RETENTION OF A JUST-LEARNED BAR PRESS RESPONSE AFTER SINGLE ELECTROCONVULSIVE SHOCK

by

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CHAPTER I

Introduction

Consolidation Theory

In 1900 Müller and Pilzecker, as the result of investigations with verbal learning, proposed a "consolidation" hypothesis of learning. Lewis and Maher(1965) have given a review of the work of Müller and Pilzecker:

> They knew that a list of verbal materials interpolated between the learning of an original list and its recall would result in interference with the retention of the original list--the phenomenon of retroactive inhibition. Müller and Pilzecker hypothesized that the engrams produced by the recitation of the items from the original list were not yet consolidated and thus were susceptible to disruption produced by reciting items from the interpolated list. Although widely accepted for some years by experimenters on verbal learning, the consolidation theory has few adherents among this group today. Too few of the variables manipulated by the verbal learners show the temporal effects demanded by the consolidation theory. (1965, p. 2)

According to this consolidation hypothesis, a learning experience produces a "trace" which remains after the learning and which must continue to exist undisturbed for a finite span of time in order to insure a temporal stability for the original learning event. This finite span of time-of-trace-existence after learning is labeled the consolidation period and the activity which goes on during this period is consolidation or acquisition of temporal stability.

The words "trace" and "consolidation" may have vague psychological referents, but in order to make them more

tangible and hence more meaningful for the study of physical organisms, the existence of possible physiological correlates of the above constructs ought to be investigated. D. O. Hebb(1949, 1958) has provided quite an elaborate theoretical physiological mechanism for the concepts of "trace" and "consolidation." He has based his theorization on several observations of Santiago Ramón Y Cajal, the great Spanish anatomist. Cajal(1960: transl. from orig. Spanish, 1929) proposed that the mechanism of functional neuronal contact might be similar to the mechanism of axon growth. Both might proceed by the same mechanism: ameboid extension. This mechanism had been well established for axonal growth: the monogenetic axonal growth hypothesis proposed by His and Kupffer(Cajal, 1960). Hebb proposed that learning might possibly involve a rapid ameboid outgrowth at the synapse where the gap is not very great to begin with. This synaptic growth could decrease the extent of the synaptic gap and thereby facilitate subsequent specific neuronal association. Cajal(1960) has also stated that growth of this type is brought about by increased exercise of those parts of the nervous system in which growth occurs. To provide for "increased exercise" Hebb borrows another of Cajal's findings: re-entrant circuits observed in the central nervous system. Hebb has interpreted these circuits to be anatomical loops providing for functional neural impulse cycling or "reverberation" giving the

particular circuit the necessary exercise to stimulate axonal synaptic ameboid movement. The "trace" has been compared to the reverberating circuit, and "consolidation" has been compared to the resultant synaptic ameboid movement prerequisite to the facilitation of neuronal association which Hebb has defined as the critical operation giving learning its temporal stability.

Electroconvulsive Shock and Consolidation

Müller and Pilzecker(1900) showed that interjection of a second verbal learning task different from the first disturbed the "consolidation" of the first task so that the temporal stability of the first task was affected detrimentally as measured in retention testing. Presumably the mechanism of this effect operates by overlap of circuitry of the second "trace," since "trace" number one had not yet consolidated. It seems plausible to assume that any intense stimulus could interfere with the consolidation process to produce a detrimental effect on the temporal stability of the trace, since an intense stimulus affecting a large fraction of the brain has a high probability of involving the original "trace." Certainly a stimulus affecting activity of the entire nervous system would be expected to produce a detriment. Electroconvulsive shock (ECS) has been studied as an ideal consolidation disruptant on grounds that it is probably as intense a neural stimulus as possible, short of

lethal stimulation, and it has been shown to be a rather diffuse stimulus affecting the entire brain which would therefore have a high probability of including the consolidating circuit(Hartelius, 1952). Barring organic damage, ECS might disrupt reverberating or re-entrant circuits by bringing about massive neural fatigue momentarily halting neuronal firing. This momentary break in reverberation might stop the cycling leading to a lack of temporal stability for the prior learning event. Gunnar Holmberg(1963) reports that EEG studies have shown that "immediately after the convulsion, the EEG shows a brief period of electrical silence, followed by a gradual return of activity which is at first sporadic and slow but later assumes its preconvulsive pattern."(1963, p. 392) If it can be assumed that the EEG is reflective of neuronal activity, it would appear as though ECS does halt brain activity and hence any "reverberation" going on at the time of ECS.

Presumably the longer the interval between learning and ECS the smaller should be the decrement to the learning, since the nervous system would have had more exercise and thus more time for ameboid movement at the synapse. Duncan, in a now-classical ECS experiment, showed that learning decrement was, in fact, negatively related to learning-ECS interval. Duncan's(1949) study used avoidance learning in a shuttle-box (a two compartment apparatus wherein the animal must escape a foot shock administered in the first compartment by running to the second compartment) and showed

that the ECS-induced decrement (commonly referred to as retroactive inhibition, retroactive amnesia, or simply RA) was total at the shortest learning-ECS interval, 20 seconds, and undetectable at one hour and beyond. Intermediate intervals resulted in intermediate amounts of retroactive amnesia. In other words, within 20 seconds, ECS could abolish learning of a shuttle-box avoidance task, while after one hour, ECS would have no observable effect on the learning. Scores of studies followed Duncan's work supporting reverberatory theory and exploring parameters of ECS effects. The interested reader should consult Stainbrook(1946), Glickman(1961), Deutsch(1962), and Lewis and Maher(1965) for comprehensive reviews of behavioral ECS studies. Before long the essentially unopposed consolidationists or reverberationists stimulated enough research to promote sharp controversy.

Alternative Interpretations of ECS-produced RA

The first objections to a purely amnesic hypothesis suggested that the trauma of ECS produced aversive effects specific for the conditions of ECS administration. Friedman (1953), employing avoidance learning techniques, found what he believed to be conditioned aversive effects of ECS specific to place of electroconvulsive shock administration. All subjects were trained to bar press. Subsequently experimental S's were given one subconvulsive foot shock contingent on approaching the bar. The experimental group which

was given ECS in the training box showed less tendency to press the bar than another group given ECS outside the box. Friedman interpreted these results to suggest a confounding of amnesic and aversive effects in this situation. Consolidation disruption alone would have resulted in amnesia for the foot shock and thus essentially no depression of bar pressing. On the other hand, aversive effects alone would have resulted in bar-press depression equivalent to that obtained with subconvulsive shock, unless ECS were not perceived as being as aversive a stimulus as subconvulsive shock. In this connection, ECS is never reported to be as behaviorally depressive as is subconvulsive shock. Results of human studies pertinent to this point will be discussed later.

Coons and Miller(1960), using Duncan's apparatus, found that ECS and subconvulsive shock both result in conditioned emotionality as evidenced by increased defecation and urination. Stainbrook(1943) has reported that emotionality is observed after the second to the fifth ECS. However, human studies have shown that pain due to electroconvulsive therapy (ECT) is virtually unheard of (Zubin & Barrera, 1941). These workers have shown that fear, nevertheless, develops in the human following a number of ECT shocks and has been attributed to psychological feelings of disorientation and confusion following ECT. Such emotional states have been clearly shown to be symptomatic of brain damage(Robinson,

1963), suggesting an alternative explanation of ECS effects which will be discussed later.

Adams and Lewis(1962), who have persistently led the opposition to the consolidation theorists, have replied to Duncan's theories with several severe criticisms. First, they seconded the finding that Friedman had reported earlier, that of the situation-bound nature of ECS-produced "amnesia." Replicating Duncan's experiment, Adams and Lewis found that if ECS is given outside of the experimental apparatus, RA does not occur; however, amnesia does occur if ECS is administered in the experimental apparatus. They also found that after ECS, a recovery from amnesia will occur if the animal is allowed to stay in the non-convulsive shock compartment of the experimental apparatus for several minutes each day for five successive days. If, indeed, ameboid synaptic growth were halted by the ECS with disruption of reverberation, it is difficult to understand how the organism could recover from such an amnesia merely by sitting in the start box. The most important objection Adams and Lewis have offered has to do with "proactive amnesia" caused by ECS given before shuttle-box training. Three days after ECS in the apparatus (start box) the convulsed animals show greater difficulty in learning to avoid the start box by running to the goal box than do non-convulsed rats. Consolidation theory leaves something to be desired when trying to explain this "proactive phenomenon."

Adams and Lewis have offered an alternative explanation of ECS effects, contrary to the reverberation disruption hypothesis, but not incompatible with the aversive hypothesis. They suggest that ECS given in a certain environment conditions responses in anticipation of recurrent ECS specific to environmental cues. Rats that have received electroconvulsive shock in a box supposedly crouch in anticipation of another ECS when replaced in the box. This crouching competes with any behavior learned in the box leading to a decrement in performance of the learned response. Adams and Lewis hold that this decrement is what consolidationists have mistakenly interpreted as ECS-produced "amnesia."

Recently Lewis and Maher(1965) have modified the previous position of Adams and Lewis. According to the Lewis-Maher theory of ECS effects, the convulsive shock conditions a kind of stupor to the environmental cues, and it is this stupor which competes with any learned behavior leading to a performance decrement or "retroactive amnesia."

All of the above-mentioned experimental results were obtained under multiple electroconvulsive shock conditions (a series of ECS treatments of variable number were given after learning before retention was tested), a treatment shown to lead to emotionality(Stainbrook, 1943). The realization of this effect of multiple shock led to the use of single shock techniques and single trial learning methods.

Workers such as McGaugh(1961, 1963, 1964), Pearlman (1959, 1961), and Weissman(1964) incorporated both single-trial learning and single ECS in order to avoid emotionality and to get at the consolidation process as soon as possible after learning begins. Typical of this approach is the method employed by McGaugh(1961) wherein a rat placed on a raised platform received subconvulsive foot shock after jumping off the platform. Five seconds later, one group of animals received ECS while another did not. Apparently, the convulsed animals had not consolidated the experience of being foot-shocked for jumping off the platform. These results have been replicated by others using this technique and have been interpreted to show that electroconvulsive shock causes amnesia or consolidation disruption even when aversive effects of multiple ECS are reduced by using single ECS. However, even though single-ECS techniques reduce aversive effects of multiple ECS, it has been shown that single ECS treatments still contain measurable aversive effects (McGaugh, 1963).

Misanin and Smith(1964), using shuttle box avoidance apparatus and single ECS, reported that the avoidance decrement was response-specific. These workers found that if ECS was administered while the rat was actually performing the avoidance response (AR), there was an impairment of acquisition and retention of this AR. However, if the ECS is given before the animal could begin to avoid, the shock produced

no impairment. Since in both cases convulsive shock was introduced after acquisition of the AR and hence after consolidation and synaptic growth, it is difficult to explain why ECS would have such a differential effect in terms of reverberation disruption.

An answer to the above questions which tends to embarrass consolidation theory might be found in experiments in which ECS appears to have a facilitory effect on learning. Vanderwolf(1963) showed that a series of 21 ECS treatments before training facilitated acquisition of a bidirectional[1] shuttle box avoidance response, while the treatment did not facilitate unidirectional avoidance learning. Vanderwolf has suggested that ECS leads to damage of the neural system underlying the freezing behavior accompanying fear. This loss of freezing behavior permits an early appearance of active responses to avoid foot shock. In other words the rat's responses are not delayed by freezing behavior due to fear of the avoidance apparatus when the animal has received a series of electroconvulsive shocks prior to training. Simple unidirectional avoidance learning was unaffected because time saved by freedom from freezing is negligible for this type of single trial event. In the shuttle box rats

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[1]Bidirectional refers to the alternation of goal and start box. When the animal avoids the first start box by running to the goal box, the goal box becomes a new start box for another avoidance task. Avoidance was, therefore, an alternation and bidirectional task as opposed to simple unidirectional avoidance wherein one box is always the start box and the other is always the goal box and the animal always avoids in only one direction.

were compelled to avoid until they had reached a criterion of 9 avoidances in 10 successive trials or until they had received 140 trials. (Of course, adequate rest periods were interjected between trials.) Under these circumstances time lost due to freezing behavior was reflected in a higher number of trials necessary to reach criterion. Lack of effect of ECS on unidirectional avoidance was also interpreted as suggestive that ECS does not increase the rat's ability to learn to avoid nor does ECS supernormally motivate the animal to avoid foot shock.

Specifically, where in the brain does Vanderwolf suggest that this damage leading to disinhibition of behavior occur? He hypothesizes that "frightened animals exhibit a reciprocal relation between a tendency toward immobility and a tendency toward active movement"(Vanderwolf, 1964, p. 37). Immobility is supposedly mediated by septal-hippocampal (freezing system) structures, while active movement is mediated by brain-stem (initiating system) structures. This contention is supported by evidence suggesting the hippocampus can inhibit functions of the brain-stem reticular formation (Grastyan & Karmos, 1962; Grastyan, Lissak, Madarasz & Donhoffer, 1959). Vanderwolf mentions two specific tracts as possibly subserving an inhibitory function: (1) fornix projections to the diencephalon and tegmentum (Guillery, 1956; Nauta, 1956), and (2) projections descending from frontal cortex and passing through the subcallosal septal

area and medial hypothalamus(Kaada, 1960). It is hypothesized that ECS produces damage somewhere in the freezing system (septal-hippocampal structures) because it is in this system that we find structures notorious for their susceptibility to seizure discharges (Gastaut & Fischer-Williams, 1959).

Vanderwolf has proposed that ECS produces damage to brain inhibitory centers. Evidence relevant to brain damage after ECS may yield important results for Vanderwolf's hypothesis. A look at the literature related to brain damage after ECS does yield information pertinent for an evaluation of Vanderwolf's hypothesis. Studies such as those of Bjerner, Broman and Swensson(1944) and Heilbrunn (1943) strongly suggest the possibility of brain damage due to seizure-produced hemorrhages. Hartelius(1952), in what is probably the most carefully done neuropathological ECS study on animals reports that after a series of electroconvulsive shocks administered to cats "histopathological examination . . . reveals edema, commencing with a distension of the perivascular spaces," and "fairly slight, more or less reversible cellular changes, usually in the form of an increase in the glial elements, and irregular nerve cell degeneration."(Holmberg, 1963, p. 400) Hartelius suggests in his report that cerebral ischemia as a result of the actual seizure is the most probable causative mechanism of brain injury after ECS. Also, as mentioned previously, ECT

in human patients produces feelings of confusion and disorientation(Zubin & Barrera, 1941) which have been shown to be symptomatic of brain damage(Robinson, 1963). Hence, Vanderwolf's general prediction of ECS-produced brain damage is borne out.

But what of the specific areas of brain damage hypothesized by Vanderwolf? A look at metrazol convulsive shock--which has been compared to ECS as differing only in that metrazol convulsions are more intense(Holmberg, 1955, 1963)--may offer suggestions as to specific loci of ECS-produced brain damage. Whitehead, Neuburger, Rutledge, and Silcott (1940) conclude that the action of metrazol is to produce vascular spasms causing insufficient blood supply and anoxemia producing neuronal convulsion and resultant lesions in brain tissue. More specifically: the histological findings were paleness and degeneration of scattered nerve cells and disappearance of a few cells in the hippocampus and neuronophagia in several cells, slight glial reaction and small "gliarasen" in the temporal cortex. Similar but less marked changes were observed in the frontal cortex, thalamus and interbrain. These areas appear to include the areas suggested by Vanderwolf as loci of damage to inhibitory centers. Hence, it would appear that metrazol findings offer some reason to expect verification of Vanderwolf's assumptions in ECS histological studies to come.

In the light of Whitehead's report it comes as quite a surprise to this writer that Pearlman, Sharpless, and Jarvik (1959, 1961) in defense of consolidation theory are hard put to explain the observation in their own laboratory that a single metrazol convulsion produced very marked impairment of retention of an avoidance response four days after learning, especially since the classical reverberation time is one hour and even this seems to be contracting as consolidation continues to be studied. Rather than look for other explanations of the ECS-induced behavioral decrement, Pearlman, et. al.(1961), offer the following explanation:

> This is in contrast with the results with anesthesia, where the maximum interference time was approximately 15 minutes. It is difficult to avoid the conclusion that the mode of interference is different when the disturbing event occurs within a few minutes of the learning trial and when it occurs days later. In the first case, the disturbing event disrupts a consolidation process, which requires only a few hours at most to bring the memory to a stable, permanent condition. In the case of impairment of memory by a single convulsion or multiple convulsions days after the initial learning, it is likely that a different process is involved. It would be in accord with both clinical experience and previous experimental studies to expect that in the latter case, the memory impairment would be temporary and concomitant with the confusional state that comes as the aftermath to convulsant therapy.(1961, p. 111)

This writer would like to offer an alternative explanation to that of Pearlman, et. al., in terms of the work of Whitehead and Vanderwolf. If the behavioral task involved is studied, it is apparent that loss of "freezing behavior" could produce the "retroactive amnesia" observed after ECS

and metrazol convulsive shock. Animals were trained to a high and stable rate of bar depression and then given one foot shock contingent on lever depression. Classically the effect of this foot shock would be to cause the rat strongly to inhibit his lever depression. A treatment such as ECS or metrazol convulsive shock which might destroy portions of inhibitory tracts would disinhibit the foot shock-produced inhibition, and the animal would return to bar depression not because he had forgotten about the foot shock but because he was unable to inhibit his need for water. And since the convulsive treatment produced brain damage, it would be expected to be effective independent of the consolidation period.

Interesting behavioral evidence also points to a disinhibition-via-brain-damage hypothesis as the source of retroactive amnesia. Gellhorn(1946) and Griffiths(1961) report that extinguished bar press responses (generally thought to be brought about by active inhibition) can be reinstated by ECS. Consolidation theory would have predicted that ECS would have no effect here, since it was administered after extinction learning had consolidated: after the animals had extinguished the bar press response. The conditioned fear hypothesis would have predicted no effect on bar depression but evidence of aversive reaction to the apparatus, such as crouching, defecation and urination would have been predicted. The competing response hypothesis would have given essentially

the same prediction as the conditioned fear hypothesis except for the aversive signs. Crouching responses would be observed. The conditioned unconsciousness hypothesis would have predicted only decreased activity and no return to bar depression. The response-specific hypothesis would also predict no effect, since the only response affectable would be random movement. The brain damage disinhibition hypothesis is the only alternative explanation of ECS effects which would predict a return to bar depression by disinhibition of the inhibition involved in extinction. Therefore, Vanderwolf's theory appears to be the only useful explanatory tool left to us to explain this extinction recovery phenomenon. However, Griffiths has also reported that ECS only restores the extinguished response (bar pressing) when the electroshock is applied four hours after extinction of a bar press response while at 20 and 60 seconds ECS does not have this effect. It appears that conditions under which suggested ECS-produced brain damage occurs must be studied with an aim to discovering more of its parameters. Since the damage appears to be intimately linked with neural-circulatory interacting factors, research relevant to these factors may prove fruitful.

An Experimental Test of Hypotheses: An Approach Situation

The great majority of previous ECS behavioral studies have utilized avoidance learning procedures on grounds that

this would allow early access to the consolidation process due to the rapidity with which simple avoidance tasks can be learned. This technique has resulted in the confounding of learning with emotionality. Hence, ECS effects must be interpreted as effects on both learning and "fear of foot shock." The remaining studies utilizing approach techniques (e.g., maze learning) either are criticizable on grounds that training to criterion requires so much time that consolidation may have already occurred to a considerable extent before it can be disrupted by ECS or that the learning is highly confounded with motor activity variables. Hence, ECS may affect both activity and learning, and it is difficult to determine which factor is most affected. ECS has typically been reported to increase time of running the maze and not number of errors. Even so, Ericksen, Porter, and Stone (1948) found that after a series of ten electroconvulsive shocks a small but significant and apparently permanent deficit in maze learning ability was detected which was attributed to brain damage.

A behavioral task which avoids confounding of learning with emotionality and motor activity and yet retains the values of approach behavior and early access to the consolidation process would appear desirable for the study of ECS effects of learning alone. This investigator employed extensive magazine training prior to standard lever depression training in a box of very limited area. Lever depression is

an example of appetitive approach learning uncontaminated by aversive effects. The extensive magazine training has the effect of accelerating acquisition to criterion so that the consolidation process is available for ECS-induced disruption within five minutes of the inception of bar press training. This effect is produced by teaching the rat that the dispenser "click" signals pellet dispensation. From this point the rat learns very quickly (from the first few accidental bar presses) that bar pressing leads to the dispenser click which leads to food. The limited area of the training box has the effect of reducing the dependency of the response on motor activity as well as increasing the probability of bar depression leading to acceleration of learning. Needless to say the habit is "just learned" after five minutes, but it is readily apparent from observation that the rat recognizes that lever depression leads to pellet dispensation. Also, fortunately, the five minute learning period is ideal for testing the consolidation hypothesis, since Weissman (1964) has shown that the optimal learning-ECS interval leading to maximal retroactive amnesia is five minutes (a fact which tends to embarrass consolidation theory, since supposedly cessation of reverberation at its onset would seem to produce maximal RA).

As a test of the previously-discussed alternative explanations of ECS effects the following predictions of ECS effects in the above situation are possible:

1) Consolidation hypothesis: Since ECS should disrupt consolidation of the bar press learning, ECS should reduce rate of response to its pre-learning level, and retention testing should actually appear to be a repetition of the original learning period.

2) Conditioned fear hypothesis: ECS should produce fear of the shock environs. The fear effect should have maximal response depression either at the beginning of the retention testing period (fear conditioned to place of ECS administration) or at the end of the retention testing period (fear conditioned in terms of a temporal expectancy- the rat expects ECS after five minutes of bar pressing) or at both the beginning and end of retention testing (fear conditioned to place as well as in terms of a temporal expectancy: the place-conditioned fear gives way to the hunger drive as bar depressions result in reinforcement, but as the hunger drive is diminished and the end of the five minute period approaches fear again takes precedence in terms of a temporal expectancy).

3) Conditioned competing responses hypothesis: ECS should produce results similar to hypothesis (2) since conditioned crouching could be place or temporally specific.

4) Conditioned unconsciousness hypothesis: Same as (2) and (3) since unconsciousness decrements could also be place or temporally specific.

5) Response-specific hypothesis: No effect predicted, since critical behavior not immediately contiguous with ECS.

6) Brain damage disinhibition hypothesis: In an approach situation ECS-produced disinhibition would have little effect unless it would obliterate initial minor behavioral inhibition produced by fear resulting from handling and/or environmental change. Also, single ECS may not produce enough damage to yield a detectable disinhibiting effect.

CHAPTER II

Method

Subjects.

Subjects were 24 male albino Sprague-Dawley rats 90-120 days old. They were obtained from the Rolfsmeyer Colony, Madison, Wisconsin. All animals were maintained on a 23 hour deprivation schedule for two weeks prior to experimentation and during the actual study.

Apparatus.

The training apparatus was a standard Skinner box, which was reduced in size, thereby limiting the S to a very restricted area about the bar and food tray. The training box was placed in a larger insulated and ventilated box which minimized the effect of extraneous environmental stimuli. A variable interval programmer was used during magazine training.

The ECS apparatus consisted of a high voltage neon transformer (3000v, 30ma. A.C.) with a Hunter-type electric timer set for 0.3sec. shock duration. The neon transformer was used because it has a special built-in magnetic shunt that acts as a constant current generator. The ECS, delivered to the S's pinnae through cotton-padded alligator clips soaked in bicarbonate of soda, was invariably found to produce full tonic-clonic seizure (typical grand mal as described by Braun, Russell, and Patton, 1949) without notice-

able injury to the animal.

Procedure.

Animals were divided randomly into four groups:

Group I: Seven animals were magazine trained for four days (15 minutes per day). On the fifth day they were given five minutes of bar press training followed immediately by ECS. On the sixth day animals were tested for retention by giving them another five minutes of bar press training.

Group II: Seven animals were treated the same as Group I except that no current was administered after application of the ear-clip electrodes (mock-ECS).

Group III: Five animals were not given magazine training prior to bar press training. Animals were given fifteen minutes of bar press training (time required to equal rate of response of Groups I and II) followed immediately by ECS. On the following day a five minute retention test identical with that given Groups I and II was given.

Group IV: Five animals were treated the same as Group III except that they received mock-ECS in place of ECS.

Prior to any experimental treatment all S's were subjected to a standardized adaptation to handling and change of environment procedure: all rats were picked up, moved, and put down in the same manner. All S's were also introduced to the Skinner box environment and noise of the dispenser click for five minutes per day for three days prior to experimental treatment. Three pellets were available in the food cup each day to (1) teach the animal to eat this type of food and (2) to act as a deterrent to freezing behavior resulting from fear due to environmental change or noise of the dispenser click. Three clicks per minute at variable

intervals served to adapt S's to this stimulus as a means of preventing freezing.

Magazine training involved pretraining with the presentation of a pellet on signal (the dispenser click) at variable intervals. Three pellets were dispensed per minute for fifteen minutes per day, and all animals were exposed to magazine training for four days. By this time all rats that did not "freeze" in the Skinner box appeared to recognize that the "click" signaled availability of reinforcement. This was apparent from the animal's observed investigation of the food cup immediately after each dispenser click together with a lack of attention to the food cup between clicks. Also, the presence of an empty food cup after the magazine training was taken to be a gross indicator of the effectiveness of the magazine pretraining. Animals freezing in the training box throughout the magazine training procedure were discarded. Magazine training was accomplished with the bar removed from the Skinner box.

Twenty-four hours after the last magazine training trial the bar was placed into position in the box, and each S was allowed five minutes training time during which bar press responses were recorded on an event recorder. Immediately after learning, experimental rats were given ECS and replaced in their cages and fed. Twenty-four hours after training a five minute retention trial was given each subject. Groups III and IV differed from the above treatment

of Groups I and II only in that the initial training session lasted for fifteen minutes.

Criteria of learning were: (1) association of bar pressing with food dispensation as defined by observation of temporal contiguity between bar depression and attention to and eating from the food cup, (2) an increasing rate of bar depression or a stable high rate of response, (3) at least 7 bar presses per minute maximum rate of response (this is safely above the 3 or 4 bar presses per minute maximum rate observed as the result of random movement in the box without any reinforcement). Rats that did not satisfy all three criteria were discarded.

CHAPTER III

Results

Figures I and II represent records of group average responses per minute for Groups I and II and Groups III and IV respectively. Several statistical tests were performed to aid in the interpretation of these results. It is necessary that matched experimental and control groups have essentially the same rates of learning and approximately the same final level of responding in order to compare the groups after the experimental treatment meaningfully. In order to determine whether or not the experimental and control groups experienced differential rates of learning analyses of variance were performed on the learning segments. Tables I and II report no difference between Group I and Group II and between Group III and Group IV respectively ($F = 1.69$, $df = 1$, $p > .20$; $F = 0.69$, $df = 1$, $p > .20$). Hence rates of initial learning for Groups I and II and for Groups III and IV are not statistically different. However, Tables I and II do report that the trials or minutes-of-learning effect was significant ($F = 13.0$, $df = 4$, $p < .01$; $F = 13.7$, $df = 14$, $p < .01$). Rates of responding increase significantly from minute one to minute five. This result suggests that learning occurred and corroborates results of the previously established learning criteria.

Since Groups I and II did not have exactly equal final levels of response prior to ECS, as an added precaution they

were statistically equated by means of analysis of covariance. Table III summarizes the results of the analysis of covariance for retention between Groups I and II. The "Groups" effect only approaches significance ($F = 3.38$, $df = 1$, $0.05 < p < 0.10$). Again, the only significant effect is that of minutes-of-learning. However, Figure I shows that this time the rate of response from minute one to minute five is significantly diminished ($F = 6.7$, $df = 4$, $p < .01$).

Since Groups III and IV had exactly equal final levels of response prior to ECS, the groups were compared for retention with a regular analysis of variance. Again, the only significant effect is that of minutes-of-learning ($F = 13.7$, $df = 4$, $0.05 > p > 0.01$), although the retention minutes-of-learning effect is in the same direction as is the learning minutes-of-learning effect: rate of response is not diminished as in retention for Groups I and II. Also, both experimental and control groups (III and IV) show obvious drops in rate of response after the first training period. These observations will be discussed in a later section.

The difference in response rate between the last minute of learning and the first minute of retention was considered to be the most sensitive index of ECS effect, since re-learning effects for the first minute of retention would be minimal. A "t" test on these differences for Groups I and II and Groups III and IV gave quite insignificant values ($t = 0.2$, $p > 0.8$; $t = 0.2$, $p > 0.8$).

Figure I. Group Average Responses Per Minute For Groups I and II During Learning and Retention

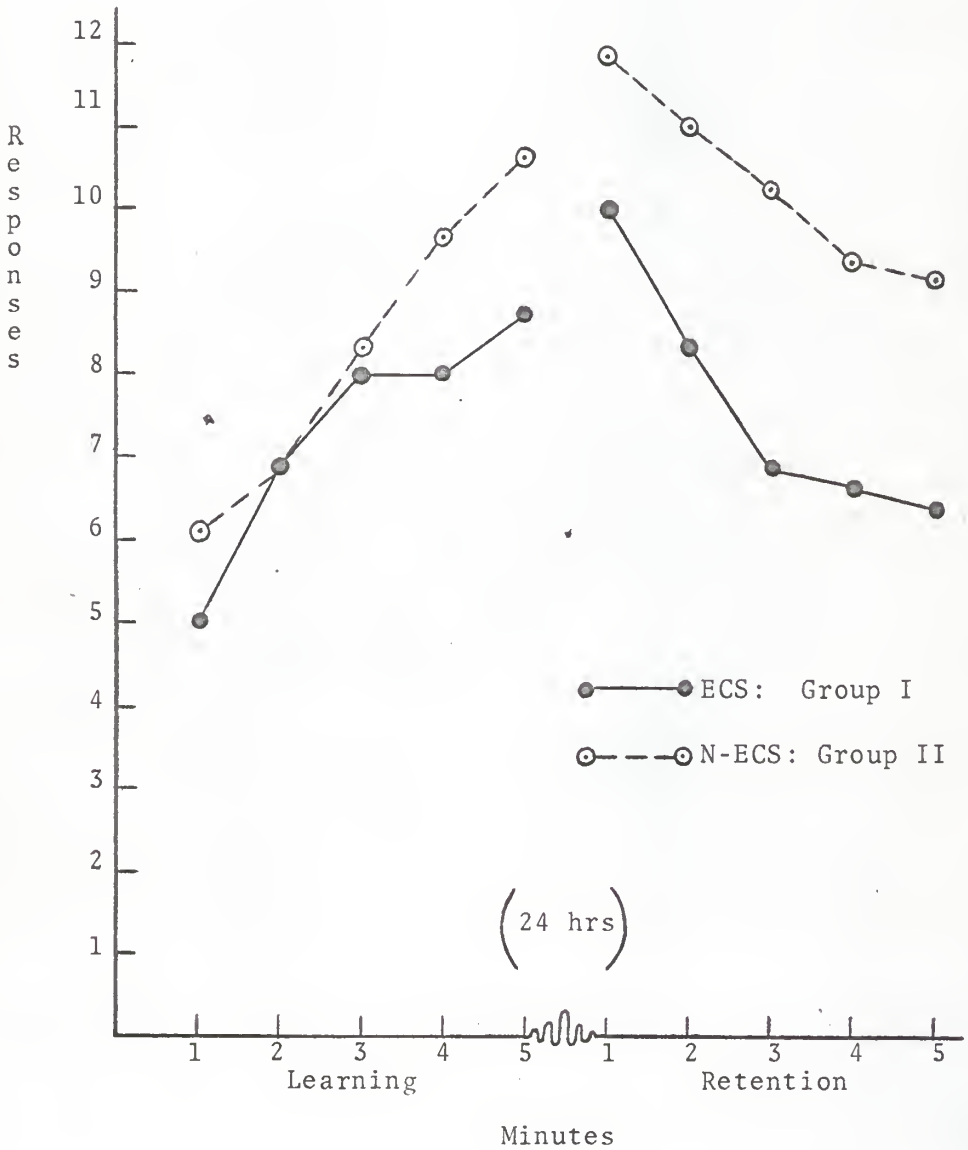

Figure II. Group Average Responses Per Minute For Groups III and IV During Learning and Retention.

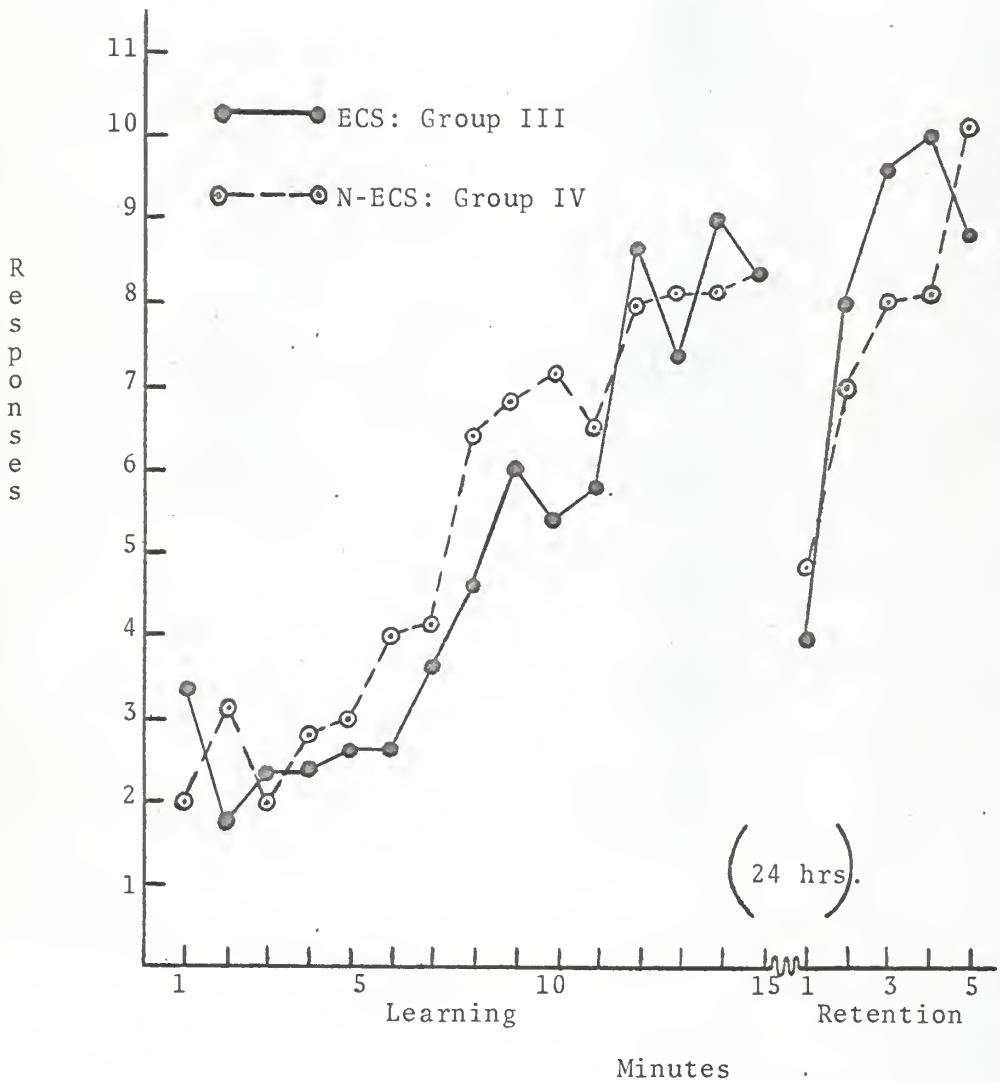

Table I. Summary Analysis of Variance for Groups I and II During Acquisition.

Sources	df	MS	F
Between-Subjects	13		
Groups	1	18.6	1.69 n.s.
error between	12	11.0	
Within-Subjects	56		
Trials	4	37.7	13.0**
Groups X Trials	4	2.6	0.9 n.s.
error within	48	2.9	
Total	69		

*$p < .05$
**$p < .01$

Table II. Summary Analysis of Variance for Groups III and IV During Acquisition.

Sources	df	MS	F
Between-Subjects	9		
Groups	1	8.6	0.69 n.s.
error between	8	12.4	
Within-Subjects	140		
Trials	14	58.9	13.7**
Groups X Trials	14	2.3	0.5 n.s.
error within	112	4.3	
Total	149		

*p < .05
**p < .01

Table III. Summary Analysis of Covariance for Groups I and II During Retention Testing.

Sources	df	MS	F
Adjusted Values			
Between:			
Total	13		
Groups	1	260.9	3.38 n.s.
error between	11	77.3	(.05<p<.10)
Unadjusted			
Within	56		
Trials	4	23.5	6.7**
Groups X Trials	4	1.2	0.3 n.s.
error within	48	3.5	

*p < .05
**p < .01

Table IV. Summary Analysis of Variance for Groups III and IV During Retention Testing.

Sources	df	MS	F
Between Subjects	9		
Groups	1	2.0	0.06 n.s.
error between	8	35.2	
Within Subjects	40		
Trials	4	43.9	13.7*
Groups X Trials	4	5.8	1.8 n.s.
error within	32	3.2	

*$p < .05$
**$p < .01$

CHAPTER IV

Discussion

Results indicate that ECS given in an approach learning situation has no detectable effect on retention performance. Hence, predictions of ECS effects by the consolidation hypothesis, the conditioned fear hypothesis, the conditioned competing responses hypothesis, and the conditioned unconsciousness hypothesis are not borne out. The only remaining alternative hypotheses are the response-specific hypothesis and the brain damage disinhibition hypothesis. This study was primarily designed to test the consolidation hypothesis in an approach learning situation, and results suggest that either ECS does not disrupt consolidation or consolidation may have to be re-evaluated as a concept explaining temporal stability of learning. Evidence previously discussed in this paper strongly suggests that if consolidation, as reverberation, does indeed follow learning and if consolidation is crucial for temporal stability of learning, then ECS should act as a disruptant to consolidation. (See "Electroconvulsive Shock and Consolidation," pp. 3-5.)

Consolidationists may reply by arguing that consolidation may have been completed in the five minute "learning" period employed. This, however, is in opposition to results reported by Weissman(1964) indicating that maximal RA occurs with a five minute learning-ECS interval.

Even so, it has been suggested (Vanderwolf, 1964) that consolidation may be greatly accelerated and thus protected from ECS disruption as a result of the magazine training employed. Groups III and IV of this study were designed to attempt to determine effects of magazine training of ECS-produced amnesia. Tables II and IV show that Groups III and IV (those not contaminated by magazine training) are still not differentiated by ECS treatment, even though the ECS was given to animals which had "just learned" the task to a level of response rate approximately equivalent to that of Groups I and II.

Although none of the groups show ECS effects, they do show other effects which require explanation. First, a depression of response rate was observed in experimental and control groups (I and II) during retention testing. Since the effect was noticed in both ECS and N-ECS groups, the depression cannot be linked to ECS. But since there is no depression in the "acquisition" segment and the only procedural difference between "acquisition" and retention segments is the presence of ear-clipping after the acquisition segment and since ear-clipping is clearly aversive to the rat, the depression may be due to an expectancy of another ear-clipping. The lack of retention depression in Groups III and IV can be explained in terms of a temporal expectancy, since ear-clipping was introduced after fifteen minutes of "learning," while "retention" was only five minutes long.

Hence the depression of response rate may have not yet appeared.

A second effect to be explained is the observed drop in response rate between the last minute of learning and the first minute of retention for both Groups III and IV. This drop can be traced to the effect of massed learning over that of learning preceded by magazine training. Apparently massed learning is not retained as readily as that preceded by magazine training, although the increased slope of the "retention" curve shows that original "learning" had a definite lasting effect.

Since the deviations of "retention" between Groups III and IV and between Groups I and II have been explained to be training procedure effects, and Groups III and IV have not been shown to be statistically different for retention, it may be concluded that magazine training did not protect the rats from ECS effects in Groups I and II. Therefore ECS can be said to have no effect in this situation.

Behavioral studies may have suggested weaknesses of the consolidation-reverberation theory of learning, but recent anatomic evidence may add to a growing suspicion of reverberation theory. In the Hebbian reverberation model growth was hypothesized from input to output or from axon ending to the next cell. To the contrary, recent evidence suggests that growth occurs from output to input or from the cell body to an adjacent axon ending. Larramendi(1965) discovered

this growth effect while studying post natal development of cat cerebellum in hopes of uncovering the mechanism of functional neuronal association. He studied post natal development in order to discover an actual neuronal growth mechanism of functional association which could suggest a mechanism for neuronal association during learning. He chose the cerebellum because of its high degree of cell-type homogeneity. This point is important for statistical considerations. The growth effect he has observed under electron microscopic analysis shows the cell body with a finger-like projection poking into an adjacent axon ending. This is interpreted as a growth effect because it occurs reliably throughout the cerebellum at a certain stage of post natal cerebellar development and disappears thereafter- the finger-like projection is withdrawn. Larramendi has suggested that such a mechanism may well be involved in the learning process.

CHAPTER V

Summary and Conclusions

Rationale of consolidation theory and of the application of ECS to the study of consolidation were presented with a general review of pertinent ECS studies and alternative explanatory hypotheses of ECS effects. It was shown that previous studies were contaminated with ECS effects on emotionality and motor activity as well as on learning. The need for an approach task incorporating values of early accessibility of the reverberation and little dependence on motor activity was established and a possible task meeting the above needs was presented. The task was standard operant lever depression learning accelerated by extensive magazine pre-training and a cut-down compartment which also delimited dependence on motor activity. The effect of ECS on the learning and the effect of magazine training on the ECS effect was tested. Both factors were found to have no effect. It was also suggested that consolidation may have to be reconsidered in the light of much contradictory evidence. It was also suggested that the ECS effect is primarily one of brain damage disinhibition.

REFERENCES

Adams, H.E., & Lewis, D.J. Electroconvulsive shock, retrograde amnesia, and competing responses. J. comp. physiol. Psychol., 1962, 55, 299-301. (a)

Adams, H.E., & Lewis, D.J. Retrograde amnesia and competing responses. J. comp. physiol. Psychol., 1962, 55, 302-305. (b)

Braun, H.W., Russell, R.W., & Patton, R.A. Duration of decrements in learning and retention following electroshock convulsions in the white rat. J. comp. physiol. Psychol., 1949, 42, 87-106. (a)

Braun, H.W., Russell, R.W., & Patton, R.A. Duration of effects of electroshock convulsions on retention of a maze habit in white rats. J. comp. physiol. Psychol., 1949, 42, 332-337.

Bjerner, B., Broman, T., Swensson, A. Acta Psychiat. Neurol., 1944, 19, 431-452.

Cajal, Santiago Ramón Y. Studies on Vertebrate Neurogenesis. Translated by Lloyd Guth. Springfield, Ill.: C.C. Thomas, 1960.

Coons, E.E., & Miller, N.E. Conflict versus consolidation of memory traces to explain "retrograde amnesia" produced by ECS. J. comp. physiol. Psychol., 1960, 53, 524-531.

Deutsch, J.A. Higher nervous function: the physiological bases of memory. Ann. Rev. Physiol., 1962, 24, 259-286.

Duncan, C.P. Retroactive effect of electroshock on learning. J. comp. physiol. Psychol., 1949, 42, 32-34.

Eriksen, C.W., Porter, P.B., & Stone, C.P. Learning ability in rats given electroconvulsive shocks in late infancy. Part I. J. comp. physiol. Psychol., 1948, 41, 144-154.

Friedman, E. ECS as a traumatic (fear producing) experience in Albino rat. J. Abn. & Soc. Psych., 1953, 48, 555-562.

Gastaut, H., & Fischer-Williams, M. The physiopathology of epileptic seizures. In J. Field (Ed.), Handbook of physiology, I. Washington, D.C.: American Physiological Society, 1959, 329-363.

Gellhorn, E. Is restoration of inhibited conditioned reactions by insulin coma specific for Pavlovian inhibitions? Arch. Neurol. Psychiat., Chicago, 1946, 56,

Glickman, S. Perseverative neural processes and consolidation of the memory trace. Psychol. Bull., 1961, 58, 218-233.

Grastyan, E. & Karmos, G. The influence of hippocampal lesions on simple and delayed instrumental conditioned reflexes. In Physiologie de l'hippocampe (Colloque internationale, No. 107), Paris: Editions C.N.R.S., 1962, 225-234.

Grastyan, E. Lissak, K., Madarasz, I., & Donhoffer, H. Hippocampal electrical activity during the development of conditioned reflexes. EEG Clin. Neurophysiol., 1959, 11, 409-430.

Griffiths, W. Jr. A note on effect of electro-convulsive shock on an extinguished response in albino rats. Psychol. Rep., 1961, 81, 154.

Guillery, R.W. Degeneration in the post-commissural fornix and the mammillary peduncle of the rat. J. Anat., 1956, 90, 350-370.

Hartelius, H. An experimental study on cats. Acta Psychiat. Neurol. Scand., Suppl. 77, 1952.

Hebb, D.O. The organization of behavior. New York: Wiley, 1949.

Hebb, D.O. A textbook of psychology. Philadelphia: W.B. Saunders Co., 1958.

Heilbrunn, G. Prevention of hemorrhages in the brain in experimental electric shock. Arch. Neurol. Psychiat., 1943, 50, 450-455.

Heilbrunn, G. & Liebert, E. Biopsies on brain following artificially produced convulsion. Arch. Neurol. Psychiat., 1941, 46, 548-550.

Heilbrunn, G. & Weil, A. Pathological changes in the central nervous system in experimental electric shock. Arch. Neurol. Psychiat., 1942, 47, 918-930.

Holmberg, Gunnar. Biological aspects of electroconvulsive therapy. Intern'l. Rev. of Neurobiology, 1963, 5, 389-412.

Horowitz, M.W., & Stone, C.P. The disorganizing effects of electro-convulsive shock on a light discrimination habit in Albino rats. J. comp. physiol. Psychol., 1947, 40, 15-21.

Kaada, B.R., Rasmussen,E.W., & Kveim, D. Impaired acquisition of passive avoidance behavior by subcallosal, septal, hypothalamic, and insular lesions in rats. J. comp. physiol. Psychol., 1962, 55, 661-670.

Larramendi, L.M.H., 1965, Personal Communication.

Lewis, D.J., & Maher, B.A. Neural consolidation and electro-convulsive shock. 1965, Unpublished paper.

Madsen, M.C. & McGaugh, J.L. The effect of ECS on one-trial avoidance learning. J. comp. physiol. Psychol., 1961, 54, 522-523.

McGaugh, J.L. Analysis of the amnesic and fear-inducing effects of electroconvulsive shocks. Paper delivered at Western Psychological Association, 1963.

McGaugh, J.L., & Madsen, M.C. Amnesia and punishing effects of electroconvulsive shock. Science, 1964, 144, 182-183.

Misanin, J.R., & Smith, N.F. Role of response-linked fear in the effects of a single ECS on an avoidance response. J. comp. physiol. Psychol., 1964, 58, 212-216.

Müller, G.E., & Pilzecker, A. Experimentelle Beitrage zur Lehre vom Gedachtnis. Z. Psychol., 1900, Suppl. No. 1.

Nauta, W.J.H. An experimental study of the fornix system in the rat. J. comp. Neurol., 1956, 104, 247-271.

Pearlman, C.A., Sharpless, S.K., & Jarvik, M.E. Effects of ether, pentobarbital, and pentylenetetrazol of ether upon one trial learning of an avoidance response. Fed. Proc., 1959, 18, 432.

Pearlman, C.A., Sharpless, S.K., & Jarvik, M.E. Retrograde amnesia produced by anesthetic and convulsant agents. J. comp. physiol. Psychol., 1961, 54, 109-112.

Porter, P.B., Stone, C.P., & Eriksen, C.W. Learning ability in rats given electroconvulsive shocks in late infancy. Part II. J. comp. physiol. Psychol., 1948, 41, 423-431.

Robinson, V. (Ed.), The new modern home physician. New York: Wm. H. Wise & Co. Inc., 1963.

Routtenberg, A., & Kay, K.E. Effect of one electroconvulsive seizure on rat behavior. J. comp. physiol. Psychol., 1965, 59, 285-288.

Stainbrook, E.J. Maze behavior of rat after ECS. J. exp. Psychol., 1943, 33, 247-252.

Stainbrook, E.J. Shock therapy: psychological theory and research. Psychol. Bull., 1946, 43, 21-60.

Vanderwolf, C.H. Improved shuttle-box performance following electroconvulsive shock. J. comp. physiol. Psychol. 1963, 56, 983-986.

Vanderwolf, C.H. Effect of combined medial thalamic and septal lesions on active-avoidance behavior. J. comp. physiol. Psychol., 1964, 58, 31-37.

Weissman, A. Effect of electroconvulsive shock intensity and seizure pattern on retrograde amnesia in rats. J. comp. physiol. Psychol., 1963, 56, 806-810.

Weissman, A. Retrograde amnesic effect of supramaximal electroconvulsive shock on one-trial acqusition in rats: a replication. J. comp. physiol. Psychol., 1964, 57, 248-250.

Whitehead, R.W., Neuburger, K.T., Rutledge, E.K., Silcott, W.L. Pharmacologic and pathologic effects of repeated convulsant doses of metrazol. Amer. J. of Med. Sciences, 1940, 199, 352-359.

Zubin, J., & Barrera, S.E. Effect of electric convulsive therapy on memory. Proc. Soc. Exp. Biol. Med., 1941, 48 596-597. (Abstract).

ADDENDUM

Since the completion of the above study, several very recent works important for the interpretation of the above report have come to this writer's attention. Two of these studies were specifically designed to test the effect of ECS in approach learning situations. The first experiment (Chevalier, 1965) reports no RA after ECS, while the second experiment(Tenen, 1965) clearly shows amnesic effects of electroconvulsive shock. A closer look at these studies may suggest possible clues to the source of these conflicting reports.

Chevalier(1965) gave young rats(39 days) single ECS (100v.a.c., 0.3sec., corneal electrodes) five minutes after the initiation of reversal training on an underwater T-maze and did not detect RA. The use of a five minute training-ECS interval would appear appropriate in light of work by Weissman(1963, 1964). Recent work important for the training-ECS interval will be discussed later. However, one might argue that use of the water-maze learning task may not actually separate appetitive and aversive motivations. Avoidance of water and drowning may be just as important as seeking air in this task. If such is the case, then this method cannot be said to definitively test the effect of ECS in a purely approach learning situation. Nevertheless, the results reported by Chevalier have considerable importance

for consolidation theory, since past work would have predicted RA here. Inability to detect any effect of ECS in this task casts more doubt on reverberation-consolidation theory. What would competing ECS effects theories have predicted in this situation:

1) Conditioned fear hypothesis: ECS should have produced fear of the shock environs, and this fear should have competed with the motivation for air producing an increase in errors.

2) Conditioned competing responses hypothesis: ECS should have produced results similar to (1).

3) Conditioned unconsciousness hypothesis: ECS should have produced results similar to (1) and (2).

4) Response-specific hypothesis: No effect is predicted, since the response was not contingent with the ECS.

5) Brain damage disinhibition hypothesis: ECS should not have produced any effect, since inhibition of behavior was not crucial to the response.

The only predictions consistent with Chevalier's results are those derived from the response-specific hypothesis and the brain damage disinhibition hypothesis.

But what of the conflicting report by Tenen(1965)? He gave ECS(150m.a., 0.2sec., ear-clip electrodes) to rats 22 seconds after they had begun to receive water reinforcement for hole exploration: head insertion into a hole in the wall in the training compartment. Rats that had not received ECS showed increased incidence of hole exploration, while animals that had received ECS showed no consequent increase of hole exploration. However, animals given delayed ECS(shock was

administered 3 hours after learning- post consolidation) showed an increase of hole exploration similar to that of non-ECS animals and hence no RA. Rats given foot shock(650v. through a 330,000 ohm series resistor) in a different environment showed no RA.[1] Apparently the subjects given ECS had suffered amnesia for the water reinforcement. The above results serve as a direct contradiction of the findings reported in this thesis, and this writer would call attention to two factors as possibly contributing to the discrepancies between these reports: (1) Tenen used 150ma. as opposed to 35ma. used by this writer (brain damage is generally correlated with intensity and duration of current), (2) Tenen's learning-ECS interval was 12 to 22 seconds, while this researcher used a five minute learning-ECS interval. (This point is discussed further below.)

Chorover and Schiller(1965) gave ECS(30-50ma., 0.2sec., ear-snap electrodes) to rats from 0.5 to 60.0 seconds after single passive avoidance training. They report: "However, unlike earlier studies, impairment was observed only at relatively short(0.5-10.0sec.) ECS-delays"(1965, p. 73). An important factor to consider when reviewing ECS studies is the relative inconstancy of ECS stimulus parameters. ECS

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[1] An interesting point for consolidation theory is that while Müller and Pilzecker observed RA following interjection of a second list of nonsense syllables before the first list had consolidated, such treatments as severe foot shock and audiogenic seizures (full tonic-clonic) have not led to RA(1963, Stern & Gollender).

current typically ranges from 25 to 150ma.(RA may be directly related to amount of current just as tissue damage is related to amount of current), the stimulus duration is variable around 0.2 to 0.3 seconds, and point of application has varied from cornea to ear (this may alter effective ECS current). These factors quite probably are important sources of variability in results reported. On the other hand, Chorover and Schiller's results may have important implications for learning theory in general. If ECS cannot produce RA 10 seconds after learning, perhaps a lengthy consolidation-reverberation period--as suggested by Hebb--is not a necessary process for learning. Or as Chorover and Schiller put it: "Second, assuming that ECS produces RA which, irrespective of whether it is 'brief' or 'prolonged,' appears to be a true amnesia (i.e., loss or absence of memory), is it valid to attribute this effect to interference with 'memory trace consolidation?'(1965, p. 78).

Before a definitive answer can be given to the above question of ECS effects, techniques must be standardized, and programmatic studies must be employed. More carefully controlled histological, lesion, and electrophysiological studies must also be initiated before theories suggesting ECS-produced brain damage can be verified. However, this writer believes that the argument presented in this paper offers strong reason to expect the ultimate predominance of a brain damage hypothesis of ECS effects.

REFERENCES

Chevalier, J.A. Complete retention of t-maze reversal following single post-trial ECS. Paper delivered at MPA, 1965.

Stern, R.M., & Gollender, M. Audiogenic seizures: are they remembered? J. comp. physiol. Psychol., 1963, 56, 979-982.

Tenen,S.S. Retrograde amnesia from electroconvulsive shock in a one-trial appetitive learning task. Science, 1965, 148, 1248-1250.

RETENTION OF A JUST-LEARNED BAR PRESS RESPONSE AFTER SINGLE ELECTROCONVULSIVE SHOCK

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The effect of ECS was tested in a new approach situation incorporating early accessibility of the consolidation process and low dependence on motor activity. Magazine training prior to bar press training in a cut-down "Skinner box" provides the desirable qualities. ECS is shown to have no effect in this approach situation, and magazine training is also shown to have no effect on the ECS effect. Suggestions are made for a reconsideration of reverberation theory, and Vanderwolf's hypothesis of brain damage disinhibition produced by ECS is suggested as the most plausible effect of electroconvulsive shock.