THE GENERALITY OF LEARNED HELPLESSNESS THEORY: EFFECT OF ELECTROCONVULSIVE SHOCK

by

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INTRODUCTION

Overmier and Seligman (1967), while doing experiments on the relationship of Pavlovian fear conditioning to instrumental learning, found that dogs given prior exposure to inescapable shock in one situation failed to learn to escape shock in another situation where escape from shock was possible. Two possible explanations were offered to account for these data. First, it was suggested that massive parasympathetic rebound could produce this proactive interference, and second, as an alternative explanation, it was postulated that the interference could be a result of the dogs learning to be "helpless". They further speculated that the learned helplessness might be a result of the organism not being able to control the aversive stimulus.

Later, Seligman and Maier (1967), in a simple but well designed study, demonstrated that the escape/avoidance deficit was a result of the uncontrollability of the original shocks and not a result of shock itself. In this study three groups of dogs were used. The first group, the escape group, was trained to escape shock in a Pavlovian harness by pressing panels located on the sides of the dog's head. The second group, a yoked group, received the same intensity and duration of shock as the escape group but had no control over shock termination. The third group, a naive control group, was strapped in the harness but received no shock. All groups then received 10 trials of shuttlebox escape training, 24 hours following the harness pretreatment.

Both the escape and naive groups learned the task equally well, whereas the yoked group showed an escape/avoidance deficit (i.e., the helplessness effect). These data suggested very strongly that the behavioral deficit found was a result of the organism's lack of control over previous shock and not a result of shock alone.

The two studies presented above marked the beginning of learned helplessness theory. Since then a number of studies have demonstrated that learned helplessness can be obtained in a variety of experimental situations, with different modes of uncontrollable events, and in several different species including man. Since these studies are reviewed in detail elsewhere (see Seligman, 1975 and Maier & Seligman, 1976) they will not be reviewed here; however a review of learned helplessness theory and several key alternative explanations will be presented.

LEARNED HELPLESSNESS THEORY

Recently Maier and Seligman (1976) presented a comprehensive and detailed account of the learned helplessness position. They argued that the key ingredient in learned helplessness is the uncontrollability of aversive events. That is, "when an organism is faced with an outcome that is independent of his responses, he sometimes learns that the outcome is independent of his responses" (p17). This learned uncontrollability purportedly produces three types of disruptions: (a) it reduces the organism's motivation to respond; (b) when an appropriate response is made, the organism fails to perceive an association between reinforcement and response; and (c) it modifies the organism's emotional state. Since these disruptions are assumed to be a direct result of perceived uncontrollability, two questions become important. First, how can one determine that learned helplessness is a result of experiencing uncontrollability as opposed to just experiencing the outcome of an aversive event? Second, what processes are involved in the learning of helplessness?

In answer to the first question, the reader will recall the study by Seligman and Maier (1967) in which three groups were used: an escape group, a yoked group, and a no-shock control group. This "triadic" design is basic to the theory of learned helplessness, especially since it isolates the effects of controllability. For example, the escape group receives as its pretreatment a series of electric shocks which can be terminated by making a specific response; therefore, this group has control over the outcome. On the other hand,

the yoked group receives exactly the same duration and intensity of shock; however, no response will terminate the shock. Thus, this group has no control over the outcome. The third group gets no shock or pretreatment. Later, usually after 24 hours, all groups are tested on a new or different task. If behavioral deficits occur in the yoked group but not in the escape or control groups then one has a direct test of the helplessness hypothesis. That is, the differences would be due to controllability and not a result of electric shock per se.

As for the second question - the processes involved in learned helplessness - Maier and Seligman proposed that learned helplessness theory has three stages. First, the organism must gain information about the response-outcome contingency. In other words, it must receive information that the probability of an outcome is independent of its responses. This is achieved by having the outcome controlled by external agents and not by the receiver of the information. This is specifically the function of the yoked group in the triadic design. The second, and what Maier and Seligman view as the most crucial step, involves the organism processing the information perceived in step one. Therefore, the organism must not only learn that no response-outcome contingency exists, but it must develop an expectation that response and outcome will be independent on new tasks. It is during this second step that a reduction in response initiation occurs. Maier and Seligman state "we assume that the incentive to initiate voluntary responses in a traumatic situation is partly produced by the expectation that responding produces relief" (p18). Thus, when incentive is removed, the

likelihood of voluntary responding will decrease. The third and final step involves the demonstration that the learned expectation (i.e., responses are independent of outcomes) generalizes to new learning situations. This is referred to as the learned helplessness effect.

With one exception, this is basically the theory of learned helplessness. That exception is a recent extension of helplessness theory to emotionality effects. Seligman (1975) proposed that when an organism is first exposed to trauma a heightened state of emotionality occurs. This increased emotionality is usually referred to as fear. According to Seligman, once an organism learns it can control the trauma, fear is reduced; however, if that organism learns it cannot control the trauma, then fear also decreases but is replaced by depression. This is an overly brief statement of Seligman's position on emotionality changes; however, a more detailed discussion will be presented later when the topic of human depression is presented. For now, the discussion will turn to alternative explanations for the learned helplessness effect.

ALTERNATIVE EXPLANATIONS

Several alternative explanations have been offered to account for the learned helplessness effect (e.g., Anisman & Waller, 1973; Bracewell & Black, 1974; Glazer & Weiss, 1976a; 1976b; and Weiss, Glazer, & Pohorecky, 1976). These positions have been proposed mainly to account for the behavioral effects of prior exposure to inescapable aversive stimulations in infrahuman subjects and not for the more general and broader human and infrahuman data presented by Maier and Seligman (1976). Basically these alternative explanations can be broken down into two types of positions: motivational and motor theories. The former, motivational accounts (i.e., adaptation and sensitization to shock), since they present no particular difficulty to the helplessness position, will not be presented here (for a detailed critique of these theories, see Maier & Seligman, 1976). On the other hand, the motor accounts appear to present more difficulty for the learned helplessness position; therefore, a brief review of these positions will follow.

The first hypothesis to be discussed, a competing motor response position, was introduced by Bracewell and Black (1974). These investigators demonstrated that when rats were preexposed to conditions which were arranged to punish movement (i.e., a positive correlation between movement and shock intensity) and later tested on FR-1 shuttlebox acquisition, a decrement in escape/avoidance behavior was produced. These data led Bracewell and Black to proposed that the learned helplessness effect was a result of explicit punishment of movement in the inescapable shock condition, especially since they also found a similar FR-1

decrement when rats were preexposed to either restraint or inescapable (high intensity) shocks alone. Thus, Bracewell and Black's position is basically that animals learn to be inactive, a response which is incompatible with active escape/avoidance responses.

A second competing motor response theory has been proposed by Anisman and Waller (1973). They argue that when an organism is exposed to inescapable shock which decreases the effectiveness of a coping response, then freezing will become the dominant response in that situation. Therefore when the organism is given a subsequent active escape/avoidance task to learn, the freezing response will compete with the active task and produce a decrement in escape/avoidance behavior. Simply stated, they argue the learned helplessness effect is a result of the organism learning to freeze in the presence of electric shock and not that responses are independent of outcome. Anisman and Waller cited a variety of experimental evidence which demonstrates that procedures designed to decrease freezing facilitate active avoidance; whereas procedures designed to increase freezing have a detrimental effect on active avoidance. For example, the administration of anticholinergic drugs such as scopalamine, which reduces freezing, facilitates active avoidance, and the use of high intensity shock, which increases freezing, interferes with active avoidance behavior more than weak or low intensity shocks.

The third and final theoretical alternative to learned helplessness theory is termed the "motor activation deficit hypothesis". This position was first outlined by Weiss, Stone, and Harrell (1970) and later refined by Weiss, Glazer, Pohorecky, Brick, and Miller (1975) and Weiss,

et. al., (1976). According to Weiss et. al., (1976) learned helplessness theory is not an adequate explanation for the behavioral deficit found in animals following exposure to inescapable shock. Aside from the point that helplessness theory infers a cognitive expectation of uncontrollability to explain the behavioral effect, which Weiss and his co-workers claim is not measurable in nonverbal animals, several other factors led Weiss et. al., (1976) to develop the motor activation deficit theory. First, Weiss et. al., (1976) noted with importance that in the original dog experiments (e.g., Overmier & Seligman, 1967; Overmier, 1968) that the helplessness effect dissipated if the dogs were tested 48 hours following exposure to inescapable shock. This dissipation over time, argued Weiss and co-workers, is not consistent with a learning position, especially since learning is considered to be relatively stable over time (e.g., Skinner, 1950; Wendt, 1937). Second, this suggested that inescapable shock induced a time-dependent physiological change, which in turn led Weiss and associates to suspect changes in central catecholamines. Central catecholamine depletion has not only been shown to recover over time (e.g., Rech, Bovys, & Moore, 1966), but has also been shown to greatly affect an animal's ability to initiate an active response (e.g., Herman, 1970; Moore, 1966). Third, Weiss in several experiments (e.g., Weiss, 1971a; 1971b; 1971c) has demonstrated that rats exposed to inescapable shock when compared to rats that receive escapable shock experience a great deal more stress (e.g., gastric lesions, plasma steroid levels, loss of body weight, and other physiological indices of stress). Finally, Weiss, et. al., 1975 reported that rats exposed to inescapable shock exhibit a

depletion in brain norepinephrine levels and rats exposed to escapable shock do not.

While the rational presented above can account for the original dog helplessness studies, it cannot account for the finding that helplessness has been shown to last up to a week in rats (e.g., Seligman, Rosellini, & Kozak, 1975). Therefore, Glazer and Weiss, (1976a; 1976b) have extended the position taken by Weiss, et. al., (1976) to account for both the short-term helplessness effect and the long-term helplessness effect. Glazer and Weiss prefer to use the term "interference effect" rather than learned helplessness effect.

In a series of seven experiments (i.e., Glazer & Weiss, 1976a, 1976b) Glazer and Weiss set out to clearly differentiate the conditions which give rise to both the short-term and long-term "interference effect". In the first four experiments, Glazer and Weiss (1976a) found that the interference effect occurs in at least two different ways. First, they found that if animals receive prior exposure to a single session of inescapable shock that is of a high intensity (4 MA) and brief duration (2 sec.) the interference effect (i.e., escape/avoidance deficit) dissipates over a short period of time (72 hrs.); however, if rats are given prior exposure to moderate intensity (1 MA), long duration (5 sec. or more) electric shock, the escape/avoidance deficit will last up to a week. Therefore, the results found in the high shock conditions are consistent with a time-dependent physiological change position such as the motor activation deficit theory and not consistent with a learning position. The results of the low shock condition (i.e., long-term interference), however, are not consistent with the motor activation

deficit theory; therefore, Glazer and Weiss (1976a) assumed that the rats had learned something. The question then became: What did the rats learn? Helplessness? Inactivity?

Glazer and Weiss, (1976a) strongly prefer a "learned inactivity" position which is similar to the Bracewell and Black's (1974) position presented earlier in this paper. Two important factors provoked Glazer and Weiss to assume a "learned inactivity" position. First, in Experiment 2 (Glazer & Weiss, 1976a), they found that animals receiving 2, 3, and 4 sec. duration inescapable shocks did not demonstrate an interference effect, while animals receiving inescapable shocks of 5 sec. duration or longer did show a subsequent interference effect. This finding suggested that shock duration was an important variable in demonstrating an interference effect. Since all the groups received inescapable shock, the learned helplessness theory has difficulty handling these data; moreover the learned helplessness theory makes no predictions about shock duration. Second, Glazer and Weiss made some behavioral observations which reinforced their ideas. These investigators (Experiment 2) observed that an animal's initial response to shock onset was to increase movement rather drastically and as shock continued the activity or movement decreased. Thus, the offset of prolonged shock reinforced the inactivity present at that time, thereby producing a learned response (i.e., inactivity) which competes with an active escape/avoidance task.

In the second series of experiments, Glazer and Weiss (1976b) set out to test the learned helplessness position and compare it to a learned inactivity position. Glazer and Weiss demonstrated that when

animals were exposed to inescapable shocks of a 5 sec. or longer duration they could perform better than no shock controls an escape/avoidance response (i.e., nose-poking escape response) which did not require the animal to make an active motor response. This, on the other hand, was not the case when Glazer and Weiss tested animals in an active/avoidance task such as FR-2 shuttle and FR-3 bar press. One strong criticism Maier and Seligman (1976) had of Weiss and his associates was that they had not reproduced their data using the triadic design which is crucial to learned helplessness theory. Therefore, in the final experiment, Glazer and Weiss (1976b) demonstrated that animals exposed to inescapable shock, when compared to an escape group and a no-shock group, performed the nose-poking escape response (i.e., a relatively inactive escape response) significantly better than either the no-shock or the escape group. However, when the escape/avoidance task was a FR-3 bar press, the typical helplessness effect was found. Thus, Glazer and Weiss concluded that when animals were given moderate intensity, long durations inescapable shock the subsequent escape/avoidance deficit is a result of learned inactivity and not learned helplessness.

In summary, Weiss and his associates propose that there are at least two types of interference effects produced by prior exposure to inescapable electric shock. That is, a short-term effect found when high intensity, short duration inescapable shock is administered, which is most probably attributable to central norepinephrine depletion; and a long-term effect found when moderate to low intensity, long duration inescapable shock is administered, which is proposed to be the result of

animals learning a motor response which is incompatible with an active escape/avoidance response (i.e., learned inactivity).

To this point, learned helplessness theory and the key alternative theoretical approaches have been briefly reviewed. Learned helplessness differs from these alternative competing response theories in, at least, two important ways. First, learned helplessness theory accounts for the escape/avoidance deficit, found following exposure to inescapable stress, by postulating a cognitive mechanism; whereas, competing motor response positions account for the same behavioral phenomenom in the more traditional S-R framework. Second, learned helplessness theory attempts to incorporate parallel experimental results from human and infrahuman subjects into one theoretical framework (e.g., Seligman, 1975), while the competing response theories are limited to those date from infrahuman studies (c.f., Levis, 1976). The generality of learned helplessness in humans (e.g., Hiroto & Seligman, 1975) involves not only the helplessness effect but includes a model for naturally occurring depression in man. It is to this helplessness model of depression that the discussion will now turn.

LEARNED HELPLESSNESS AND DEPRESSION

Through the years, a voluminous literature on depression has accumulated and, for the most part, this literature is confusing and quite often contradictory. According to Seligman (1975) much of the confusion surrounding the research on depression is a result of a "proliferation" of depressive categories. For example, Seligman cites a list of subtypes of depression that were originally presented by Mendels (1968), which included but was not limited to such categories as: psychotic, neurotic, endogenous, reactive, agitated, involutional, and psychotic reactive. In addition, Seligman argues that definitions of clinical depression are usually very broad and open-ended, thus decreasing the usefulness of such definitions in research. In other words there are many symptoms which are sufficient to define or diagnose depression; however, not one of them is uniquely necessary for that diagnosis. Seligman illustrated the problem this way:

> Depressives often feel sad, but sadness need not be present to diagnose depression; if a patient doesn't feel sad, but is verbally and motorically retarded, cries a lot, has lost twenty pounds in the last month, and the onset of symptoms can be traced to his wife's death, then depression is the appropriate diagnosis. Motor retardation also is not necessary, for a depressive can be quite agitated (1975, p. 80).

This does not in any way deny the existence of different types of depression, for some typologies have proven useful (e.g., the reactive-endogenous dichotomy), but only points out this as one source of problems associated with research on depression.

Seligman (1974; 1975) and others (e.g., McKinny & Bunny, 1969) have suggested that one way to overcome problems like those stated above is to develop laboratory models (i.e., basically animal models) for clinical phenomena such as depression. Seligman (1975) argued that since laboratory models have necessary behaviors which define the presence or absence of a particular phenomenon, they may reduce the open-endedness of the clinical definitions and thereby increase the testability of specific claims made by both the model and the clinical entity. Additionally, if a laboratory model were to be proven valid, then many disorders labeled depression could possibly be reclassified as separate disorders and thus reduce some of the confusion surrounding depression or other clinical phenomena.

Recently, Seligman (1975) proposed just such a laboratory model for human depression. Seligman postulated that learned helplessness, a phenomenon found in both humans and infrahumans, provides a reasonably good laboratory model for naturally occurring reactive depression in humans. While reactive depressions are the "primary focus" of the model, learned helplessness is more specifically a model for those depressions" in which the individual is slow to initiate responses, believes himself to be powerless and hopeless, and sees his future as bleak - which began as a reaction to having lost his control over gratification and relief from suffering" (p81). In fact, Seligman

argues that a belief in helplessness may be the unitary factor underlying all depressions.

At this point, our attention will turn to a review of the learned helplessness model. This review will follow four lines of parallel evidence: (1) behavioral and physiological symptoms; (2) cause; (3) prevention; and (4) cure. Since this has already been extensively reviewed by Seligman (1975), only a brief review, with elaborations on points of importance for this dissertation, will be presented. The interested reader is directed to Seligman (1974; 1975) for more details.

First, Seligman (1975) presented evidence that there are six symptoms of helplessness which have parallels in human depression: (1) passivity or a reduction in the initiation of voluntary responses is a common feature of both helplessness and depression; (2) both depressives and helpless subjects exhibit a negative cognitive set (i.e., demonstrates a reduction in their ability to learn response-outcome contingencies); (3) helplessness and depression have a time course; (4) both helpless subjects and depressed persons exhibit a diminished ability to initiate aggressive responses; (5) helpless animals and depressed humans experience anorexia, weight loss, and a reduction in social and sexual responding; and (6) helpless rats show a depletion of whole brain norepinephrine (NE), and drugs that increase or maintain NE levels of the appropriate sites are successful in alleviating depression.

Since the dissertation presented here was designed to examine the effects of electroconvulsive shock (ECS) on learned helplessness, and it has been demonstrated that NE levels in the nervous system are

affected by the administration of ECS (e.g., Schildkraut & Kety, 1967), it is necessary to elaborate somewhat on the NE parallels of depression and learned helplessness.

One of the more accepted physiological theories of depression is the catecholamine hypothesis (Schildkraut, 1965). The basic postulate of the hypothesis is that NE is either depleted or lowered in the central nervous system of depressives; however, much of the evidence which supports this position is indirect. That is, the evidence comes basically from studies which examine the effects of drugs which alter NE levels at the appropriate sites in the central nervous system. Schildkraut and Kety (1967) reviewed the pharmacological observations compatible with the catecholamine hypothesis and report that: (1) reserpine, a drug that depletes NE, produces a sedation effect in both humans and animals and occasionally produces depression in humans; (2) monoamine oxidase (MAO) inhibitors which increase levels of NE in the brain produce an excitation effect in animals, prevent and reverse reserpine-induced depression, and are useful as antidepressants in humans; and (3) tricyclics such as imipramine, which inhibit cellular uptake of NE in the brain, prevent reserpine-induced depression in animals and alleviate depression in humans. In addition to the above, Seligman (1975) reported that physostigmine, a drug which facilitates cholinergic activity, produces almost immediate depression in humans and when these same subjects are injected with atropine, an anticholinergic drug, the depression dissipates almost as immediately as it appeared.

Studies compatible with learned helplessness theory have also

demonstrated the importance of the effects of NE depletion. For example, the reader should recall the previous discussion of the research done by Weiss and his associates. These investigators (e.g., Weiss et. al., 1975) have consistently demonstrated that rats exposed to uncontrollable trauma show a significant depletion of whole brain NE; whereas rats exposed to controllable trauma show an increase in whole brain NE. While Weiss et. al., do not accept the helplessness position (primarily because it is a cognitive theory of animal behavior), they acknowledge that the data on NE could be concomitant with the helplessness effect. Other data cited by Seligman (1975) further substantiate the role of NE in both helplessness and depression: (1) alpha-methylparatyrosine (AMPT), a specific NE depletor (i.e., this drug inhibits the synthesis of NE), produces escape deficits in rats that are like those deficits found after exposure to inescapable shock; and (2) AMPT when injected in monkeys produces behavior which parallels depression in humans. A discussion of the effects of ECS on NE will be presented later, but for now the discussion will turn to etiological factors.

Seligman (1975) argued that the etiologies of learned helplessness and depression are similar. Learned helplessness is caused by learning that responding is independent of reinforcement; whereas depression is caused by "the belief that responding is useless". To support this, Seligman presents evidence from several sources (e.g., Beck, 1967) which indicate that basic to most depressions is a belief in helplessness. Furthermore, Seligman sees this approach as consistent with the behavioral view (e.g., Ferster, 1973) that depression

is a result of loss of reinforcers or extinction, since extinction (i.e., a zero probability of reinforcement) may be seen as a special case of learned helplessness, in which reinforcement may still occur but would be independent of responding.

A third line of evidence presented by Seligman (1975) indicates that learned helplessness can be prevented by "immunizing" subjects with prior exposure to mastery of response-reinforcement contingencies. For example, rats given early experience with escapable shock do not become helpless when exposed to inescapable shock later in their life (Maier and Seligman, 1976). While data on prevention of depression are for the most part not available, Seligman has speculated that early exposure to mastery could prevent depression or immunize a person against this disorder; whereas early exposure to uncontrollable trauma could predispose a person to depression.

Finally, Seligman (1975) presented evidence which indicates that alleviation of helplessness has parallels in depression. First, directive therapies cure both helplessness and depression. For example, helplessness is alleviated in dogs and rats by forcing exposure to the response-reinforcement contingency and depression is cured by demonstrating to the depressive or by helping him to see that his responses can produce the desired outcome. Some of the directive therapies which alleviate depression, reviewed by Seligman, which are consistent with the learned helplessness model were: assertive-training; cognitive therapy; rational-emotive therapy; and the graded-task treatment. Second, both helplessness and depression, when untreated, have been shown to dissipate in time. Third, drugs

which facilitate NE in the brain alleviate depression in humans; however, data on the effects of these drugs on helplessness are sorely needed. Finally, ECS has been shown to alleviate depression (i.e., primarily endogenous depression) and to attenuate helplessness in dogs. Since ECS is the primary focus of the dissertation, before continuing the discussion of the learned helplessness model, per se, a brief review of the literature on ECS is in order.

Since Cerletti and Bini (1938) first produced therapeutic "fits" in humans by passing an electrical current through two electrodes placed on the forehead, the use of electroconvulsive shock as a therapeutic device has proven to be quite successful in alleviating several types of psychopathology, especially depression (Sargent and Slater, 1972).

Clinically, ECS has been used for over thirty-five years. The normal practice (c.f. Sargent & Slater, 1972) is to pass a controlled pulse of electricity through the brain, causing an initial tonic contraction, followed almost immediately by a generalized clonic convulsion which imitates in many respects a grand mal seizure. Currently the practice is to anesthetize patients with a rapid-acting sedative and control the muscular effects with muscle relaxants. It has been demonstrated (e.g., Winokur, 1972) that the clonic convulsion is the key to successful outcome with ECS and that subconvulsive seizures are ineffective. Memory loss, contrary to the lay person's view, has not been shown to be related to outcome (Klerman, 1972). The post-treatment effects of ECS are usually confusion, muscular pains, headache, and memory loss. Memory loss, being the major

adverse effect of ECT, is rarely severe and usually dissipates in a week or two.

Based on available data (e.g., Kolb, 1968; Nystrom, 1964; Sargent & Slater, 1972) ECS is effective in approximately 80 per cent of all depressions. In addition, Nystrom (1964) and others (e.g., Winokur, 1972) have provided some useful information on clinical indications for ECS. That is, favorable outcome of ECS is associated with depressives who exhibit the following symptoms: acute onset; psychomotor retardation; early morning awakening; previous good functioning; and profound or severe depressive mood. Unfavorable outcomes are associated with: ideas of references; paranoid symptoms; inability to define specific onset; and a history of psychiatric problems. Of course, these do not exhaust the clinical indicators for the use of ECS in depression; however, they have some striking parallels with learned helplessness, such as psychomotor retardation and acute onset.

The reason ECS is so effective with depression is not really known; however, the catecholamine hypothesis may hold some promise for answers to questions related to the effectiveness of this treatment. The reader will recall the previous discussion of the role of norepinephrine (NE) in depression and learned helplessness which indicated that drugs which facilitate the presence of NE in the brain also alleviate depression. ECS also has been shown (Hendley & Welch, 1974) to have a facilitating effect on brain NE. For example, Hendley and Welch (1974) demonstrated that multiple ECS reverses reserpine-induced depression in mice. In addition, Schildkraut

and Kety (1967) reported research which indicated that following unmodified ECS (i.e., lack of sedative and muscle relaxants) plasma and urinary levels of NE and epinephrine increased. Based on this and our previous discussion of the influence of NE in depression, it appears that NE may play either a primary or secondary role in the etiology of both clinical depression and learned helplessness.

In addition to the clinical use of ECS, it has also been employed as an experimental tool to study memory and behavior in animal laboratories. To date, several major hypotheses have been advanced to account for the retrograde effect of ECS on learned behavior. The first and probably still most widely accepted hypothesis is that ECS disrupts memory consolidation processes (Duncan, 1949). According to this view the memory trace is held in the reverberatory circuit until a permanent neural structural change is effected (Hebb, 1949). If ECS is administered shortly after learning the memory trace would be disrupted. Since Duncan's (1949) work, many studies (Lewis, 1969) have demonstrated that disruption of learning is a decreasing function of the temporal interval between training and ECS (i.e., as the training-ECS interval increases, disruption of learning decreases).

Coons and Miller (1960) objected to Duncan's position and proposed that ECS is a highly aversive stimulus which disrupts previous learning through punishment, rather than disrupting memory consolidation. Thus the performance decrement which decreases as a function of the learning-ECS interval is best described as a delay-of-punishment gradient rather than a fixation of memory. A similar hypothesis has been offered by Adams and Lewis (1962a; 1962b), which suggests that

a competing response (i.e., a partial convulsive response) is classically conditioned to situational cues. In this situation ECS serves as an unconditioned stimulus and situational cues act as conditioned stimuli. Thus, the performance decrement observed following ECS is produced by a competing response interfering with the performance of the learned response. In addition to these interpretations, Chorover and Schiller (1966) suggested that ECS administered after long post-learning intervals merely alleviated a conditioned emotional response (freezing); and Pinel and Cooper (1966a; 1966b) proposed that the retrograde effects of ECS were due to an interference with the incubation of fear (i.e., increase in strength over time) rather than memory. More recently, Miller and Springer (1973) have suggested that ECS produced a failure to retrieve information from long-term memory, rather than a disruption of the consolidation processes.

Whether or not ECS disrupts consolidation, acts as a punishment, blocks retrieval or influences behavior in ways suggested by Pinel and Cooper or Chorover and Schiller is still not clear, and further experimentation is needed. However, for the purposes of this dissertation, the interest is more in the attenuating effects of ECS on learned behavior patterns. Some of the studies which have examined the attenuating effects of ECS on learned behavior have demonstrated that ECS: attenuated experimental neurosis in cats (Masserman & Jacques, 1974); eliminated a well established conditioned emotion response (suppression of bar pressing) in rats (e.g., Hunt & Brady, 1951); reversed a well extinguished avoidance response in rats (Gellhorn, Kessler, & Minatoya, 1942); reversed reserpine-induced depression

in mice (Hendley & Welch, 1974); and attenuated learned helplessness in three of six dogs (Dorworth, 1971).

Thus far, the learned helplessness model of depression has been briefly reviewed, with elaboration in areas of particular importance to this dissertation (i.e., norepinephrine and electroconvulsive shock). As Seligman (1975) has argued, no laboratory model of a clinical phenomenon can ever hope to account for all aspects of that phenomenon. However, if the model can be proven valid, it can reshape or help sharpen some of the clinical definitions, as well as, provide valuable information on, for example, prevention and cure of a clinical disorder such as depression. One method to strengthen a laboratory model, according to Seligman, is to test parallel claims. Thus, Seligman argues that if two phenomena, such as learned helplessness and depression, converge on one or two criteria such as symptoms and etiology, then the model can be tested by looking for parallels along a third criteria. As an example Seligman stated it this way:

> .... if learned helplessness in dogs presents behavior similar to reactive depression in man and the etiology of the two is similar, as we shall argue, and it turns out that the only way to cure learned helplessness is to expose dogs forcibly to responding that produces relief, one has a prediction about cure of depression in man: the recognition that responding is effective in producing reinforcement should be the central issue in successful therapy (1974, p. 84).

If this is examined and proven valid, then one has strengthened the helplessness model; however, if disconfirmed the model is weakened. In addition, the model should work in the opposite direction as well.

For example, if ECS will alleviate or relieve depression in humans it should also attenuate learned helplessness in animals. This statement represents one of the reasons for the present research.

The rational for the present research was basically twofold; first, it was an attempt to provide an experimental procedure which would reliably produce the helplessness effect in rats, which until recently has been difficult (e.g., Seligman & Beagley, 1975). Second, it was designed to demonstrate whether or not ECS will attenuate the learned helplessness effect in rats. If ECS-induced attenuation could be demonstrated, two purposes would be served: (1) it would expand the generality of helplessness theory to rats by increasing the parallels between helpless dogs and helpless rats, especially since ECS has been shown to partially attenuate helplessness in dogs (Dorworth, 1971); and (2) it would increase the validity of the learned helplessness model of human depression.

Prior to turning our attention to the experimental studies presented in this dissertation, a definitional issue concerning the term "learned helplessness" needs clarification. For example, at times there appears to be some confusion as to whether Seligman and his associates have used the term learned helplessness to define a <u>process</u> (e.g., learning that outcomes are independent of responses) or whether they have employed the term to define a <u>product</u> of learning that outcomes are independent of responses (e.g., a failure to learn to escape in a new situation). In addition, but not mutually exclusive of the above, at times it is not exactly clear whether the term learned helplessness is used as an explanatory concept or a descriptive concept.

Thus, in order to clarify this issue in the context of the research presented here, the term learned helplessness is defined as follows: rats given prior exposure to yoked-inescapable shock, when compared to rats given prior exposure to escapable shock and rats given no exposure to shock (i.e., no-shock controls), show an escape/avoidance deficit when tested on a new or different task.

EXPERIMENT 1

The rational for the experiment presented here was twofold. First, while the learned helplessness effect and the experimental procedures for demonstrating this effect have been found to be quite reliable in humans and some animals (e.g., dogs), there is still considerable controversy surrounding the demonstration of learned helplessness in rats. For example, in rats exposure to inescapable shock produces an escape deficit on some tasks such as an FR-2 shuttle escape task and an FR-3 bar-press escape task, but not on either an FR-1 shuttle escape task or an FR-1 or FR-2 bar-press escape task. One explanation offered by Maier and Seligman (1976) for these discrepant results is that the FR-1 shuttle and FR-1 and FR-2 bar-press tasks are tasks that the rat learns easily and because of this the associative interference produced by inescapable shock is easily overcome. In addition, Maier and Seligman argue that the FR-1 shuttle response for the rat is a "high probability initial response to shock" and for the rat could possibly be a species-specific defense response (p23). If these arguments are correct then one wonders why this is not also true of the dog and the cat, especially since the learned helplessness effect can be demonstrated in both the dog and cat using an FR-1 shuttle escape task? Surely crossing a shuttlebox once for the dog and cat is an effortless response that should be easily learned.

In addition, when learned helplessness is obtained in rats it is usually just a failure to perform as well as the escape and no-shock control groups rather than a failure to learn to escape shock; whereas

in the dog it is clearly a failure to learn to escape shock. This also presents considerable difficulty to the learned helplessness position, especially since learned helplessness theory postulates a failure to learn. Thus, this experiment was designed to determine if the experimental procedure employed here - a progressive fixed-ratio bar press escape task was utilized for initial training and a FR-2 shuttlebox escape/avoidance task was used to test for helplessness - could produce a learned helplessness effect in rats (c.f., Seligman & Beagley, 1975) which is comparable to that found in dogs.

If learned helplessness could be demonstrated with the experimental procedures employed here, then the second reason for the experiment was to determine whether or not ECS could effectively attenuate this phenomenon. If, in fact, ECS does attenuate learned helplessness, then we have identified another technique for alleviating or reversing helplessness in rats, thereby increasing the parallels between rat helplessness and dog helplessness, and strengthening the learned helplessness model of human depression. Since ECS is not typically given to humans until after they exhibit depression, then ECS in this study was administered following the test for helplessness. Specifically, rats were given either escapable-shock training, inescapable-shock training, or no-shock training in a Skinner Box on Day 1. On Day 2, all subjects were tested for helplessness in a shuttlebox escape/avoidance task, and half of each group was given ECS immediately thereafter. All subjects were then retested for helplessness in the shuttlebox on Day 3.

Method

Subjects. The subjects were 48 experimentally naive male hooded rats obtained from the departmental colony. Subjects were all 90 - 110 days old at the time of the experiment, were housed individually under continuous 24-hour illumination, and were maintained on ad libitum food and water.

Apparatus. Two fully automated 30x23x26 cm. Skinner boxes with retractable levers, in sound attenuating chambers, were used for the training phase. The long sides of the Skinner boxes were clear Plexiglas, and the ends of each box were stainless steel. The floor of each box was constructed of .3-cm. diameter stainless steel grids which were 1.0-cm. apart. Each Skinner box was illuminated by 2 houselights which remained on during all experimental sessions. A 75-db. white noise and blower motor (which ventilated the chamber) provided the masking noise. Each box was connected to a shock device which provided a 1.0 mA scrambled shock through the grid floor.

A partially automated two-way shuttlebox located in a sound attenuating chamber was used for the test and retest phase. The shuttlebox was constructed by placing two 22x19x15 cm. Skinner boxes back to back. The two long arms and top door of each Skinner box were clear Plexiglas, and the ends were stainless steel. A manually operated plywood door covered a 8 x 13 cm. opening between the two compartments. The floor of each compartment was constructed of .2-cm. diameter stainless steel grids located 1.0-cm. apart. The chamber was illuminated by two 7.5-w. white light bulbs and had a one-way window through which

each subject and the entire shuttlebox could be observed. A 80-db. white noise provided the masking noise. The grid floors in both compartments were connected to a shock device which delivered a 1 mA electric shock.

A standard shock device was used as the ECS generator. The generator delivered 40-ma. at 1,200-v. for .5-sec. through alligator ear clips covered with gauze which had been soaked in 0.9-NaCl solution. This produced a full tonic-extensor seizure in all subjects.

Procedure. Each subject was randomly assigned to 1 of 3 groups (n=16): escape, yoked-inescapable, and no-shock control. Training of the escape group was conducted in the Skinner box with the lever out and operative. Each subject in the escape group received 80 trials of escapable 1.0-ma. scrambled shock delivered through the grid floor. Shocks were administered on a VI-60 sec. schedule, were not predicted by any signal, and were terminated only after the subject responded appropriately. Initially, the subjects were required to make one bar press to escape shock. After one bar press on 5 consecutive trials with a latency of less than 5.0-sec., the contingency was increased to FR-2. Following 5 consecutive FR-2 escapes with a latency less than 5.0-sec., the contingency was increased to FR-3 to escape shock. The remainder of the trials were conducted on the FR-3 schedule. This procedure was a modified version of that used by Seligman and Beagley (1975).

The yoked-inescapable group received exactly the same intensity, frequency, and duration of shocks its escape partner received; but no

response would escape shock. The bar was present in the training chamber; bar presses were counted but had no other consequences. The no-shock control subjects received only a 105-min. pre-exposure to the training chambers. The pre-exposure time was the approximate mean time the other two groups took to complete 80 trials.

Shuttlebox escape/avoidance testing was conducted 24-hours following training for the escape and yoked-inescapable groups and 24-hours following pre-exposure for the no-shock control. Each rat was placed in the shuttlebox facing away from the door, and after 1-min. exploration period the door which separated the two compartments was manually lifted. Lifting the door initiated the onset of the CS (a 6,000-cps. 80-db. tone). Failure to respond within 10-sec. following CS presentation resulted in the onset of the UCS (1.0-ma. of scrambled shock, delivered through the grid floor) after which a response was an escape rather than an avoidance response. In order to terminate (lowering the door terminated the trial) the CS or CS-UCS combination the rat had to make a complete FR-2 shuttle response (i.e., the rat had to enter the opposite compartment completely and return to the original compartment; shock was on in both compartments simultaneously). Failure to make the appropriate response within 60-sec. resulted in the door being lowered and termination of the trial. Each rat received 15 escape/avoidance trials programmed with a 30-sec. intertrial interval.

Immediately following the test phase one-half of the subjects in each of the 3 groups (n=8) were administered one ECS while the other half received no treatment. Rats that received ECS were taken

out of the test chamber following completion of 15 trials and placed on a table top where the clips were attached and immediately given ECS. All subjects were then retested 24-hours following the test phase. Retest was conducted exactly the same as the test phase described above.

Results

Training. All rats in the escape group learned to bar press to escape shock as shown by decreasing latencies over trials. As the bar press escape contingency changed, escape latencies initially increased then decreased over trials. The mean duration of shock per trial was 6.38 sec. for the escape groups and yoked-inescapable groups. In addition, the mean number of bar presses during this session for the escape groups was 302.38; the yoked-inescapable group showed a mean bar press frequency of 46.13.

Test. The mean escape/avoidance latencies for each of the 15 trials were computed for each of the six subgroups, and those data were subjected to an analysis of variance. These means are presented in the left half of Figure 1 for blocks of three trials. Since the treatment variables were not introduced until after test, the left half of Figure 1 combines the ECS and NO-ECS (NECS) subgroups.

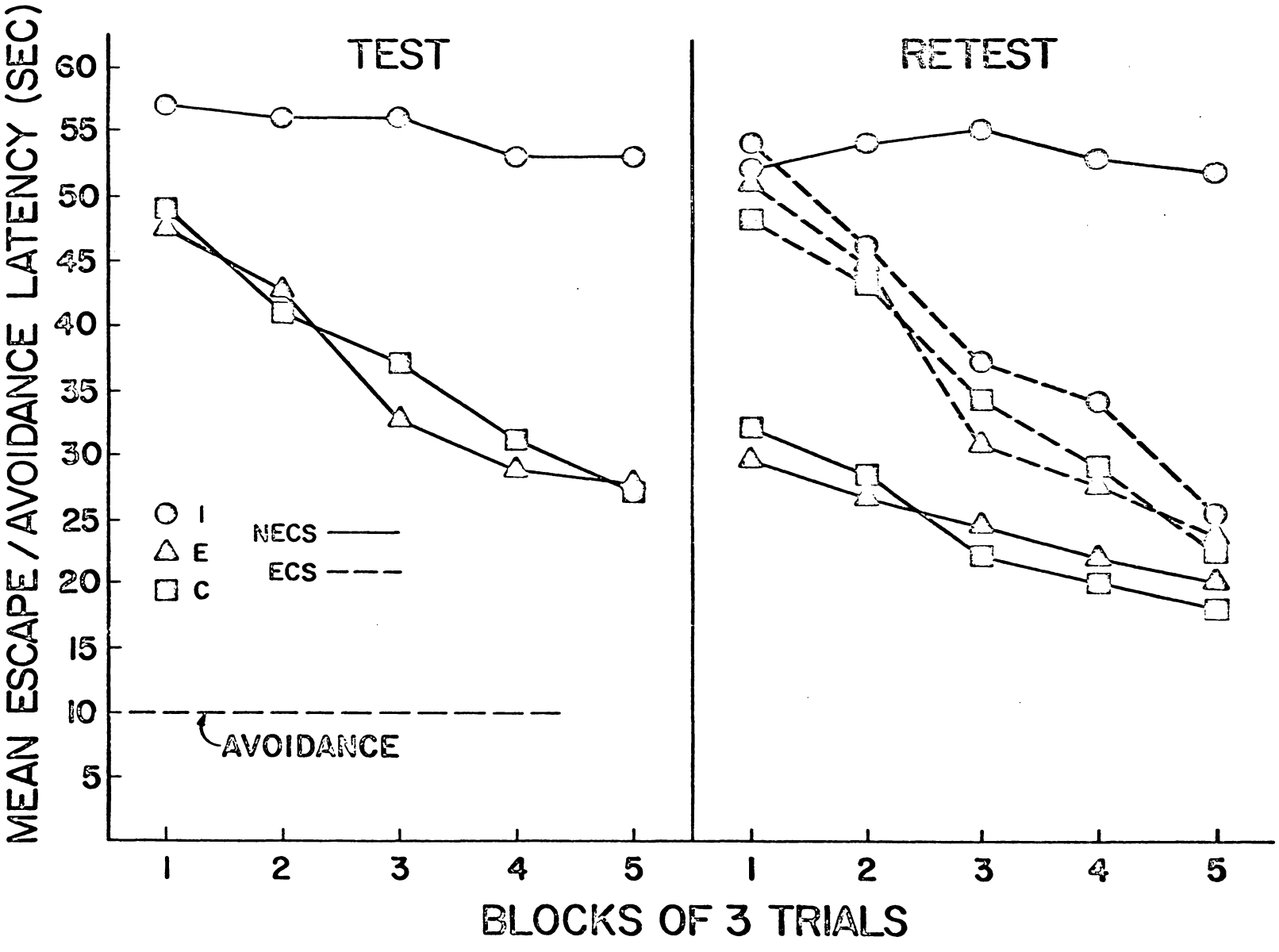
It is evident from the left half of Figure 1 that both the escape (E) and no shock control (C) groups show declining latencies over trials; however, the inescapable (I) group shows essentially no change in latencies over trials. These findings clearly demonstrate the learned helplessness effect and were confirmed by the overall analysis

of variance which yielded a significant Groups X Trials interaction (F=2.87, df=28/588, $p<.001$).

Retest. The mean escape/avoidance latencies for each of the 15 trials were computed for each of the six subgroups, and those data were subjected to an analysis of variance. These means are presented in the right half of Figure 1 for blocks of three trials. Figure 1 shows that retest performance was affected by both the ECS by NO-ECS manipulations and by prior exposure to inescapable shock. Specifically, in the NO-ECS groups performance during retest seems to represent simple carryover of performance from the test phase, with the control conditions showing increasingly more rapid escape behavior, and the inescapable group showing essentially no change in latencies over trials. On the other hand, the ECS groups, regardless of performance at the end of the test phase, show highly similar performance during retest. All ECS groups show relatively slow latencies at the start of retest and considerable improvement across the retest session. Within-subjects t-tests comparing the last block of 3 trials in the test phase to the first block of 3 trials in the retest phase indicated that the escape group (t=2.88, df=7, $p<.05$) and the no-shock control group (t=4.84, df=7, $p<.01$) in the ECS condition showed a significant increase in escape latencies from test to retest; the remaining groups showed no reliable differences.

Analysis of variance of the retest data confirmed the conclusions suggested by Figure 1. Specifically, the overall analysis yielded a significant Groups X Treatments interaction (F=8.93, df=2/42, $p<.001$) and a significant Treatments X Trials interaction (F=9.35, df=14/588,

Figure 1. Mean escape/avoidance latencies plotted across blocks of three trials for both the test and retest sessions. The yoked-inescapable group (I) is represented by solid circles, the escape group (E) by solid triangles, and the no-shock group (C) by solid squares. The NO-ECS (NECS) condition is represented by solid lines, and the ECS condition by broken lines.


TEST
RETEST
MEAN ESCAPE / AVOIDANCE LATENCY (SEC)
60
55
50
45
40
35
30
25
20
15
10
5
I
E
C
NECS
ECS
AVOIDANCE
1
2
3
4
5
BLOCKS OF 3 TRIALS

$p < .001$). Inspection of the right half of Figure 1 suggests that the Groups X Treatments interaction effect stems from the apparent absence of differences in performance among the ECS groups together with large differences in performance among the NO-ECS groups. This impression is confirmed by simple effects analyses which yielded a significant Groups effect for the NO-ECS condition ($F=23.35$, $df=2/42$, $p < .001$) but not for the ECS condition. The Treatments X Trials interaction effect is obvious from the right half of Figure 1. That is, the ECS groups showed considerable improvement across the retest session; while little or no improvement was shown by the NO-ECS groups.

The behavior of the rats in the yoked-inescapable group which failed to perform the escape response during the test and retest phases is worthy of description. During the test phase these rats squeaked and hopped about the apparatus in a crouched position rather frantically on all 15 trials; whereas during the retest session there was a marked diminution in this behavior. During the retest phase the yoked-inescapable rats in the NECS condition which continued to show a failure to perform the escape response exhibited less squeaking and a more passive (i.e., less hopping) crouching response in the presence of shock. Backrolling - a competing response observed by Seligman and Beagley (1975) - was not observed at any time in this study. On the other hand, the behavior of the rats in the escape and no-shock control groups during test was characterized by frantic hopping on the first few trials which rapidly diminished and was replaced by obvious goal-directed voluntary responding. During retest these rats performed the escape response quite rapidly with little or no reflexive behavior.

Discussion

The experimental manipulations employed in this study were shown to produce the learned helplessness effect in rats. Rats which received either bar-press escape or no shock (i.e., only exposure to the training apparatus) and then were tested for escape/avoidance learning on a FR-2 shuttlebox task escaped readily. However, rats which were given yoked-inescapable shock training demonstrated a substantial failure to escape. These rats endured the entire 60 sec. of shock, and even after a successful escape response was made they reverted back to taking the full 60 sec. of shock on subsequent trials. In addition, the behavior of the rats in the yoked-inescapable group in the shuttlebox during the test phase did not resemble that of an animal which had originally learned to be inactive as Glazer and Weiss (1976b) has suggested, nor did it resemble the behavior of helpless dogs (i.e., passivity) as described by Maier and Seligman (1976). On the other hand, the behavior of these rats during retest (i.e., the last 15 trials) was highly similar to the behavior of preshocked rats described by Seligman and Beagley (1975, Exp. 2). These behavioral observations suggests that activity alone is not critical for the demonstration of learned helplessness. It should also be noted with importance that the learned helplessness effect found in this experiment strongly resembles that which is found in dogs (i.e., a failure to learn to escape shock).

The administration of a single ECS immediately following the test session attenuated the learned helplessness effect in the retest session. Helpless rats that were given ECS showed "normal" learning

of the escape response the following day; whereas, helpless rats were not given ECS continued to show a failure to learn the escape response. In addition, ECS temporarily attenuated previously established escape behavior in both an escapable-shock group and a no-shock control group. These latter rats looked as if they had not previously learned the escape response. Therefore, it appears that ECS has a comparable effect on retest-escape behavior regardless of whether rats were previously helpless or had learned to escape. Unfortunately, exactly what this effect is cannot be inferred from this experiment.

The demonstration that ECS attenuates the learned helplessness effect not only provides parallels to dog helplessness but increases the parallels between learned helplessness and depression, thereby giving the learned helplessness model of depression added strength. However, since ECS was given immediately after the test for helplessness - a necessary condition to test the learned helplessness model of depression - it is not clear whether the effects of ECS are on learned helplessness training or the learned helplessness effect (i.e., test phase training) or both. In addition, it is not clear whether ECS is attenuating learned helplessness directly (i.e., retrograde effects) or indirectly through some type of prograde effects such as producing a response that competes with the previously learned response (e.g., Adams & Lewis, 1962).

Experiment 2 was designed to determine if ECS given either immediately following training or 23.5 hours following training but before the test phase would produce effects on behavior comparable to those found

in Experiment 1. Experiment 2 was also designed to provide a replication of the learned helplessness effect in rats using the experimental manipulations which were demonstrated to be effective in this experiment. In Experiment 2 training was conducted exactly as that described in Experiment 1. Then, one-third of the rats in each group was given either NO-ECS, immediate ECS or delayed ECS. All rats were tested for helplessness 24 hours later.

EXPERIMENT 2

Method

Subjects. Seventy-two naive rats like those described in Experiment 1 were subjects.

Apparatus. The apparatuses used in this experiment were exactly the same as that described in Experiment 1.

Procedure. Each rat was randomly assigned to 1 of 3 groups: escape, yoked-inescapable, and no-shock control. Training procedures for the three groups were conducted exactly like those described in Experiment 1. Following training one-third of the rats in the escape and yoked-inescapable groups (n=8) were given one ECS immediately following the last training trial, one-third were given one ECS 30-min. prior to test, and one-third received no treatment. No-shock control rats were not preexposed in this experiment and testing occurred 24-hours following ECS for one-third of the rats, 30-min. following ECS for one-third, and without ECS for one-third. Shuttlebox escape/avoidance testing was conducted exactly the same as the test phase described in Experiment 1.

Results

Training. All rats in the escape group learned to bar press to escape shock as shown by decreasing latencies over trials. As the bar press escape contingency changed, escape latencies initially increased followed by a subsequent decrease. The mean duration of shock per trial was 4.39 sec. for the escape groups and yoked-in-

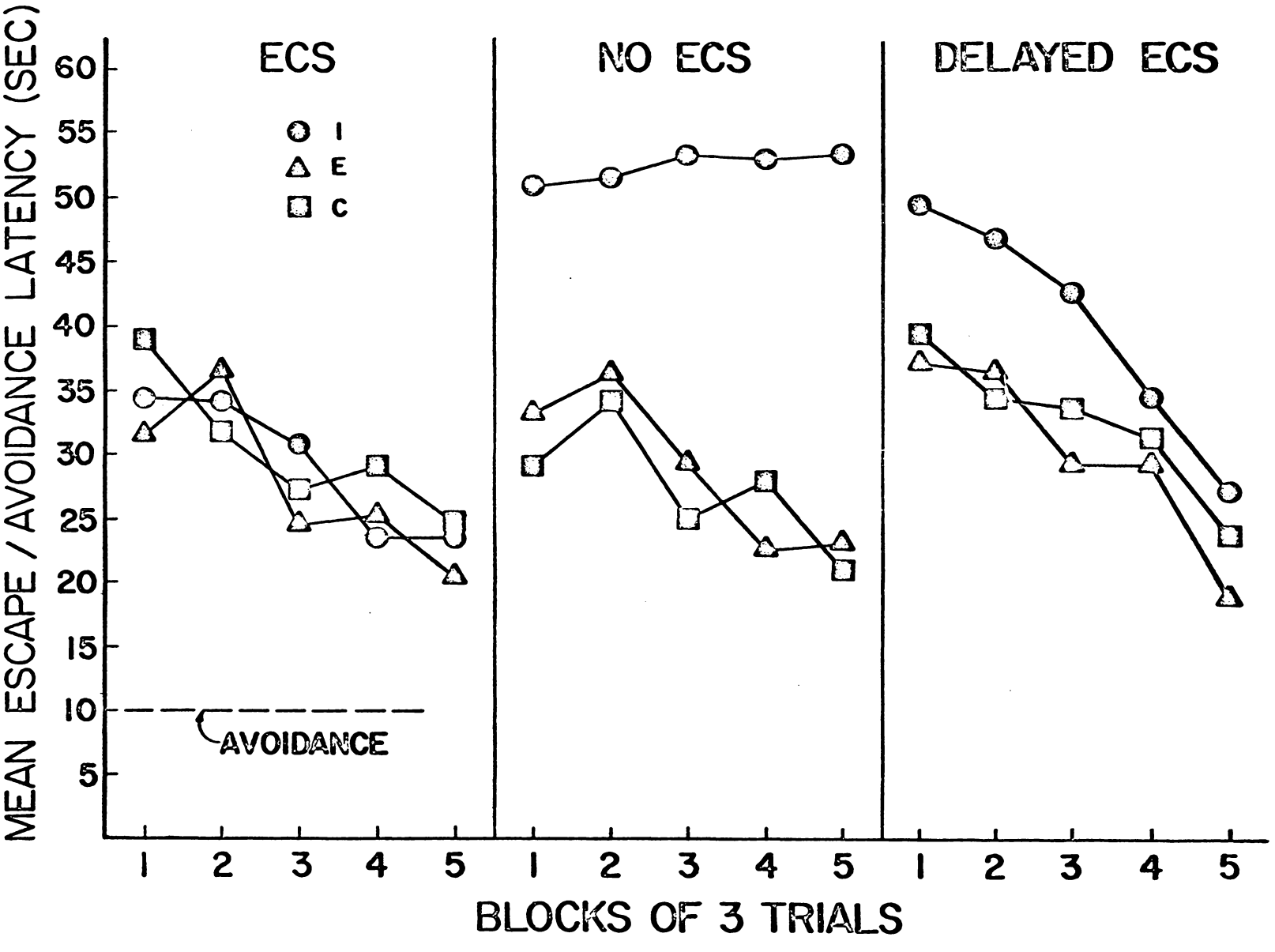
escapable groups. The mean number of bar presses for the escape group was 311.29, and for the yoked-inescapable group, 29.19.

Test. The mean escape/avoidance latencies for each of the 15 trials were computed for each of the nine experimental groups, and those data were subjected to an analysis of variance. The means are presented in Figure 2 for blocks of three trials for the immediate ECS condition (ECS), the NO-ECS condition (NECS), and the delayed ECS condition (DECS).

Figure 2 shows that in the NECS condition both the escape (E) and no-shock control (C) groups showed short and declining latencies over trials, whereas the inescapable (I) group showed essentially no change in latencies over trials. These findings essentially replicated those for the test phase in Experiment 1 (i.e., they show the learned helplessness effect). On the other hand, the immediate ECS condition produced equivalent rapid and improving performance in all 3 groups, again indicating an attenuation of the learned helplessness effect by ECS. It will be noted that these data appear very similar to those in Experiment 1 during the retest phase.

The effects of DECS were less clear cut. Compared to the immediate and NECS condition DECS produced an overall increase in latencies of approximately 3 sec. in the escapable and no shock groups. With the inescapable group DECS produced an intermediate level of performance. Initially the performance of DECS was as poor as that of the NECS inescapable (helpless) group, but DECS performance does improve and eventually appeared to be comparable to that of the immediate-ECS inescapable group. Thus, while DECS may attenuate the helplessness

Figure 2. Mean escape/avoidance latencies plotted across blocks of three trials for the immediate ECS (ECS) condition, the NO-ECS condition, and the delayed ECS condition. The yoked-inescapable group (I) is represented by solid circles, the escape group (E) by solid triangles, and the no-shock group (C) by solid squares.


ECS
NO ECS
DELAYED ECS
I
E
C
MEAN ESCAPE / AVOIDANCE LATENCY (SEC)
60
55
50
45
40
35
30
25
20
15
10
5
AVOIDANCE
1 2 3 4 5
1 2 3 4 5
1 2 3 4 5
BLOCKS OF 3 TRIALS

effect, the attenuation was not nearly as dramatic as that for immediate ECS.

The effects described above were confirmed by the overall analysis of variance which yielded a significant Groups X Treatments interaction effect ($F=5.21$, $df=4/63$, $p<.01$). It should be noted from Figure 2 that all groups showed improvement across test trials except for the NECS inescapable group. Normally this difference should be expected to generate a significant Groups X Treatments X Trials interaction. However, in the present analysis this interaction term was not significant ($F=0.97$). The significant Groups X Treatments interaction was further analyzed with simple effects analyses. These analyses yielded a significant Groups difference in the NECS condition ($F=20.69$, $df=2/63$, $p<.001$), but not in the immediate ECS condition ($F<1.0$) nor in the DECS condition ($F=2.87$, $df=2/63$, $p>.05$). Similarly, orthogonal analyses of simple effects across treatment conditions yielded a significant effect among treatments for the inescapable shock groups ($F=13.85$, $df=2/63$, $p<.01$) but not for either the escapable or no shock groups ($F<1.0$). Thus, the results of statistical analyses clearly confirmed the conclusions of Figure 2 for the ECS and NECS conditions, but indicate that the apparent effects in the DECS condition were not statistically reliable.

A close look at the DECS inescapable group revealed that 4 out of 8 or 50 per cent of the rats showed a failure to escape over the first seven trials, but over the remaining eight trials only 2 of those 4 rats continued to show this escape deficit. On the other hand, only 2 of the 8 rats in each of the three groups of the immediate ECS condition

showed an escape deficit.

The behavior of the rats in the yoked-inescapable group which failed to perform the escape response over the first few trials in this experiment was essentially similar to that observed in the comparable groups during the test session of Experiment 1. However, as the trials proceeded there was a marked diminution in the squeaking and hopping, and the rats demonstrated a more passive response to shock (i.e., like the behavior described during the retest phase of Experiment 1). The behavior of the rats in the escape and no-shock groups was also highly similar to the rats in the comparable groups of Experiment 1.

Discussion

As in Experiment 1, the results of this experiment confirmed that the experimental procedures (i.e., training and test tasks) employed are capable of producing a learned helplessness effect in rats that is comparable to that found in dogs. Rats trained to escape shock by bar pressing and rats given no prior exposure to shock, when tested on the following day in a shuttlebox escape/avoidance task, escaped rapidly. Rats given prior training with yoked-inescapable shock showed a substantial deficit in escape performance. In addition, the yoked-inescapable rats which failed to learn the escape response endured the full duration of shock on almost every trial, and when a successful escape was made, reverted back to taking the full duration of shock on subsequent trials. The behavior of the helpless rats, while initially active, became quite passive as the

test session progressed. This is essentially the same behavior as that of preshocked rats described by Seligman and Beagley (1975). It is particularly noteworthy that rats in this study remain helpless whether they are actively taking shock, as on the initial trials or passively taking shock, as in later trials.

Further, it was demonstrated that ECS administered immediately following training attenuates learned helplessness. Rats given immediate ECS, whether they received escape training, yoked-inescapable shock, or no shock (i.e., just ECS 24 hours prior to test), when tested on the following day escaped well. Since the no shock controls, which received only ECS prior to test, performed as well as the escape group in both the immediate and NO-ECS condition, it is clear that ECS in this study does not produce proactive interference at the 24 hour interval. This essentially rules out the argument that the effects of ECS are a result of proactive interference rather than retroactive, thereby indicating that ECS reverses helplessness training. This further implies that the attenuation of learned helplessness by ECS in Experiment 1 was a result of the retrograde effects of ECS on previous learning and not a result of ECS producing a response that competes with previous training.

On the other hand, the effects of delayed ECS (i.e., ECS given 23.5 hours following training) are not as clear. While it appears that delayed ECS attenuates learned helplessness, it does so less dramatically than immediate ECS. In addition, rats in the escape group and no shock group given delayed ECS escaped shock well, but less rapidly than the comparable groups in either the immediate ECS or

NO-ECS conditions. This suggests that ECS given 30 minutes prior to test may have some proactive effects which were not dramatic enough to produce reliable differences, and/or that ECS is less effective in attenuating learned helplessness when not given either immediately after test as in Experiment 1 or immediately after training. At any rate, it is clear that a single ECS can attenuate or reverse learned helplessness.

GENERAL DISCUSSION

The results of the foregoing experiments have two important implications for learned helplessness theory. The first is that it was reliably demonstrated that the learned helplessness effect could be produced in rats, a finding which in the past has been difficult to obtain (e.g., Maier & Testa, 1975; Seligman & Beagley, 1975). The behavior of the yoked-inescapable groups matched the criteria which according to Maier and Seligman (1976) are characteristic of the learned helplessness effect: (1) failure to initiate the escape response in the presence of shock; and (2) failure to maintain escape behavior even after occasional successful escape responses occur. In addition, the criterion that conditions 1 and 2 above are a result of inescapability and not a result of shock per se was also matched. The behavior of helpless rats during the retest session of Experiment 1 and the test session of Experiment 2 - passive crouching and reduced squeaking - was essentially the same as the behavior of preshocked rats described by Seligman and Beagley (1975) and similar to that of helpless dogs described by Maier and Seligman (1976). It can be argued that the helpless rats have learned a competing motor response, such as "learned inactivity" (e.g., Glazer & Weiss, 1976a; 1976b) which interferes with the escape response, rather than learning response-outcome independence. Since the experiments presented here were not specifically designed to attack this argument and since a detailed rebuttal of the competing response position is presented elsewhere (e.g., Maier & Seligman, 1976), the arguments will

not be presented here.

The second important implication for learned helplessness theory was that it was demonstrated that a single ECS given either immediately after the test for helplessness or immediately following helplessness training effectively alleviated learned helplessness in rats. In addition, a single ECS given 23.5 hours following helplessness training (i.e., 30 minutes prior to test) also attenuated helplessness; however, this effect is somewhat less dramatic than immediate ECS. This suggests that ECS is most effective in alleviating helplessness when given immediately after helplessness training or immediately after the test for helplessness. Thus, it is clear that ECS attenuates a learned pattern of behavior, but it is not clear by what underlying mechanism this is accomplished (i.e., disruption of memory consolidation, blocking retrieval, inhibiting cellular uptake of NE, etc.). Since delayed ECS only partially attenuated helplessness, whereas immediate ECS almost completely eliminated helplessness, one might speculate that ECS disrupts memory consolidation (e.g., Lewis, 1969). Unfortunately, since these experiments were not designed to look at the underlying mechanism of ECS, but were intended to demonstrate whether or not ECS attenuates helplessness, such inferences would be speculative at best. For the purposes of the research presented here, it will suffice to state that ECS attenuated learned helplessness in rats and leave the question of how this is accomplished for further, more direct, experimentation with ECS.

In conclusion, by identifying a technique in addition to forcible exposure to the response-outcome contingency (Seligman, Rosellini, &

Kozak, 1975) which attenuates or reverses learned helplessness in rats, we have expanded the generality of learned helplessness theory by indicating additional parallels between rat helplessness and dog helplessness. For example, it has been shown that both forcible exposure (Seligman, Maier, & Geer, 1968) and ECS (Dorworth, 1971) reverses helplessness in dogs. In addition, since ECS alleviates depression in humans and, as demonstrated primarily in Experiment 1, also attenuates learned helplessness in rats, we have expanded the parallels between human depression and learned helplessness, thereby increasing the validity of learned helplessness model of depression.

REFERENCES

Adams, H. E. & Lewis, D. J. Electroconvulsive shock, retrograde amnesia and competing responses. Journal of Comparative and Physiological Psychology, 1962, 55, 299-301 (a).

Adams, H. E. & Lewis, D. J. Retrograde amnesia and competing responses. Journal of Comparative and Physiological Psychology, 1962, 55, 302-305 (b).

Anisman, H. & Waller, T. G. Effects of inescapable shock on subsequent avoidance performance: Role of response repertoire changes. Behavioral Biology, 1973, 9, 331-335.

Beck, A. T. Depression: Clinical, experimental, and theoretical aspects. New York: Hoeber, 1967.

Bracewell, R. J. and Black, A. H. The effects of restraint and noncontingent pre-shock on subsequent escape learning in the rat. Learning and Motivation, 1974, 5, 53-69.

Chorover, S. L. & Schiller, P. H. Short-term retrograde amnesia in rats. Journal of Comparative and Physiological Psychology, 1965, 59, 73-79.

Chorover, S. L. & Schiller, P. H. Reexamination of prolonged retrograde amnesia in one-trial learning. Journal of Comparative and Physiological Psychology, 1966, 61, 34-42.

Coons, E. E. & Miller, N. E. Conflict versus consolidation of memory traces to explain "retrograde amnesia" produced by ECS. Journal of Comparative and Physiological Psychology, 1960, 53, 524-531.

Dorworth, T. R. The effect of electroconvulsive shock on "helplessness" in dogs. Unpublished doctoral dissertation, University of Minnesota, 1971.

Duncan, C. P. The retrograde effect of electroshock on learning. Journal of Comparative and Physiological Psychology, 1949, 42, 32-44.

Ferster, C. B. A functional analysis of depression. American Psychologist, 1973, 28, 857-870.

Gellhorn, E., Kessler, M., & Minatoya, H. Influence of metrazol, insulin, hypoglycemia, and electrically induced convulsions on re-establishment of inhibited conditioned reflexes. Proceedings of the Society of Experimental Biology, 1942, 50, 260-262.

Glazer, H. I. & Weiss, J. M. Long-term and transitory interference effects. Journal of Experimental Psychology: Animal Behavior Processes. 1976, 2, 191-201 (a).

Glazer, H. I. & Weiss, J. M. Long-term interference effect: An alternative to "learned helplessness", Journal of Experimental Psychology: Animal Behavior Processes, 1976, 2, 202-213 (b).

Hebb, D. O. The organization of behavior, 1949, New York: John Wiley & Sons, Inc.

Hendley, E. D. & Welch, B. L. Electroconvulsive shock: Sustained decrease in norepinephrine uptake affinity in a reserpine model of depression. Life Sciences, 1974, 16, 45-54.

Herman, Z. The effects of noradrenaline on rat's behavior. Psychopharmacologia, 1970, 16, 369-374.

Hiroto, D. S. & Seligman, M. E. P. Generality of learned helplessness in man. Journal of Personality and Social Psychology, 1975, 102, 311-327.

Hunt, H. F. & Brady, J. V. Some effects of electroconvulsive shock on a conditioned emotional response ("anxiety"), Journal of Comparative and Physiological Psychology, 1951, 44, 88-98.

Kolb, L. C. Noyes' modern clinical psychiatry (7th ed.) 1968, Philadelphia: W. B. Saunders.

Levis, D. J. Learned helplessness: A reply and an alternative S-R interpretation. Journal of Experimental Psychology: General, 1976, 105, 47-65.

Lewis, D. J. Sources of experimental amnesia. Psychological Review, 1969, 76, 461-472.

Maier, S. F. and Seligman, M. E. P. Learned helplessness: Theory and evidence. Journal of Experimental Psychology: General, 1976, 105, 3-46.

Maier, S. F. and Testa, T. Failure to learn to escape by rats previously exposed to inescapable shock is partly produced by associative interference. Journal of Comparative and Physiological Psychology, 1974, 88, 554-564.

Masserman, J. H. & Jacques, M. G. The effects of cerebral electroshock on experimental neurosis in cats. American Journal of Psychiatry, 1947, 104, 92-99.

McKinney, W. T. and Bunney, W. E., Jr. Animal model of depression. Archives of General Psychiatry, 1969, 21, 240-248.

Miller, R. R. & Springer, A. D. Amnesia, consolidation, and retrieval. Psychological Review, 1973, 80, 69-79.

Moore, K. Effects of a methyltyrosine on brain catecholamines and conditioned behavior in guinea pigs. Life Sciences, 1966, 5, 55-65.

Nystrom, S. On relation between clinical factors and efficacy of ECT in depression. Acta Psychiatrica Scandinavica, 1964, 40, 5-140.

Overmier, J. B. Interference with avoidance behavior: Failure to avoid traumatic shock. Journal of Experimental Psychology, 1968, 78, 340-343.

Overmier, J. B. & Seligman, M. E. P. Effects of inescapable shock upon subsequent escape and avoidance learning. Journal of Comparative and Physiological Psychology, 1967, 63, 23-33.

Pinel, J. P. J. & Cooper, R. M. Demonstration of the Kamin effect after one trial avoidance learning. Psychonomic Science, 1966, 4, 17-18. (a)

Pinel, J. P. J. & Cooper, R. M. Incubation and its implications for the interpretation of the ECS gradient effect. Psychonomic Science, 1966, 6, 123-124. (b)

Pinel, J. P. J. & Cooper, R. M. The relationship between incubation and ECS gradient effects. Psychonomic Science, 1966, 6, 125-126. (b)

Quartermain, D., Paolino, R. M., & Miller N. E. A brief temporal gradient of retrograde amnesia independent of situational change. Science, 1965, 149, 1116-1118.

Rech, R. H., Bovys, H. K., & Moore, K. Alterations in behavior and brain catecholamine levels in rats treated with a-methyltryosine. Journal of Pharmacology and Experimental Therapeutics, 1966, 153, 412-419.

Sargent, W. & Slater, E. An introduction to physical methods of treatment in psychiatry, New York: Science House, 1972.

Schildkraut, J. J. The catecholamine hypothesis of affective disorders: A review of supporting evidence. American Journal of Psychiatry, 1965, 112, 509-522.

Schildkraut, J. J. & Kety, S. S. Biogenic amines and emotion. Science, 1967, 156, 21-29.

Seligman, M. E. P. Learned helplessness and depression. In the psychology of depression: Contemporary theory and Research, New York: Halstead Press, 1974.

Seligman, M. E. P. Helplessness. On depression, development and death. San Francisco: W. H. Freeman, 1975.

Seligman, M. E. P. and Beagley, G. Learned helplessness in the rat. Journal of Comparative and Physiological Psychology, 1975, 88, 534-541.

Seligman, M. E. P. & Maier, S. F. Failure to escape traumatic shock. Journal of Experimental Psychology, 1967, 74, 1-9.

Seligman, M. E. P., Rosellini, R. A., & Kozak, M. Learned helplessness in the rat: Reversibility, time course, and immunization. Journal of Comparative and Physiological Psychology, 1975, 88, 542-547.

Skinner, B. F. Are theories of learning necessary? Psychological Review, 1950, 57, 193-216.

Weiss, J. M. Effects of coping responses on stress. Journal of Comparative and Physiological Psychology, 1968, 65, 251-260.

Weiss, J. M. Effects of coping behavior in different warning signal conditions on stress pathology in rats. Journal of Comparative and Physiological Psychology, 1971, 77, 1-13. (a)

Weiss, J. M. Effects of punishing the coping response (conflict) on stress pathology in rats. Journal of Comparative and Physiological Psychology, 1971, 77, 14-21. (b)

Weiss, J. M. Effects of coping behavior with and without a feedback signal on stress pathology in rats. Journal of Comparative and Physiological Psychology, 1971, 77, 22-30. (c)

Weiss, J. M. Psychological factors in stress and disease. Scientific American, 1972, 226 (No. 6), 104-113.

Weiss, J. M., Glazer, H. I., & Pohorecky, L. A. Coping behavior and neurochemical changes: An alternative explanation for the original "learned helplessness" experiments. In G. Serban & A. Kling (Eds), Animal models in human psychobiology. New York: Plenum Press, 1976.

Weiss, J. M., Glazer, H. I., Pohorecky, L. A., Brick, J., & Miller, N. E. Effects of chronic exposure to stressors on avoidance-escape behavior and on brain norepinephrine. Psychosomatic Medicine, 1975, 37, 522-534.

Weiss, J. M., Krieckhaus, E. E., & Conte, R. Effects of fear conditioning on subsequent avoidance behavior and movement. Journal of Comparative and Physiological Psychology, 1968, 65, 413-421.

Weiss, J. M., Stone, E. A., & Harrell, N. Coping behavior and brain norepinephrine level in rats. Journal of Comparative and Physiological Psychology, 1970, 72, 153-160.

Wendt, G. R. Two and one-half year retention of a conditioned response. Journal of General Psychology, 1937, 17, 178-180.

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THE GENERALITY OF LEARNED HELPLESSNESS THEORY: EFFECT OF ELECTROCONVULSIVE SHOCK

by

Claude William Brett

(ABSTRACT)

While the learned helplessness effect has been reliably found in dogs and other species (e.g., cats, mice, fish, and humans), it has been somewhat difficult to obtain in rats. In addition, it has been demonstrated that electroconvulsive shock (ECS) reverses learned helplessness in dogs, but ECS induced reversal has not been demonstrated in the rat. Thus, the purpose of this dissertation was two-fold: (1) to determine if the learned helplessness effect could be reliably demonstrated in rats; and (2) if so, will a single ECS attenuate this phenomenon. If it could be shown that ECS attenuates helplessness, then two purposes would be served: (a) it would extend the generality of learned helplessness theory by indicating additional parallels between dog helplessness and rat helplessness; and (b) it would expand the parallels between learned helplessness and human depression, thereby increasing the validity of the learned helplessness model of depression.

In Experiment 1, rats were randomly assigned to one of three groups: escape, yoked-inescapable, and no shock control. Each rat in the escape group received 80 trials of unsignaled escapable shock. The escape group rats were required to perform a progressive fixed-ratio bar press to escape shock. The yoked-inescapable group received exactly

the same intensity, frequency, and duration of shock its escape partner received; but no response would escape shock. The no-shock control group received only pre-exposure to the training apparatus. The following day all rats were tested on a FR-2 shuttlebox escape/avoidance task. After test, half the rats in each group were given a single ECS and then were retested 24 hours later in the shuttlebox. The learned helplessness effect was clearly demonstrated during the test phase. In addition, a single ECS attenuated the learned helplessness effect in rats.

In Experiment 2 rats were given training exactly as described in Experiment 1. Following training, one-third of the rats in the escape and yoked-inescapable groups were given a single ECS immediately, one-third were given a single ECS 23.5 hours later, and one-third received no treatment. In the no-shock control group one-third of the rats were given a single ECS 24 hours prior to test, one-third of the rats were given ECS 30 minutes prior to test, and one-third of the rats were not given ECS. Then, all rats were tested 24 hours following training. The test session was identical to the test session in Experiment 1. The learned helplessness effect was clearly demonstrated during test in the NO-ECS condition. In addition, it was demonstrated that ECS attenuates or reverses learned helplessness training when given immediately following training. Delayed ECS also reverses helplessness, but less dramatically than immediate ECS.

In both experiments the criteria which characterize learned helplessness were matched: (1) Failure to initiate the escape response in the presence of shock; (2) failure to maintain escape behavior even

after occasional escape response occur; and (3) that conditions 1 and 2 above are a result of inescapability and not a result of shock per se. In addition, since ECS attenuates helplessness, the generality of helplessness theory was extended to rats, and the validity of learned helplessness model of depression was strengthened.