LEARNED HELPLESSNESS IN RATS: THE EFFECTS OF ELECTROCONVULSIVE SHOCK IN AN ANIMAL MODEL OF DEPRESSION

THESIS

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By

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The response deficit following exposure to inescapable shock has been termed "learned helplessness." This experiment was designed (a) to determine whether learned helplessness following an inescapable footshock induction procedure extends to 48 hours, and (b) to test the hypothesis that electroconvulsive shock (ECS) reverses learned helplessness in rats.

Subjects were tested for helplessness in a bar-press shock-escape task. Results indicated that helplessness was not present 48 hours after exposure to inescapable shock. A slight indication of helplessness was observed in the first 10 trials of the 60-trial task. In addition, ECS was shown to enhance performance in the test task; however, this facilitation effect was seen only in control animals that were not previously exposed to inescapable footshock.
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LEARNED HELPLESSNESS IN RATS: THE EFFECTS OF ELECTROCONVULSIVE SHOCK IN AN ANIMAL MODEL OF DEPRESSION

Psychological depression has been encountered in clinical practice more than any other mental disorder (Beck, 1967). The National Institutes of Mental Health have estimated that 15% of the adults in the United States—about 20,000,000 people—may suffer from serious depressive disorders in any given year (Gallant & Simpson, 1976). Not only has depression been a leading cause of human suffering but its by-product, suicide, has been ranked among the top 10 causes of death in the United States and has held "number one" status in certain age groups (Beck, 1970).

With the introduction of electroconvulsive shock (ECS) therapy in 1939 and subsequent improvements in its administration (Goldman, 1962), some progress has been made in the treatment of depression. Nevertheless, our understanding of the nature and etiology of depression has not kept pace with the advances in treatment. Many theories of depression have been proposed (Flach & Draghi, 1975). But, because of ethical considerations, much of the research necessary for a systematic investigation of the theories could not be accomplished using human subjects. In many cases such
research would be impossible if an adequate animal model of depression were available (Heath, 1976).

Animal models of human pathology have proven to be invaluable in medical research. For the experimenter who proposed to induce, observe, and manipulate a serious pathological condition in living subjects, an available animal model has often been a necessary prerequisite. Since the use of animal models has met with a great deal more skepticism and resistance in psychology than in medicine (Kubie, 1939), animal models of psychological disorders such as depression have only recently become widely accepted (Heath, 1976).

McKinney (1976) has suggested the following criteria for use in evaluating the adequacy of animal models of human pathology:

1. The behavioral manifestations of the syndrome being modeled should be similar to those seen in the human condition.
2. These behavioral changes should be capable of being objectively detected by independent observers and different laboratories.
3. The behavioral state induced should be persistent and generalizable.
4. Conditions which produce abnormal behavior in animals should be similar to those etiologic conditions responsible for human psychopathology.
5. Treatment modalities effective in reversing the human disorder should be effective in primates.

6. There must be sufficient reference control data available on the species under study. (p. 3)

While performing an experiment on the relationship between fear conditioning and instrumental learning, Overmier and Seligman (1967) discovered a phenomenon of animal behavior which has since been seriously suggested as an animal analog of human depression. In this experiment, dogs were restrained in a Pavlovian hammock and given classical conditioning with tones followed by electric shocks. These dogs then were tested in a shuttle box where they were required to jump a low barrier dividing the box in order to escape or avoid shock.

Contrary to their expectations, prior experience with inescapable shock did not result in better performance in the shuttlebox escape-avoidance test task. Instead, after a brief period of frantic and disorganized reaction to the onset of shock, these dogs typically ceased responding and lay down whining. Even when a dog accidentally jumped the barrier and escaped shock in one trial, he failed to escape in later trials. To account for this unusual behavior, Seligman hypothesized that the response deficit resulted from prior exposure to inescapable shock. An equal amount of escapable shock, he felt, would not produce such a deficit.
To test this hypothesis, Seligman and Maier (1967) constructed a simple three-group (triadic) experimental design to compare the effects of an identical amount and pattern of shock delivered to animals under escapable and inescapable conditions. In the triadic design one group of dogs received shock which they could control by pressing a panel. A second group was yoked to the first with each dog in the second group experiencing exactly the same physical outcomes (shock) as his counterpart in the controllable-shock group, but without control over the shock. A third group received no shock. All groups were tested in a shock escape/avoidance barrier-jump task 24 hours after treatment. Although receiving the same pattern and duration of shock as group two, the controllable-shock group readily learned and performed the barrier-jump while the uncontrollable-shock group demonstrated the same helpless behavior described in the original study. Dogs in the uncontrollable-shock group were significantly slower to escape/avoid than the dogs in the controllable-shock and no-shock groups. Six of the eight dogs in the uncontrollable-shock group failed completely to escape shock.

In subsequent research using the triadic design, uncontrollable aversive treatment has produced helplessness in goldfish, pigeons, mice, rats, cats, dogs, and humans (Braud & Russo, 1969; Dorworth & Overmier, 1977; Maier & Testa, 1975; Overmier, 1968; Padilla, 1973; Seligman, 1974;
and Welker, 1976). In addition, helplessness has been shown to transfer readily from one type of situation to another. For example, humans exposed to inescapable loud noise later demonstrated helplessness in a hand-shuttle task (Hiroto & Seligman, 1974). Rats exposed to uncontrollable immersion in water have demonstrated a later deficit in a shock-escape task, and rats first exposed to uncontrollable shock have shown a later deficit in a water-escape task (Altenor, Kay, & Richter, 1977). Generality and transferability have been considered essential to the animal model because human depression has been shown not to be specific to any particular situation or variety of traumatic events (Beck, 1967).

Seligman's explanation of the helplessness phenomenon has hinged on the subject's learning that his responses can or cannot control significant outcomes. According to Seligman (1974), helpless animals have learned that the probability of reinforcement granted the occurrence of a response equaled the probability of reinforcement granted the nonoccurrence of a response. In such a case, the outcome (reinforcement) was uncontrollable, and responding ceased because the incentive to respond (expectation that responding may succeed) was absent.

Human depression has not been carefully defined. Instead, it has been a confusing and often controversial syndrome (Flach & Draghi, 1975), and its relationship to
learned helplessness must be determined primarily on the basis of similarity and plausibility (Wolpe, 1967). Seligman (1974) has described the following symptoms of learned helplessness and pointed out the similarity with certain of the more commonly accepted symptoms of human depression: (a) lowered initiation of voluntary responses, (b) negative cognitive set, (c) lowered aggression, (d) loss of appetite, (e) physiological change, and (f) time course.

Animals and humans who have experienced uncontrollability have shown reduced initiation of voluntary responses (Seligman, Klein, & Miller, 1974). In severe depression, patients have often shown no desire to do anything, even those things which were essential to life. This has been described as a paralysis of the will (Beck, 1967) or a negative cognitive set (Bensen & Kennelly, 1976). During a severe depression these people believed themselves to be even more ineffective than they actually were (Friedman, 1964). They saw small obstacles to success as impassable barriers and viewed difficulty in dealing with a problem as complete failure. In fact, they often misconstrued success as failure.

Helpless animals and men have been shown to initiate fewer aggressive and competitive responses, and their dominance status diminished (Kurlander, Miller, & Seligman, cited in Seligman, 1975). The absence of overt hostility in depressed patients led Freud (1917/1957) to consider this the basis of depression. The "Tuscaloosa Plan" (Taulbee &
Wright, 1971), a program of systematic harassment designed to
provoke and reward angry responses, has recently been shown
to be effective in the treatment of human depression.

Helpless animals have shown a loss of appetite, a loss
of weight, and a lessening of sexual and social abilities
(Suomi & Harlow, 1972). A reduction in gratification has
been one of the most common symptoms of human depression.
In a systematic study of depressive symptoms (Beck, 1967),
this reduction was observed in 92% of the severely depressed
patients. For these patients, food, sex, and social activ-
ities (friends, work, etc.) lost their pleasurable
properties. In mild cases, patients often compensated for
this initial loss by increasing the satisfaction gained from
activities involving less of a sense of duty or responsi-
bility (giving) and more of a tangible and easily obtained
satisfaction (getting), such as recreation or relaxation.
Often depressed persons sought unusual or regressive
activities in order to regain some of their former thrills.
One patient reported that he could always pull himself out
of a mild depression by watching a performance of deviant
sexual practices. As depression became more severe, how-
ever, even these activities eventually became boring.

Physiological changes in helpless animals have been
shown to parallel those seen in depressed humans. The
neurotransmitter norepinephrine (a brain catecholamine
suspected to mediate the motivation to respond) has been
shown to be depleted in rats subjected to uncontrollable stress (Weiss, Stone, & Harrell, 1970). The most prominent theory of the physiological basis of human depression has been the "catecholamine hypothesis" (Kraines, 1957; Schildkraut, 1965). The evidence linking a catecholamine deficit, specifically a norepinephrine deficit, with human depression has been indirect. First, two kinds of "antidepressant" drugs, the monoamine oxidase inhibitors and the tricyclics, functioned by keeping norepinephrine available in the brain (Cole, 1964). Also, there is some evidence that, at least in the rat, progressively administered ECS resulted in an increased turnover of norepinephrine (Ebert, Baldessarini, Lipinski, & Berv, 1973; Kety, Javory, Thierry, Julou, & Glowinski, 1967; Schildkraut & Draskoczy, 1974). These results have suggested a possible explanation for the 70% to 90% effectiveness of ECS (Beck, 1967; Kalinowski, 1959) in the treatment of human depression.

Almost all depressions have shown a specific time course (Kraines, 1957). In depressions labeled "endogenous," in which there was no obvious precipitating event, mood often cycled with regularity. In depressions labeled "reactive," in which prior trauma was evident, mood was self-limiting; thus it was therapeutically important for the patient to know that the despair would lift if he waited long enough (Seligman, 1975). Animal helplessness has also been shown to have a time course and, in some cases, the
deficits resulting from uncontrollable aversive treatment dissipated within 48 hours (Overmier & Seligman, 1967). In other cases the deficits produced persisted for as long as a week (Seligman, Rosellini, & Kozak, 1975).

Seligman has stated that multiple induction sessions resulted in helplessness which was relatively permanent. The short-term deficits seen in the dog studies (Overmier & Seligman, 1967) became more persistent when the induction procedure was administered more than once (Seligman, 1975). However, Seligman concluded that helplessness in the rat, in contrast to that in the dog, did not dissipate for up to 7 days following a single session of inescapable shock (Seligman, Rosellini, & Kozak, 1975).

Glazer and Weiss (1976), on the contrary, have found that the effects of uncontrollable shock have a definite time course in the rat. Depending on the intensity and duration of inescapable shock, both transitory and long-term deficits have been demonstrated. When inescapable shock was delivered to rats under moderate intensity/long duration conditions, performance deficits were not present after 30 minutes but were seen 48 hours, 72 hours, and 7 days after induction. However, response deficits after high intensity/short duration shock were found 30 minutes after treatment but dissipated within 48 hours. These results seemed to suggest that the specific form of helplessness, either
transitory or relatively persistent, depended on the specific parameters of the helplessness induction procedure.

"Disaster syndrome" in humans may have been similar to the transitory form of helplessness which, according to Glazer and Weiss (1976), resulted from high intensity/brief duration trauma. For example, Wallace (1956) observed that after a tornado many people became nearly stuporous for about 24 hours. The symptoms of "disaster syndrome" usually dissipated within 24 to 48 hours, and only if they persisted for several days or weeks longer would the person be considered to have suffered from "reactive" depression. The rapid dissipation of these symptoms suggested a purely physiological response to stress rather than the learning of response-outcome independence (Miller & Weiss, 1969), since learned responses have generally been more persistent (Miller, 1967; Skinner, 1950).

If a persistent learned helplessness in the rat were indeed an adequate model of human depression, then treatment modalities effective in reversing depression should also have been effective in reversing learned helplessness (McKinney, 1976). Electroconvulsive shock (ECS) therapy has been an effective treatment for human depression (Beck, 1967), but previous attempts to evaluate the effect of ECS on learned helplessness have not been convincing. Dorworth & Overmier (1977) found some evidence that ECS reversed helplessness in dogs, but these results were not clearcut.
A significant reversal of helplessness in the rat has been found after administration of ECS (Porsolt, Anton, Blavet, & Jaffre, 1978; Porsolt, Pichon, & Jaffre, 1977). However, in these studies ECS was administered immediately after helplessness induction, leaving their results open to an alternative explanation. Since ECS has been shown to produce retrograde amnesia (McGaugh & Herz, 1972), memory for the response-outcome independence experienced during helplessness induction may simply have been disrupted before the memory consolidation process was complete.

In order to test the effectiveness of ECS in reversing learned helplessness in the rat, researchers first devised an induction procedure in which, unlike yoked induction, the treatment parameters (pattern, intensity, and duration of shock) could be held constant for all subjects (Lambert, Harrell, & Emmett-Oglesby, 1978). This was accomplished by first administering moderate intensity (2 mA) footshock to yoked pairs of rats using the standard induction procedure of the triadic design (Seligman, 1975). Testing was conducted 24 hours later in an FR2 barpress escape task. As in other helplessness studies, the rats exposed to inescapable shock performed significantly worse in the test task than rats that did not receive shock. The yoked escapable-shock group, however, showed no such deficit.

Next, the duration of shock received during induction by the yoked pairmates was averaged across trial blocks. On
the basis of these averages a graduated series of programmed, inescapable shock was developed. By simply administering this programmed series of inescapable shocks to rats, researchers could induce helplessness without the use of the cumbersome yoking procedure. Thus, the within-group variations in treatment parameters inherent in the triadic design were eliminated. And, since rats receiving roughly the same treatment under controllable conditions were shown not to be helpless in the test task, it seemed reasonable to assume that deficits resulting from this induction procedure reflected learned helplessness rather than the effects of shock per se.

Rats exposed to the Lambert et al. helplessness induction procedure were divided into two groups. One group received a single ECS treatment 23 hours after helplessness induction. The other group received sham ECS treatment. One hour later both groups, as well as a no-shock control group, were tested in an FR2 barpress escape task. The sham ECS group performed significantly worse in the test task than the no-shock control group; i.e., the sham ECS group was helpless. The rats which received ECS, however, showed no such deficit. Thus, ECS reversed learned helplessness 24 hours after administration of the Lambert et al. procedure.

The purpose of the present study was to test the effects of the Lambert et al. helplessness induction procedure on escape performance 48 hours after induction and to
determine whether ECS administered 47 hours after induction would reverse the expected response deficit. This experiment was designed to test two specific hypotheses.

1. Rats exposed to the helplessness induction procedure would perform significantly worse than rats that received no footshock (higher mean escape latency) in an FR2 barpress escape task administered 48 hours after helplessness induction.

2. Inescapably shocked rats that received ECS 1 hour prior to testing would perform significantly better (lower mean escape latency) than inescapably shocked rats that did not receive ECS.

Method

Subjects

The subjects were 32 male rats (ARS Sprague-Dawley, Madison, Wisconsin), 60 days old at the beginning of the experiment, that were individually housed and maintained on ad libitum food and water throughout the experiment. Of the rats given ECS, 50\% (15 of 30) were paralyzed by the treatment and had to be replaced. Thus, 47 rats were required to complete the experiment.

Apparatus

Training and testing were conducted in a modified shuttlebox (BRS/LVE, Lehigh Valley Electronics) which had a floor of stainless steel bars wired to a constant-current electric shock generator (Model SGS/003, BRS/LVE). A
partition divided the shuttlebox into two chambers, each measuring 43.2 cm long by 30.3 cm wide by 23.5 cm deep. For the test phase, a lever was inserted 3.2 cm into the end wall of the shuttlebox. A 25-gm weight depressed the lever and closed a microswitch. Recording of microswitch closures and schedule programming were carried out with combinations of BRS/LVE solid-state and electromechanical equipment. A Lafayette A-615B Electroconvulsive Shocker (Lafayette Instrument Company) was used to administer ECS.

**Procedure**

Each of the rats was randomly assigned to one of the four groups which received inescapable footshock and ECS, inescapable footshock and no ECS, no footshock and ECS, or no footshock and no ECS. The shock/ECS and shock/no ECS groups received 90 trials of inescapable, scrambled, 2-mA pulsating footshock (0.5 seconds off) delivered on a variable schedule. The inter-trial interval had a mean of 90 seconds. Shock duration was 60 seconds for the first 10 trials, 15 seconds for the next 10 trials, and 5 seconds for the last 70 trials (Lambert et al., 1978). The no shock/ECS and no shock/no ECS groups were placed in the shuttlebox for a period of time equivalent to that spent by the rats in the shock/ECS and shock/no ECS groups, but they received no footshock. All rats were placed under a bell jar and subjected to ether anesthesia 47 hours later. Upon loss of the righting reflex, each animal's head was shaved and alligator
clip electrodes were fastened to the scalp adjacent to the ear. A single 50-mA ECS lasting 2 seconds was then administered to rats in the shock/ECS and no shock/ECS groups. Rats in the shock/no ECS and no shock/no ECS groups were anesthetized, shaved, and alligator clips were attached, but no ECS was delivered to them.

All rats were individually tested in the shuttlebox 48 hours after induction. The lever was inserted into the chamber, and all rats received 60 trials of FR2 barpress shock-escape training. The intertrial interval and shock parameters were identical to those used in the induction phase. An FR2 barpress after shock onset terminated shock. Barpresses during the intertrial interval had no programmed consequences. If an FR2 barpress did not occur within 60 seconds of shock onset, the trial was terminated. The mean latency to complete the FR2 barpress was used to measure shock-escape efficiency.

**Results**

The mean latency to escape for each block of 10 trials is presented in Figure 1. This graphic representation seems to indicate that the no shock/ECS group is superior to the other three groups. The statistical analyses support this conclusion.

The summary of a 2 X 2 X 6 analysis of variance is presented in Table 1. The main effects for ECS ($F = 5.98$, $p < .02$), footshock ($F = 4.07$, $p < .05$), and trial blocks
Figure 1. Mean response latency to escape for each block of 10 trials. (Closed circle = no footshock/no ECS; open circle = no footshock/ECS; closed triangle = footshock/no ECS; open triangle = footshock/ECS.)

(\(F = 4.51, p < .0007\)) are all statistically significant.
The only significant interaction is between ECS and footshock (\(F = 4.80, p < .03\)).

Because the only significant interaction is between ECS and footshock, the results of a 2 X 2 analysis of variance are presented in Table 2. Both the main effect of ECS (\(F = 5.98, p < .02\)) and the main effect of footshock (\(F = 4.07, p < .05\)) are significant. The interaction is also significant (\(F = 4.80, p < .03\)). A post-hoc Scheffe's \(F\) test indicates that the only significant differences are between the no shock/ECS group and the other three groups.
Table 1
Summary of 2 X 2 X 6 Analysis of Variance

<table>
<thead>
<tr>
<th>Source</th>
<th>Sum of Squares</th>
<th>df</th>
<th>Mean Squares</th>
<th>F</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>Between subjects</td>
<td>57352.929</td>
<td>31</td>
<td></td>
<td></td>
<td></td>
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<tr>
<td>ECS</td>
<td>8012.984</td>
<td>1</td>
<td>8012.9840</td>
<td>5.9894*</td>
<td>0.02092</td>
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<tr>
<td>Foot shock</td>
<td>5455.362</td>
<td>1</td>
<td>5455.3616</td>
<td>4.0777*</td>
<td>0.05312</td>
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<tr>
<td>ECS-foot shock</td>
<td>6424.590</td>
<td>1</td>
<td>6424.5896</td>
<td>4.8022*</td>
<td>0.03691</td>
</tr>
<tr>
<td>Within subjects</td>
<td>22241.675</td>
<td>160</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Trial blocks</td>
<td>2811.864</td>
<td>5</td>
<td>562.3727</td>
<td>4.5174**</td>
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<tr>
<td>ECS-trial blocks</td>
<td>360.571</td>
<td>5</td>
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<tr>
<td>Foot shock-trial blocks</td>
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<tr>
<td>ECS-foot shock-trials</td>
<td>867.515</td>
<td>5</td>
<td>137.5029</td>
<td>1.1045</td>
<td>0.36077</td>
</tr>
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</table>

*p < .05.

**p < .01.

Table 2
Summary of 2 X 2 Analysis of Variance

<table>
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<tr>
<th>Source</th>
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<th>df</th>
<th>Mean Squares</th>
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<tbody>
<tr>
<td>ECS</td>
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<td>1</td>
<td>48077.9053</td>
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<td>0.0209</td>
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<tr>
<td>Foot shock</td>
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<td>32732.1485</td>
<td>4.0777*</td>
<td>0.0531</td>
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<tr>
<td>Interaction</td>
<td>38547.5198</td>
<td>1</td>
<td>38547.5198</td>
<td>4.8022*</td>
<td>0.0369</td>
</tr>
</tbody>
</table>

*p < .01.
Discussion

The hypothesis that learned helplessness persist 48 hours postinduction is disconfirmed. The performance of the shock/no ECS group is comparable to that of the control group, and the difference between the means of these two groups is nonsignificant. Lack of a significant helplessness effect makes impossible a test of the hypothesis that ECS reverses learned helplessness 48 hours after induction.

The simplest explanation of these results is apparently that the helplessness present 24 hours postinduction (Lambert et al., 1978) dissipates within 48 hours. It should be pointed out, however, that the shock/no ECS group has the highest mean latency to escape on the first 10 trials of the test task. This suggests that some form of helplessness may be present at the beginning of the test task. Any suggestion of helplessness, however, dissipates by Trial Block 2. It appears that helplessness following the Lambert et al. procedure does not persist for 48 hours postinduction and is, therefore, a transient deficit.

Specific parameters of shock intensity and duration seem to be responsible for transient deficits in rats exposed to uncontrollable shock. Shocks set at 4 mA (high-intensity) and 2 seconds in duration (brief) produce transient deficits while long-duration, low-intensity (1-mA/6-second) shocks result in long-term deficits (Glazer & Weiss, 1976). The shock parameters of the present experiment (2-mA/variable
duration) appear to be similar to high-intensity/brief-duration shocks in producing a transient form of helplessness.

The transient form of helplessness in the present study may be a short-term motivational deficit which reflects an alteration of norepinephrine levels in the rat's brain (Weiss et al., 1970). The persistent form of helplessness, however, is hypothesized to result from the conditioning of inactivity to the offset of shock duration induction. Rats are initially active after the onset of shock but, after 3 or 4 seconds, this initial reaction gives way to a second phase of less active behavior which persists until the offset of shock (Anisman, deCalzano, & Remington, 1978). Inactivity could be conditioned to the offset of shock during this second phase; however, there is little evidence to support this hypothesis in the present experiment.

The duration of shock in the present study ranges from an initial 60 seconds to 5 seconds at the end of the shock series. The mean duration of shock is 15.83 seconds. In view of the findings of Glazer and Weiss (1976) regarding the duration of shock, it might be predicted that the present induction procedure would produce long-term deficits. However, it is also possible that the duration of shock in the present experiment is too long to produce long-term deficits. If such deficits result from the conditioning of inactivity to shock offset (Glazer & Weiss, 1976), maximum conditioning may occur only when the beginning of phase 2
(inactivity) and shock offset are roughly contiguous. In the present procedure, shock offset may occur as much as 55 seconds after the beginning of phase 2.

The one significant effect in this experiment is the facilitation of escape responses resulting from ECS treatment. This is seen only in animals that are not previously exposed to inescapable footshock during helplessness induction. If the effects of the induction procedure have completely dissipated, the shock/ECS and no shock/ECS groups would not be expected to perform differently in the test task. Since the only difference in the treatment of these two groups occurs during the induction phase, some residual effect of inescapable shock may be preventing the ECS facilitation effect in the shock/ECS animals. Whether this result reflects some unusual aspect of helplessness or the effects of shock per se cannot be determined from the present data.

There is no ECS facilitation effect in the previous experiments in this series. Thus, it seems likely that the ECS administered in the present experiment is somehow different. This likelihood is further supported by the fact that no animals are paralyzed by the treatment in the Lambert et al. experiments. The reason for this difference is uncertain, but it is possible that the electrode placement is slightly posterior to that of the previous experiments. This placement may result in more direct
stimulation of the rat's neck and back muscles and an increase in post-seizure excitability.

ECS is known to produce a highly excitable and perhaps supersensitive state in both humans (Beck, 197) and rats (DeVicetti & Larson, 1971). Heightened excitability is a possible explanation for the rapid acquisition of the escape contingency by the no shock/ECS animals, since remaining active and alert after shock onset is important in this test task. The rat's general activity level and shock threshold may not be altered by ECS treatment (Lambert et al., 1978); however, ECS in the present experiment appears to be different from the ECS in the previous studies.

There appears to be no precedent for the ECS facilitation effect observed in the present experiment. Some studies not involving the use of ECS, however, may be useful in interpreting this effect. For example, Cherkin (1974) reports a somewhat similar enhancement of performance, but in his study convulsions were induced in chicks by exposing them to the chemical agent flurothyl (Indoklon). Cherkin speculates that the enhancement of performance in chicks may be similar to the enhancement of memory consolidation resulting from electrical stimulation of the reticular formation reported by Block (1970). Injection of CNS-stimulating drugs prior to training facilitates learning (McGaugh, 1968; McGaugh & Herz, 1972). Electroconvulsive
shock (ECS) may produce a similar CNS-stimulating effect, but such an interpretation is highly speculative.

In summary, both hypotheses of the present experiment are disconfirmed. The results indicate that rats exposed to the Lambert et al. helplessness induction procedure and tested in the FR2 barpress escape task may not be considered adequate as an animal model of human depression. The deficits observed in the test task 24 hours after induction dissipate at the 48-hour test interval. Although human depression often has a definite time course (Beck, 1967), the rapid dissipation of deficits seen in this study would not generally be considered characteristic of human depression. In addition, an unexpected and somewhat puzzling facilitation effect is seen in the group which received ECS and no footshock.
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