and Beagley (1975) suggest that this interference effect produced with an FR-3 bar press shock escape task is characteristic of learned helplessness produced in dogs. In contrast to the failures to escape shock observed in 67% of dogs pretreated with inescapable shock, rats show a much lower percentage of failures to escape shock. Rats pretreated with inescapable shock, escape, but with a higher latency than control rats. This, however, has also been considered a demonstration of learned helplessness (Maier et.al., 1973; Maier and Testa, 1975; Seligman and Beagley, 1975). This learned helplessness effect observed in rats has recently been extended to include other forms of aversive pretreatment.

Altenor, Kay, and Richter (1977) pretreated rats with inescapable underwater exposure and found retarded acquisition of a shuttle-box escape task, in these animals, compared to animals receiving escap-

able pretreatment. Also, rats given inescapable shock pretreatment performed worse in an underwater maze than animals which received escapable shock pretreatment. The authors suggest that the learned helplessness phenomenon is not specific to any pretreatment stimulus, but is a general effect in rats. In addition to demonstrating the learned helplessness effect across 2 different pretreatment conditions, Altenor et.al. (1977) produced the interference effect using underwater exposure as a pretreatment condition. Since the interference effect has been produced with other forms of pretreatment and test, the learned helplessness hypothesis may be extended to include the uncontrollability of environmental events in general.

Maier and Seligman (1976) believe that an animal is unable to control its environment, response initiation diminishes. The belief in the uncontrollability of one's environment leads to helplessness (Seligman, 1975). Seligman also states that the learned helplessness phenomenon may serve as a model for reactive depression. He draws a parallel between the symptoms, cause, cure, and prevention of depression in man and learned helplessness. Seligman

suggests that this concept of uncontrollability may be the root of both helplessness and reactive depression. This model has also been reviewed by Eastman (1976) and studied empirically by Gatchel, McKinney, and Koebernick (1977). Eastman incorporates the learned helplessness model for depression with other similar models into a more comprehensive model. Gatchel et.al. (1977), on the other hand, reported a difference in electrodermal responses (skin conductance responses) for depressed humans compared to humans with experimentally induced helplessness. These results are interpreted as supporting a difference in underlying mechanisms for learned helplessness and depression despite the similarities.

Seligman (1975) also cites parallels between the therapies typically used for the treatment of depression and their effect upon learned helplessness. A relatively effective therapy used in the treatment of chronic depression has been electroshock therapy (ECT) or electroconvulsive shock (ECS) (Kalinowsky and Hoch, 1961). Seligman cites an unpublished study that reported a disruption of learned helplessness in 3 out of 6 dogs following ECS treatment. Another study using rats also indi-

cated disruptive effects on learned helplessness from a single ECS treatment (Pavlik, 1977).

The study by Pavlik employed 3 groups of rats, those receiving inescapable shock pretreatment, those receiving escapable shock pretreatment, and those receiving no pretreatment (Naive). Twenty-four hours following pretreatment, all animals were tested in a shuttle-box shock-escape task. A significant interference effect in escape responding was found for the animals pretreated with inescapable shock, signified by a negligible decrease in response latency over trials, while control rats learned the task, signified by a more pronounced learning curve. This was interpreted to be a learned helplessness effect. Immediately following shuttle-box testing, half of the animals in each group received a single ECS treatment. All animals were again tested 24 hours later. The results showed a disruption of the learned helplessness effect for the inescapable group treated with ECS. The inescapable group which did not receive ECS remained helpless. The control groups which received no ECS became even more proficient at escaping shock, characterized by shorter latencies than the day 1 test. The control animals

which received ECS treatment, however, performed the same as they did on day 1, that is, they had to relearn the task. These results may be a function of the particular pretreatment stressor, since both the pretreatment and the therapeutic technique involve some form of shock, as well as the test situation.

The present study was designed to investigate the effects of ECS on learned helplessness in the rat and to replicate the effects of Pavlik (1977) using inescapable underwater exposure as the pretreatment stressor. An ECS effect using this pretreatment would provide further evidence for the general effect of learned helplessness in the rat and would extend the results of Pavlik to other pretreatment stressors. This experiment was also designed as a partial replication of the experiment by Altenor et.al. (1977).

Method

Subjects. The subjects were 60 male Sprague-Dawley rats obtained from Ace Breeders in Boyertown, Pennsylvania. They were approximately 90 days old

upon receipt and were housed singly and maintained on a 12 hour light-dark cycle with Purina Rat Chow and water available ad libitum. The animals were randomly assigned to 3 pretreatment conditions; inescapable water (IW), restrained (R), and naive (N). Twenty animals were in each group. All pretreatment and testing were carried out during the light phase of the cycle.

Apparatus. The apparatus for the pretreatment procedures consisted of 3 small animal restrainers manufactured by Plas-Labs in Lansing, Mich. These restrainers (20.5 X 8.1 X 5 cm) were half cylinders with flat bottoms. They were constructed of 3 mm thick clear acrylic plastic with small holes throughout the tube which enabled the cylinder to rapidly fill with water. The restrainers were fixed to a grid platform so that three animals could be immersed at the same time. A 10 gallon capacity aquarium (29 X 24.5 X 29.5 cm) served as the immersion tank. The tank was placed in a deep sink where there was a constant flow of tap water through the tank during pretreatment. The water temperature was 20 ± 1°C.

The test apparatus was a two-way shuttle box (60.96 X 19.05 X 22.6 cm) of 0.64 cm clear acrylic plastic. A clear acrylic plastic divider separated the 2 compartments. A rounded archway was cut out of the bottom of the divider, permitting access to the opposite compartment. This opening was 7.5 cm high and 6 cm wide.

The grid floor was made of 0.32 cm stainless steel bars 1.6 cm apart and was constructed in two parts. The divided floor was hinged at the center and suspended by springs at the extreme ends of the box such that the weight of the animal entering the compartment would cause a downward deflection of the floor and produce a switch closure. Scrambled shocks of 1 mA. were delivered to the grids by a Grason-Stadler shock generator (model E6070B). Response latencies were automatically recorded by an electric timer and a print out counter to the nearest 0.1 second. ECS was administered through the same shock generator.

Procedure. Pretreatment was carried out on the IW and R groups. Animals in the IW group were placed

in restrainers and subjected to 40 inescapable submersion trials, each of which was 10 seconds in duration. Between each immersion trial, there was an intertrial interval of 60 seconds. The subjects were given 5 minutes to adapt to the apparatus before pretreatment began. Animals in the R group were placed in the restrainers for a time equal to that of the IW group, but were not submerged. Naive (N) animals received no pretreatment.

Twenty-four hours following pretreatment, all animals were tested for shock escape in the two-way shuttle box. The latency to escape the shock was recorded for 25 trials with an intertrial interval of 60 seconds. The first 5 trials were FR-1 (animal has to enter opposite compartment to escape shock) and the remaining 20 trials were double barrier or FR-2 (animal has to enter opposite compartment then reenter original compartment to terminate shock). If the animal failed to escape shock within 20 seconds, the current was automatically terminated and a failure to escape was recorded. There was no avoidance contingency for the shock escape task.

Immediately (within 5 minutes) following test,

half of the subjects in each group were given a single ECS treatment of 40 mA. intensity for 1.0 sec. (ECS group). The shock was administered through alligator clips attached to the subjects' pinnae. For the remaining subjects (NECS group), the ear clips were attached, but no current was delivered. All animals were returned to their home cages following treatment.

Twenty-four hours following treatment, the six groups of animals (IW-ECS, IW-NECS, R-ECS, R-NECS, N-ECS, and N-NECS) were again tested in the shuttle box for shock escape as on the previous day.

On day 1, the performance of the IW group was compared to the performance of the R and N groups. On day 2, the ECS groups were compared to their corresponding NECS groups for an ECS effect by a series of planned comparisons.

Results

The results of the shock escape task for both experimental days are shown in Figure 1 with latency measurments presented in blocks of 5 trials. The first block for each day contains the 5 FR-1

trials. The remaining 4 blocks are the FR-2 trials.

Table 1 contains the analysis of variance summary table for the FR-1 block for day 1. No significant differences were obtained for the comparisons between the N and R groups or between the IW group and the N + R groups.

Table 2 summarizes the analysis of variance and comparisons of the 4 blocks of FR-2 trials for day 1. Comparisons indicated that the IW group performed significantly different over the 4 blocks of trials than did the N and R groups. The IW group exhibited an increase in response latency over trials whereas the R and N groups showed either a decrease or no change in response latency across trials $\underline{F}$ (3,171)= 7.475 ($p < .001$). The R group did not differ from the N group in latency to escape shock over the 4 trials. The overall pretreatment effect was also significant, $\underline{F}$ (2,57) = 8.456 ($p < .001$). There was no significant main effect for blocks.

The analysis of variance for the FR-1 block for day 2 is summarized in table 3. No differences were found between groups N and R, however, the IW groups (IW-ECS and IW-NECS) were significantly

Figure 1

Mean escape response latency for rats in a shuttle box shock escape task before (day 1) and after (day 2) ECS treatment. The first block for each day is FR-1, the remaining blocks are FR-2.

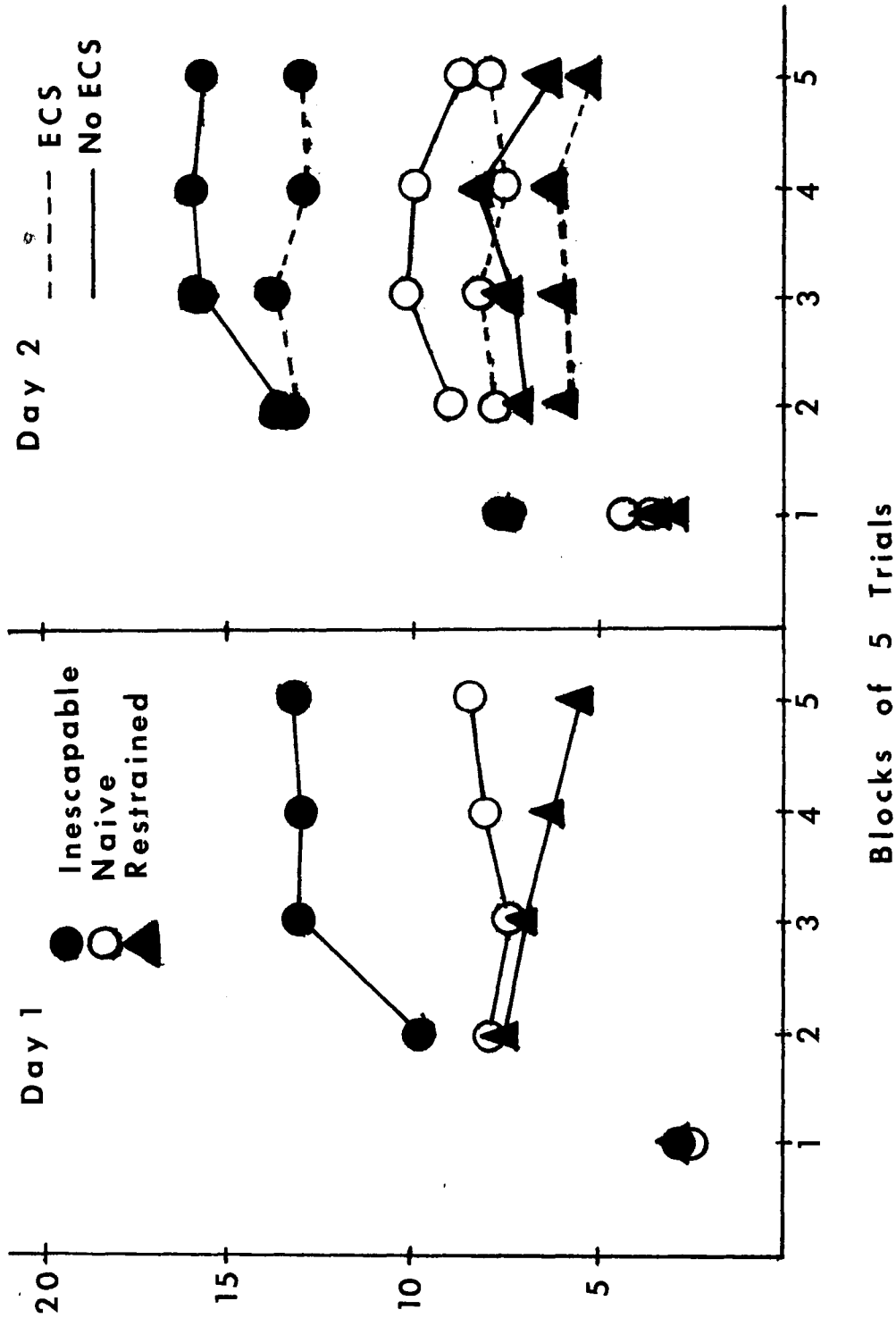
Day 1
Day 2
Inescapable
Naive
Restrained
ECS
No ECS
20
15
10
5
1
2
3
4
5
Blocks of 5 Trials
MEAN RESPONSE LATENCY (SECONDS)

Table 1

Summary of the Analysis of Variance for Response Latency Data of FR-1 Trials on Day 1.

Source	SS	df	MS	F	(df)
Pretreatment	1.02	2	0.51		
Comparisons					
N vs. R	0.595	1	0.595	<1	(1,57)
IW vs. N + R	0.425	1	0.425	<1	(1,57)
Error	215.250	57	3.780		
Total	216.270	59			

Table 2

Summary of the Analysis of Variance for Response Latency Data of FR-2 Trials on Day 1.

Source	SS	df	MS	F (df)
Pretreatment	1306.30	2	653.15	8.456*(2,57)
Blocks	22.06	3	7.35	1.188 (3,171)
Pretreat X Blks	182.93	6	30.49	
Comparisons				
N vs. R X Blks	44.12	3	14.71	2.376 (3,171)
IW vs. N+R X Blks	138.81	3	46.27	7.475*(3,171)
Subjects	4402.87	57	77.24	
Blks X Subjects	1057.84	171	6.19	
Total	6972.00	239		

* $p < .001$

Table 3

Summary of the Analysis of Variance for Response Latency Data of FR-1 Trials on Day 2.

Source	SS	df	MS	F	
Pretreatment	194.120	2	97.06		
Comparisons					
N vs. R	4.820	1	4.82	<1	(1,54)
IW vs. N + R	189.300	1	189.30	9.923*	(1,54)
Treatment (ECS)	0.296	1	0.30	<1	(1,54)
Pretreat X Treat	3.466	2	1.73	<1	(1,54)
Error	1030.200	54	19.08		
Total	1227.800	59			

* $p < .005$

Table 4

Summary of the Analysis of Variance for Response Latency Data of FR-2 Trials on Day 2.

Source	SS	df	MS	F (df)
Pretreatment	2511.567	2	1255.78	12.46*(2,54)
Treatment (ECS)	163.450	1	163.45	1.62 (1,54)
Blocks	31.361	3	10.45	2.19(3,162)
Pretreat X Treat	3.764	2	1.88	$\leq$1 (2,162)
Pretreat X Blks	20.524	6	3.42	$<$1 (6,162)
Treat X Blks	17.868	3	5.96	
Pretreat X Treat X Blks	12.940	6	2.16	
Comparisons				
IW-ECS vs. IW-NECS X Blks	19.815	3	6.61	1.382(3,162)
N-ECS vs. N-NECS X Blks	9.137	3	3.05	$<$1 (3,162)
R-ECS vs. R-NECS X Blks	1.856	3	0.62	$<$1 (3,162)
Subjects	5441.559	54	100.77	
Blks X Subjects	774.214	162	4.78	
Total	8977.246	239		

* $p < .001$

slower to escape shock than the N and R groups, F (1,54) = 9.923 ($p < .005$).

Table 4, which summarizes the analysis of variance for the performance data of the FR-2 blocks for day 2, continues to show a significant pretreatment effect, F (2,54) = 12.46 ($p < .001$). There was, however, no significant block effect and no overall treatment (ECS) effect. Comparisons, over blocks, between IW-ECS, and IW-NECS, R-ECS and R-NECS, and N-ECS and N-NECS showed no significant differences, that is, no differences in learning of the shock escape task over the four trial blocks between the ECS groups and their corresponding NECS groups. However, in all cases, the escape latency for the ECS groups was somewhat shorter than for the corresponding NECS groups.

In summary, the results indicate a significant interference effect in response latency for the IW group on days 1 and 2, although no differences were observed between the groups receiving ECS and their corresponding groups which received no ECS, on day 2.

Discussion

An interference effect was produced for the FR-2 trials on day 1, using inescapable underwater exposure as the pretreatment stressor. These results are in agreement with those of Altenor et. al. (1977) and extends their results by using a naive control group. The naive control group is assumed to occupy a zero point or baseline from which a pretreatment effect is compared. Of course, the use of the naive group also introduces a confounding since exposure to the stressor itself may modify subsequent behavior. Maier et.al. (1969), however, found no differences in response latency for dogs given escapable pretreatment compared to naive dogs (no pretreatment).

The performance of the three groups on the FR-1 trials for day 1 is consistant with the results of Maier et.al. (1973). Maier found that the single barrier crossing (FR-1) is not a sensitive measure for interference effects following pretreatment with uncontrollable events. In the present experiment, the three groups of rats did not differ in response latency for the FR-1 trials on day 1

(see Figure 1). There were, however, reliable differences in performance for the FR-1 trials on day 2. The IW group, in both the ECS and NECS treatment conditions showed significantly longer latencies to escape shock. These results are in contrast to those of Maier et.al. (1973) and the results on day 1 of the present experiment. The fact that this retarded shock escape acquisition extends to the FR-1 trials on day 2 suggests that the pretreatment stressor alone is not responsible for the interference effect produced on day 1.

The significant pretreatment effect was again present for the FR-2 trials on day 2 (retest). This suggests that despite 48 hours following pretreatment, the interference effect remains. This may also be due to the compounding of failure to escape shock as described above. This result is consistant with those of Seligman and Groves (1970) for dogs, and Seligman, Rosellini, and Kozak (1975) for rats, that this interference effect does not dissipate in time.

This prolonged interference following inescapable pretreatment would then be inconsistent with

one of the alternative explanations of learned helplessness. This hypothesis states that the retarded shock escape acquisition following inescapable pretreatment is produced by a decrease in brain norepinephrine levels, which in turn, cause a motor deficit preventing escape (Weiss, Stone, and Harrell, 1970; Weiss, Glazer, and Pohorecky, 1976). Maynert and Levi (1964) also found a stress induced depletion of brain norepinephrine. Weiss et.al. (1970) suggested that their results may explain the interference effect associated with the learned helplessness hypothesis. Maynert and Levi, however, reported a 40% decrease in brain norepinephrine followed exposure to a series of inescapable shocks, although within an hour, brain norepinephrine had risen to pre-experimental levels. Since the interference effect, in the present study, was present for at least 24 hours, this motor activation deficit hypothesis is unable to explain the present results.

Recently Glazer and Weiss (1976) have attempted to explain the long-term interference effects following inescapable shock pretreatment. This

explanation has been termed "learned inactivity" and states that animals exposed to inescapable shocks of at least five seconds in duration are retarded in acquiring a shock escape task because they learn to become inactive in the presence of subsequent electric shocks. This hypothesis has only been advanced in relation to shock pretreatment and shock test. At this point, learned inactivity could not adequately explain the results obtained in the present experiment where underwater exposure was used as the pretreatment stressor. This hypothesis also would be unable to explain the results of Altenor et.al. (1977), who also used underwater exposure as a pretreatment stressor.

Another somewhat related hypothesis advanced by Bracewell and Black (1974) has to do with competing motor responses. A series of experiments performed by Bracewell and Black indicated that animals restrained and shocked were retarded in subsequent acquisition of a shock escape task. High, fixed intensity, noncontingent preshock and lower intensity movement contingent preshock also retarded subsequent acquisition of a shock escape

task. These data were interpreted as supporting the competing motor response hypothesis which postulates the learning of other responses by the animal during pretreatment which interfers with the response the animal has to make to escape shock. Bracewell and Black also found an interference in shock escape acquisition following restrained, but not shocked, animals. The present results indicated that the restrained animals did not differ from the naive controls in latency to escape shock. Maier et.al. (1973) also found restrained animals not to differ from controls in response latency in a shock escape task. Furthermore, the response competition hypothesis, like the learned inactivity hypothesis described above, is described only in relation to shock pretreatment and shock test, and does not generalize across other pretreatment or test dimensions as does the learned helplessness hypothesis. In light of the arguments presented above, the present results seem to be incompatable with the response competition theory proposed by Bracewell and Black (1974).

The discussion above demonstrates the incompatability of the learned inactivity hypothesis and the

response competition hypothesis with the results obtained in the present experiment. Therefore, the performance of the experimental animals on day 1, may be attributed to learned helplessness.

The most important finding in the present experiment is that ECS does not disrupt the interference effect produced by inescapable underwater exposure. These results are inconsistant with the results obtained by Pavlik (1977). Pavlik found, upon retesting animals 24 hours after ECS treatment or no ECS treatment, that animals which had been pretreated with inescapable shock and had not received ECS remained helpless, while animals who received inescapable shock and ECS learned the task even though they failed to learn the task on the previous day. Control groups (escapable shock pretreated animals and naive animals) treated with ECS showed heightened latencies on the initial shock escape trials, but exhibited decreased latencies on later trials, signifying forgetting and subsequent relearning of the task. Control groups which had not received ECS continued short response latencies. The results of the present experiment, on the other hand,

show no such effect. There was, however, a non-significant tendency for the ECS groups to have slightly shorter escape latencies than the NECS groups. This was true for all pretreatment conditions (see Figure 1).

The literature on ECS describes two types of effects of ECS or ECT: (1) therapy for depression (Kalinowsky and Hoch, 1961), and (2) amnesic effects (Lewis and Nicholas, 1973; Kesner and D'Andrea, 1971; Adams and Lewis, 1962; Hudspeth, McGaugh, and Thomson, 1964; Routtenberg and Kay, 1965). Both of these effects seem to be operating in the experiment by Pavlik (1977).

The learning of independence between responses and outcome occurs during pretreatment, since animals tested 5 minutes, 1 hour, and 4 hours following inescapable shock pretreatment were retarded in acquiring the test task (Seligman et.al., 1975). Wiener (1970) reported that rats given ECS 24 hours following the acquisition of an instrumental task showed little retrograde amnesia for that task. This suggests that retrograde amnesia could not explain the results of Pavlik since ECS was given immediately following

the first shuttle box test trials, 24 hours after pretreatment. The explanation for the disruption of the interference effect following ECS treatment would then have to be explained by the antidepressive effects of ECS. Seligman (1975), in his discussion on the learned helplessness model for depression states that learned helplessness is most characteristic of reactive depression as opposed to chronic depression. This dichotomy was proposed by Kiloh and Garside (1963). Reactive or neurotic depression stems from a reaction to a stimulus. Endogenous depression or chronic depression, on the other hand, is more deeply rooted, is more severe, and is characterized by different symptoms and response to treatment than the reactive form. Mendels (1965) found ECT to be effective in reducing only endogenous depression and having little, if any, effect upon reactive depression. There is, however, extensive overlap between the symptoms of these two forms and a clear dichotomy has not been established (Mendels, 1965).

Since learned helplessness resembles the reactive form of depression then ECS should not be an

effective therapy to alleviate learned helplessness. The above evidence is consistant with the results of the present experiment and in contrast to the results of Pavlik (1977). The possibility exists that Pavlik may be observing some phenomenon other than learned helplessness.

The failure to find an ECS effect on learned helplessness in the present study compared to the robust effect reported by Pavlik may have been due to procedural differences between the two studies. Pavlik used both shock pretreatment and shock test while in the present study, inescapable underwater exposure was used as the pretreatment stressor. These aversive stimuli may contribute differently to the interference effect observed, although Myer (1971) states that the behavioral effects of underwater exposure are similar to the effects of shock as far as producing active attempts to escape during pretreatment and test situations. It would then be reasonable to assume that shock and underwater exposure have similar properties for learned helplessness. A second procedural difference between the present experiment and that of Pavlik

is the maximum amount of shock allowed for the shuttle escape task. Pavlik used 60 seconds of shock maximum whereas, in the present study, only 20 seconds were used. A ceiling effect, in the present experiment, may have prevented differences between the IW-ECS and the IW-NECS groups. A third procedural difference lies in the duration of the ECS treatment used. Pavlik used 40 mA. of current for 500 msec, whereas the present study used 40 mA. for 1 sec. This two-fold increase in ECS duration may have produced deleterious effects in the animals, although evidence from the present experiment and other studies do not support this view. This duration was used, in the present experiment, because it reliably produced convulsions in the animals, characterized by extensor tonus for approximately 12 seconds after ECS administration. No rats died during the course of the experiment and no deleterious effects were observed. Also, Buckholtz and Bowman (1972) reported increases in ECS from 400-1600 msec only marginally increased retrograde amnesia, although a strong duration effect on retrograde amnesia was evident for sub-maximal

levels of ECS (5 msec). Paolino, Quarterman, and Levy (1969) used 100 mA. of ECS for 800 msec and found no significant alteration of retrograde amnesia from 100 mA. ECS delivered for 200 msec. The results of these studies suggest that at supra-maximal levels of ECS, minimal effects of duration occur. The duration of ECS used in the present study should not have any effects different from the duration used by Pavlik (1977). It is possible that the failure, in the present investigation, to find an ECS effect is related to some combination of procedural differences from Pavlik, though just how is unclear. Further research on the effects of ECS on learned helplessness should include a replication of Pavlik (1977) using both shock and underwater exposure as pretreatment stressors.

In summary, the results of this experiment have shown no effect of ECS on learned helplessness which is inconsistent with the results of Pavlik (1977) and that this discrepancy may be due to procedural differences between the two studies. The lack of an interference effect produced for the FR-1 trials

on day 1 is consistant with those reported by Maier et.al. (1973). The FR-2 trials, however, produced a significant proactive interference effect for those animals pretreated with inescapable underwater exposure. These results were taken as evidence for the learned helplessness hypothesis, when compared to the alternative hypotheses of depletion of brain norepinephrine, learned inactivity, and competing motor responses. The results were also consistent with those of Altenor et.al. (1977) who reported a generalization of the learned helplessness effect across different parameters, namely shock and water. The significant interference effect produced on the FR-1 trials of day 2, for animals pretreated with inescapable underwater exposure was attributed to the combined effect of the pretreatment and the retarded escape response acquisition on day 1.

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