AMNESIC AND DISINHIBITORY EFFECTS OF ELECTROCONVULSIVE SHOCK

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SCOPE AND CONTENTS: Electroconvulsive shock (ECS) produces a loss of memory for the immediately preceding period, but also produces non-amnesic effects which seriously complicate the interpretation of behavioral results following convulsions. The results of the present investigation indicated that the retrograde amnesia produced by ECS is probably slight, but appears enhanced in passive-avoidance tasks and diminished in aversively-motivated tasks requiring movement, because of a concomitant impairment of movement-inhibiting mechanisms. If this interpretation is valid, it is extremely difficult to make quantitative estimates of the degree or temporal extent of the retrograde amnesia induced by ECS in animals. It may be possible, however, to separate memory mechanisms from movement-inhibiting mechanisms with procedures involving more localized effects upon the brain.

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INTRODUCTION

Impairments or losses of memory are frequently observed in cases involving closed-head injuries, convulsions, meningitis, acute cerebral anoxia and certain other diseases or traumatic events. These losses or impairments of memory are called amnesia*. Loss or impairment of memory for events which occur after the disease or trauma is usually called post-traumatic amnesia, while loss or impairment of memory for events which preceded the disease is called retrograde amnesia. The principal concern here is with retrograde amnesia**.

The usual interpretation of retrograde amnesia is that experiences generate some cerebral process which is, at first, dynamic or unstable. With the passage of time, this unstable process somehow becomes fixed or "consoli-

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*From a linguistic point of view, amnesia means loss of memory and memory impairment is called dysmnesia. However, although dysmnesia is sometimes used, particularly in Britain, more commonly loss and impairment are considered together as amnesia.

**Where the term amnesia is used alone in this document, it means retrograde amnesia.

dated" into a stable, relatively permanent memory system. Events which disrupt or prevent the brain functions necessary for this consolidation therefore produce an amnesia for a period preceding the event. The period before the disrupting event for which memory is lost or impaired is often called the consolidation period.

Thus the significance of retrograde amnesia lies in its support of the so-called consolidation theory. Similarly, it is widely held that the consolidation period and the hypothesized consolidation process can be understood through studying retrograde amnesia and, for this reason, much of the current neurological theorizing about memory is based upon observations of retrograde amnesia and conclusions about consolidation mechanisms. Thus retrograde amnesia is clearly an important phenomenon in formulating neurological ideas about memory and the present thesis involves a study of retrograde amnesia induced by electroconvulsive shock (ECS).

The idea that consolidation or fixation of unstable neural processes is required for remembering arose at least several decades ago. Hamilton in 1875 read a paper to the Medico-Legal Society of New York in which he described 26 cases of retrograde amnesia produced by traumatic head injuries (Hamilton, 1876, 1886), and this paper suggested to Ribot (1892) that "in order that a recollection may or-

ganise and fix itself, a certain time is necessary which, in consequence of the cerebral excitement, does not suffice."

Ribot (1892) also used a similar explanation for the progressive amnesia which he observed in "paralytic and senile dementia:"

> The progressive destruction of the memory descends from the unstable to the stable recollections. Recent impressions not sufficiently fixed...represent the weakest degree of recollection and disappear first of all; old impressions, well fixed..., in short all impressions which represent the stable form of recollections, disappear last.

Since these comments formed part of a well-known dictionary of psychological medicine, the idea of fixation or consolidation of memory was probably not new at the time he wrote. Certainly, very similar conclusions were advanced in his book, Diseases of Memory, written 10 years earlier (Ribot, 1882), in which he put forward the "law of regression or reversion:" that "the process or organization (he seems to mean in the sense of becoming organic or organically registered) is variable and is comprised between two extreme limits: the new state - (and) organic regis-

tration" (p. 122).

Ribot (1882) knew, of course, that retrograde amnesia was a common symptom of head injury and convulsions but he could not tell if this represented an impairment of "registration" or of "revivification" or "reproduction" of information (p. 97) -- a question still unanswered today. He believed, however, that organic registration or fixation depended upon nutrition (pp. 193, 195) whereas reproduction or revivification of memories depended upon circulation (p. 197). This view may have been part of the conventional wisdom of the time because Carpenter (1890), in an apparently widely-used textbook of mental physiology, had similar ideas. Carpenter (1890, p. 450), for example, said that retrograde amnesia is "still more direct and cogent evidence of the dependence of Memory upon a registering process that consists in some Nutritive modification of the Brain-tissue."

Although Ribot (1882) thought that improvements or "exaltation" of memory could be induced by stimulants (p. 199), he did not discuss reminiscence phenomena. On the other hand, Carpenter (1890) pointed out that:

> The same indication that <u>time</u> is needed for the effectual performance of the registration may be drawn from ... what we call 'learning by heart' ... and we seem able

> to trace the Physiological working of this process, in the fact known to every school-boy who has to commit to memory fifty lines by Virgil, that if he can 'say them to himself,' even slowly and bunglingly, just before going to sleep, he will be able to recite them much more fluently in the morning.

He concludes from this that "we have here an obvious indication that the renovation of the brain substance which takes place during sleep ... gives time for the fixation of the last impressions" (italics original).

Thus it seems that, by the end of the 19th century, the idea of a consolidation process was widely accepted and that then, as now, it was based upon observations of retrograde amnesia, drug-induced enhancement of learning and reminiscence phenomena. The interim period has primarily involved more precise observations and the use of animal experimentation.

In the next two parts of the present document, the general clinical impressions of retrograde amnesia will first be summarized and then observations and speculations deriving from the administration of electroconvulsive shock to animals will be reviewed.

I. CLINICAL RETROGRADE AMNESIA

Observations of retrograde amnesia resulting from closed-head or blunt injuries have been summarized by Russell (1959) and by Whitty and Zangwill (1966): a fairly common clinical pattern is associated with such cases. After recovering consciousness, the patient is usually confused and disoriented and retrograde amnesia affecting a relatively long time before the injury is commonly seen.

Post-traumatic amnesia (that is, amnesia for current events) frequently accompanies the retrograde amnesia, and a rough correlation exists between the duration of the post-traumatic amnesia and the period affected by the retrograde amnesia. Thus, it is rare to find a case without reported retrograde amnesia if the post-traumatic amnesia persists for more than an hour or so. Generally speaking, retrograde amnesia is reported in about 85% of the cases of blunt or closed-head injuries and some post-traumatic amnesia is reported in almost all such cases if there is a loss of consciousness.

As the patient improves with respect to current memory, lucidity and orientation, the retrograde amnesia

gradually "shrinks" in most cases until it affects only the period of a few moments before injury and, in some cases, the retrograde amnesia disappears altogether. Contrary to the usual supposition, the amnesia rarely shrinks in strict chronological sequence from the remote to the recent past (Zangwill, 1964; Whitty & Zangwill, 1966). More commonly, isolated recollections or "island of memory" are reported to occur, in what seems a haphazard sequence. Sometimes these islands are, at first, temporally disoriented. With the passage of time, further recollections are seen to occur which become correctly related in time to one another and to the "last memory" which was reported at first. The final result of this pattern of "islands" and "bridges" is a reported residual retrograde amnesia which extends to less than 1 min. in more than 50% of the cases, or no residual amnesia in about 15% of the cases (Russell, 1959).

Very rarely, however, cases are reported of prolonged retrograde amnesias following closed-head injuries. These may extend to days and even months. In such cases, very severe and general brain damage, and a correspondingly long post-traumatic amnesia, are usually also reported. Since most reported prolonged retrograde amnesias have occurred under conditions of battle, and since there are few well-attested cases of this type, they tend to be viewed with some suspicion and no satisfactory explanation has been ad-

vanced for these cases.

There are obviously great difficulties in assessing the length of the period affected by retrograde amnesia in clinical cases, but, generally, the observations of patients with closed-head injuries suggest the usual occurrence of a residual amnesia which extends to a few moments before injury or else no amnesia at all. This was also Hamilton's (1876, 1886) impression.

This retrograde amnesia following closed-head injuries is sharply contrasted with the effect of penetrating brain wounds, such as gunshot wounds. Penetrating brain wounds are almost never followed by retrograde amnesia, and, if there is any such amnesia, it usually disappears quickly. Occasionally, as with closed-head injuries, penetrating brain wounds produce a retrograde amnesia which extends back a long time before injury. These cases are accompanied by "general dementia" and the injury involves tearing of subcortical white-fibre tracts and other very severe and extensive brain damage (Whitty & Zangwill, 1966).

Russell (1948) has suggested that, in his cases of retrograde amnesia following penetrating brain wounds, the significant site of injury was probably the temporal lobes; Whitty and Zangwill (1966) are also inclined to this view. The conclusion of a temporal-lobe site of injury is derived indirectly since full autopsy studies are not available

but the conclusion makes sense in view of the marked memory impairments following surgical resection of the temporal lobes (Milner, 1966).

It may also be that the retrograde amnesia following closed-head injury is also produced by temporal-lobe damage. Since the injury-producing object does not penetrate the brain, it seems likely that the amnesia results in part from the contrecoup effects of the injury. A blow on the occipital region of the skull, for instance, would cause the brain to shift anteroventrally so that the temporal lobes would contact the irregular surface of the cranial vault. Thus, although a severe blunt injury involves a fairly general shift of the brain, the direction and landing-site of the blunt object will affect the site of any contrecoup damage, and this may be a factor in determining whether or not retrograde amnesia results. Systematic investigation of the site and direction of injury as it relates to retrograde amnesia is difficult and does not seem to have been undertaken. It might, however, prove extremely useful in formulating a coherent explanation of retrograde amnesia in general.

Retrograde amnesia is also a commonly reported sequel of electroconvulsive therapy (ECT) and more formal experimental methods can naturally be applied to this amnesia. The major problem of interpretation is that most

of the patients to whom ECT is given suffer from psychotic disorders and often have received several ECTs as well as medication. Nonetheless, ECT-induced retrograde amnesia shows many striking similarities to the retrograde amnesia produced by closed-head injuries (Williams, 1966).

For example, following ECT, patients at first cannot remember events which occurred relatively long before the treatment but, with the passage of time, there is considerable shrinking until the residual retrograde amnesia is only for a few seconds before the convulsion. The pattern of shrinking is not always in chronological sequence from the remote to the recent (Ebtinger, 1958 cited by Williams, 1966) but rather, as with closed-head injuries, involves the appearance of "islands" of memory and the subsequent "bridging" between the islands.

There is, in the case of ECT, some dispute about the severity or even existence of retrograde amnesia. While most patients do not recall visual stimuli presented to them a few seconds before ECT administration, these stimuli can sometimes be selected in a choice-recognition test (Mayer-Gross, 1943) or can be recalled with prompting (Williams, 1966). Aids of this sort also help in cases of closed-head injuries (Whitty & Zangwill, 1966) and of temporal lobe damage (Walker, 1957) however. More importantly, Hemphill (1940) reported no retrograde amnesia from ECT

for visual stimuli presented beforehand and he says that many patients remember the electrodes being put on and the passage of test currents before the ECT. However, Hemphill (1940) presented his stimuli 30 min. before treatment and does not report the interval between putting on the electrodes and delivering the current. Since this interval involved test currents, it must have been at least many seconds long. As the recent reports generally show a fairly brief retrograde amnesia following ECT, it is quite conceivable that some patients could recall having the electrodes put on because several seconds or even minutes intervened.

Although the nature of the amnesic effect produced by ECT remains somewhat obscure, and many procedural weaknesses can be pointed out in investigations of this effect, it seems fairly clear that ECT usually does produce a retrograde amnesia, and the best conclusion seems to be that it is a fairly brief amnesia.

Thus, it seems that the amnesias produced by ECT and by closed-head injuries are highly similar and probably have a common neurological basis. These amnesias are quite brief in the residual form, probably less severe than previously supposed, and may involve injury of the temporal lobes. These conclusions will assume some importance in interpreting the animal experimentation in general and the presently reported experiments in particular. The animal

experimentation, however, is somewhat less uniform in results than the clinical observations and forms the next part of this document.

II. ANIMAL EXPERIMENTS

Retrograde amnesia has been reported many times in laboratory experiments with animals as a result of anoxia, "spreading depression," convulsions, and the administration of certain drugs and antibiotics. Electrically-induced convulsions have been used in experiments involving animals since shortly after the clinical inception of ECT (Munn, 1950, p. 443 ff.) but, unlike the clinical situation, interpetation of the results of convulsions in animal experiments has involved considerable controversy. The first explicit conclusion that convulsions* induced retrograde amnesia in animals was made by Duncan (1949) and, since this report, experiments investigating retrograde effects of convulsions upon learning have abounded. These kinds of experiments have been reviewed several times (Glickman, 1961; Deutsch, 1962, 1969; Hudspeth & Gerbrandt, 1965; McGaugh, 1966; Weiskrantz, 1966; Spevack & Suboski, in press).

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*The term "convulsion" means "electrically-induced convulsion" in this document unless otherwise specified.

Most of the behavioral investigations of the effects of convulsions on animals have been addressed to certain major questions or issues. Since these questions arose in a chronological sequence, the present discussion is organized around these questions in order to impart a historical perspective for the experimental report which follows. The present discussion is therefore not wholly exhaustive. Rather, evidence considered most pertinent to the major questions of convulsive effects in animals is considered. Since many of these questions remain open, and numerous investigations continue to be reported, it was necessary to consider experimental reports published prior to a certain date. For the most part only experimental reports published prior to the beginning of 1969 have been included here.

The Question of Retrograde Amnesia

In a wide variety of test situations, it has been found that convulsions induced shortly after a learning experience impaired subsequent retention of the learned response, when convulsed animals were compared to animals which had not had convulsions. The first demonstration of this effect in animals was reported by Duncan in 1949. Duncan concluded that the convulsions induced amnesia for the learning by disrupting the consolidation period presumed to follow each learning experience. On the basis of his

results, he estimated this consolidation period as "less than 1 hr. and very probably ... not significant longer than 15 min."

At about the same time, Hebb (1949) suggested that the physiology of learning involved a two-phase process. Hebb's idea was that an experience generated a reverberating, closed-circuit process in the forebrain which served as a "holding system" until some more permanent modification occurred in the relevant neural mechanisms. It seemed eminently sensible that the massive currents used to induce convulsions would eradicate a reverberating trace system, which depended upon relatively delicate and precisely-timed nerve impulses for its integrity, while leaving unimpaired any already-permanent modifications in the brain.

Duncan's (1949) experiment, coupled with Hebb's (1949) influential theoretical ideas and with the clinical evidence of amnesic effects of convulsions, left the retrograde-amnesia explanation for convulsive effects in animals unchallenged for more than a decade and it is still a widely held notion today. In 1960, however, Coons and Miller suggested that Duncan's (1949) results could have occurred because the convulsion was an aversive event.

The Question of Fear

In order to understand Coons and Miller's (1960)

suggestion, Duncan's (1949) procedure must first be outlined. Duncan had trained rats on a one-way, active-avoidance task, giving one trial per day followed at varying intervals by a convulsion. His results were that the closer to the training trial the convulsion occurred, the slower the learning. According to Coons and Miller (1960), this result was to be expected if the convulsion was an aversive event because it meant that the ECS was, effectively, punishing the occurrence of the avoidance response. Naturally, the more contiguous the training and convulsion were, the more effective the ECS would be at stopping the avoidance response.

Coons and Miller (1960) also reported an experiment which supported this reinterpretation. They first trained animals to make a one-way, active-avoidance response using Duncan's (1949) technique and then, subsequently, trained the animals to remain in the start compartment by shocking them for making the original response. In this case they reported that the closer to the training trial the convulsions occurred, the faster the animals learned to remain in the start compartment. Thus, they reasoned, any amnesic effect of the convulsions "was overridden by increased fear induced by the ECS". Presumably this "aversive ECS" notion also explained Thompson and Dean's (1955) result that, in a visual-discrimination task involving active

avoidance, the sooner after reaching criterion a single convulsion was given, the slower the relearning.

Incidentally, Coons and Miller (1960) reported that both in their experiment and in their replication of Duncan's (1949) experiment, convulsions given 60 sec. or more after each training trial produced no performance changes, compared to non-convulsed control animals. On the other hand, according to Thompson and Dean (1955), a single convulsion 1 hr. after reaching criterion still exerted some detrimental effect on relearning.

The Question of Competing Responses

Two years after Coons and Miller's report, a fairly subtle alternative was proposed by Adams and Lewis (1962a, 1962b). They reported that convulsions induced in the training apparatus impaired active-avoidance learning more than convulsions induced outside the apparatus. This observation seems to have been made earlier as well (Hayes, 1948 cited by Munn, 1950, p. 445). In the experiments of Adams and Lewis, the convulsions were induced 15 min. after each of the first 6 daily trials. Based upon this evidence (Adams and Lewis, 1962b) plus the results of some other experiments (Adams & Lewis, 1962a; Lewis & Adams, 1963), these investigators suggested that ECS served as an unconditioned stimulus, producing a response which became condi-

tioned to the apparatus. Giving ECS thus produced a "conditioned convulsion" which "competed" with the originally-learned response to produce an apparent retrograde amnesia. While Adams and Lewis were not explicit on the point, they presumably meant that the ECS conditioned a "freezing" or immobility response which, in an active-avoidance situation, would tend to impair acquisition.

Between 1960 and 1966, most of the ECS work with animals was addressed to these questions so that it is probably best to forego later theoretical suggestions until developments on these first three questions are outlined.

Amnesia, Aversion and Competition

The major procedural problem with the early work was that it involved several training trials and, usually, several convulsions as well. Since the learning could not be accurately located in time, the consolidation period could not be quantitatively estimated. Therefore, techniques were needed to produce one-trial learning so that investigators concerned with the amnesia suggestion could undertake more precise experiments. An appropriate one-trial learning technique was described in 1960 by Jarvik and Essman, and it was quickly adopted for experiments investigating retrograde effects of convulsions.

The essential technique involved placing an animal

on a small, insulated platform located on a larger, electrified grid floor. In the original apparatus (Jarvik & Essman, 1960), the platform was actually an elevator placed against one wall, and mice were lowered on it from the top of the apparatus to the grid floor below. In many subsequent experiments, particularly where rats were used, the platform was fixed in the middle of the grid floor and the animal was simply placed on the platform at the start of the trial. In either case, when the animal stepped off the platform, which usually happened within a few seconds, it received a shock through its paws. Testing with the same procedure 24 hr. or so later showed a reliable and marked increase in latency for stepping down, and so it was concluded that this was a one-trial, passive-avoidance task. This task was deemed especially useful for investigating the effect of convulsions because, like Coons and Miller's (1960) task, aversive effects of convulsions would increase step-down latencies while amnesic effects would decrease the latencies.

Therefore, when Madsen and McGaugh (1961) found that a single convulsion, 5 sec. after an animal stepped down, produced shorter step-down latencies in a subsequent test than no convulsion, they concluded that this was a demonstration of retrograde amnesia. Their conclusion was strengthened and extended by the results of several other

experiments. In a task where animals were first trained to press a bar for food and were then shocked once through the bar, it was found that the sooner a single convulsion followed the shock, the more bar pressing (or less passive avoidance) occurred in a subsequent test (Heriot & Coleman, 1962). In this case, the convulsion followed the passive-avoidance trial by 1, 7, 26, 60, or 180 min. and all the groups except the 180 min. group pressed more often than non-convulsed control animals. Weissman (1964) explicitly set out to replicate Heriot and Coleman's (1962) experiment using bar-pressing for water rather than for food and using ESC-delay intervals of 1.25, 2.5, 5, 10, 20, 40, 80 and 160 min. Compared with non-convulsed control animals, he found more bar-pressing up to ECS-delay intervals of 5 min. and 40 min., depending upon the level of statistical significance used. King (1965) conducted a similar investigation, in which animals were first trained to run in a two-compartment box for water and then received gridshock upon entering the goal compartment. A convulsion followed the gridshock by 1.25, 5, 15 or 60 min., and, compared with non-convulsed control animals, a convulsion produced shorter latencies in the first two groups. Actually, none of the experiments except King's (1965) produced strictly monotonic results from increasing the ECS-delay interval but, just the same, the idea that a single convulsion produced

retrograde amnesia became almost universally accepted.

At the same time, the idea that ECS impaired subsequent performance because of "conditioned convulsions" which competed with the correct response was not faring so well. Some evidence compatible with this viewpoint continued to be reported (Adams & Peacock, 1965a, 1965b; Misanin & Smith, 1964) and a slight controversy about the application of this viewpoint occurred in print (Gerbrandt, 1965; Maher & Lewis, 1966; Gerbrandt, 1966). However, it was also observed several times that the place of ECS administration did not affect its retrograde effect in one-trial learning tasks (Leonard & Zavala, 1964; Quartermain, Paolino & Miller, 1965; Gerbrandt & Thompson, 1964). Nor did attaching earclips alter the retrograde effects of a single convulsion in an open-field activity test (Nielson, 1968), although the attaching of the earclips should have served as an effective conditioned stimulus, since they had been used to induce the convulsion. Attaching the earclips for testing induced more defecation, but this "response" did not compete with any other tested response. Since the competing-response idea involved conditioning the effects of ECS to task and situational cues, the evidence that these cues did not alter ECS effects was critical. Even when concerted attempts were made to condition competing-response tendencies, none was observed although some generalized immobility

developed after several convulsions (Gerbrandt & Thompson, 1964).

These developments left open the question of aversion. Because Jarvik's one-trial, step-down procedure (Jarvik & Essman, 1960) pitted any aversion induced by the convulsion against any amnesia, there was a tendency for investigators to believe the question was either amnesia or aversion, and to portray their results accordingly. This question began to be reformulated by the results of an experiment in which animals received either a convulsion alone, or gridshock followed by a convulsion, after stepping down (Hudspeth, McGaugh & Thompson, 1964). After 8 daily trials, it was found that with convulsions alone (no gridshock), the sooner after stepping down the convulsion occurred the more the animals tended to stay on the platform. But with both gridshock and a convulsion, the sooner after stepping down the convulsion occurred, the less the animals tended to stay on the platform. That is to say, the series of convulsions alone produced freezing--presumably conditioned freezing--in accordance with the general rule that the sooner a reinforcer follows a response, the faster the learning. On the other hand, the convulsion attenuated the effect of the gridshock in accordance with the expectation from the general rule of retrograde amnesia that the sooner the amnesic event follows learning, the slower the learning. Thus

Hudspeth and colleagues (1964) concluded that a series of convulsions can have both aversive and amnesic effects.

This conclusion was strengthened by the results of an experiment in which reversal was required in a T-maze to obtain water. Entering the originally-reinforced arm resulted in a convulsion for one group and a subconvulsive shock for another, and it was found that the convulsed animals learned the reversal more slowly (McGaugh & Madsen, 1964). The authors reasoned that the convulsion induced amnesia, as well as aversion, and thus produced slower learning than the subconvulsive shock.

By this time, then, the conventional wisdom was that the major effect of a single convulsion was retrograde amnesia, that aversion or fear could be induced by convulsions but this developed slowly, over several trials, and that any competing responses conditioned by ECS occurred only after several trials and probably only under special circumstances as well.

The idea that the amnesic and aversive effects of ECS were separable, because amnesia could be produced in one trial while fear was produced after several trials, received more specific support later (Gerbrandt, 1965; Chorover & Schiller, 1965, p. 76), and the idea became widely accepted. This idea resolved many of the apparent conflicts associated with experiments on ECS. This idea, for

instance, made it possible to accept both Duncan's (1949) conclusion and Coons and Miller's (1960) conclusion: since in Duncan's experiment aversive and amnesic properties would both slow the rate of learning, his results probably represented the summation of the two effects; since many convulsions were involved in Coons and Miller's experiment, and since the aversive and amnesic effects would work against one another, their results probably represent the predominance of aversion. By the same reasoning, where only a single convulsion was given, one would expect the amnesia to predominate, thus producing the results of Madsen & McGaugh (1961) and Thompson and Dean (1955).

The idea that ECS produced amnesia in one trial and fear only after several trials did not, however, resolve the opposite empirical results obtained by Coons and Miller (1960) and by Hudspeth and colleagues (1964) where both used multi-trial, passive-avoidance tasks involving several convulsions. Coons and Miller (1960) reported that, after grid shock, the sooner the convulsion the faster the passive-avoidance learning, whereas Hudspeth and colleagues (1964) reported that, after grid shock, the sooner the convulsion the smaller the percentage of passive-avoidance responses. This is to say, although Hudspeth and colleagues demonstrated an aversive effect of convulsions, when they pitted the aversive and amnesic properties against one

another, they found the amnesic effect predominant. Since the two groups of investigators used different techniques and different measurements of learning, their results may not be exactly opposite. Moreover, the factors affecting the relative degree of aversion and amnesia are not known, and, with several convulsions, the "crossover point" cannot be predicted. The aversive effect, for instance, cannot logically involve the ECS itself if it is forgotten. But as Coons and Miller (1960) pointed out, human patients often develop marked fear of, and aversion to, ECT because of the intense disorientation experienced when recovering consciousness afterwards (Gallinek, 1955). If this mechanism also applies to animal experimentation, it becomes extremely difficult to estimate such factors as the apparent delay of reinforcement.

Despite this difficulty, the idea that the amnesic effect of a single convulsion is stronger than the aversive effect continued and continues to be widely held, so that the major focus of attention shifted to the parameters of the ECS-induced amnesia. Before pursuing that development, however, it is appropriate to raise another question in order to complete the outline of the major ideas about the behavioral effects of ECS.

The Question of Incubation

Conditioned fear-motivated responses seem to change spontaneously in strength over time following training. This has been demonstrated in situations involving humans (Bindra & Cameron, 1953) and in animal learning involving either CER (McMichael, 1966) or active avoidance (Kamin, 1963). Such changes also occur after the training of a one-trial, passive-avoidance response: sometimes these changes have been monotonic increases in strength (McGaugh, 1966; Pinel & Cooper, 1966b) and sometimes biphasic changes (Irwin & Banuazizi, 1966; Pinel & Cooper, 1966a). The monotonic increase in strength of a fear-motivated response over time following training is often called the "incubation of fear" and it has been suggested (Pinel & Cooper, 1966c) that ECS produces an apparent retrograde amnesia by somehow attenuating this incubation process.

The major experiment supporting the incubation idea (Pinel & Cooper, 1966c) involved two groups which were first trained to drink and were then shocked for drinking. One group was divided into 3 subgroups which received a single convulsion 10 sec., 2 min. or 5 hr. after being trained to passively avoid drinking. These animals were then tested 25 hr. after passive-avoidance training. The other group was divided into 3 groups which were simply tested 10 sec., 2 min. or 5 hr. after passive- avoidance training. Giving

a convulsion at these three intervals and testing 25 hr. after training produced about the same respective effect as simply testing the animals at the three intervals. That is, testing animals 10 sec. after passive-avoidance training produced about the same latencies as convulsing animals 10 sec. after training and testing them 25 hr. after training. An equivalent effect was similarly obtained with the 2-min. ECS-delay interval and the 2-min. training-testing interval, and the same was true for the 5-hr. intervals. Over the three intervals used, the mean latencies increased monotonically as the ECS-delay interval or training-testing interval increased. Therefore, the investigators concluded "that the ECS gradient effect is attributable to the incubation or increase in strength of the learned response" over time. McGaugh (1966) also reached a similar conclusion.

There are, however, some difficulties with this conclusion because the latencies reported by Pinel and Cooper (1966a, 1966b, 1966c), no matter what the treatment, are over 60 sec. This is longer than most other reported latencies since most other investigations involving latency measures have had an arbitrary maximum latency of 30 sec. One can naturally argue that the other investigators have produced results unique to this maximum latency but this does not alter the fact that these results cannot be compared with Pinel and Cooper's results. Thus, Pinel and

Cooper probably demonstrated a real phenomenon, and one which may be theoretically important. The phenomenon is, however, unrelated to the typical demonstrations of amnesia at shorter latencies and therefore it cannot be invoked to explain the more common results.

Similarly, Suboski, Spevack, Litner and Beaumaster (1969) reported results which supported the notion that a convulsion may produce an incubation-type effect. But, as the authors pointed out, these results cannot "explain" other reports of retrograde amnesia since the minimum ECS-delay interval used was 100 sec. while retrograde amnesia apparently occurs in many situations only with ECS-delay intervals of 30 sec. (Quartermain and colleagues, 1965) or even less (Chorover & Schiller, 1965).

Despite this, the phenomenon demonstrated by Pinel and Cooper (1966c) is most striking and cannot merely be discarded as irrelevant. Further experiments on incubation involving procedures more like those used to demonstrate retrograde amnesia may produce very useful results. One possibility is that freezing, or inhibition or movement, develops spontaneously over time, rather than fear. For example, when animals received a single shock-escape trial in a two-compartment box and were returned to the previously-shocked compartment 5 sec., 1 min. or 5 hr. later, latencies for leaving the compartment <u>increased</u> over the in-

tervals tested (Pinel, 1968). No shock was applied when the animals were returned to the apparatus after the escape trial but, according to the incubation notion, the latencies should have decreased as the training-testing interval increased since the increasing fear would impel the animal to leave the compartment faster and faster. But if movement-inhibition were developing, then the increasing latencies Pinel (1968) observed were naturally to be expected. The possibility that movement-inhibition, rather than fear, develops spontaneously over time will assume importance in the light of the discussion at the end of the present document. This possibility cannot easily be tested in a passive-avoidance situation, since the increasing latencies seen there (Pinel and Cooper, 1966c; Suboski and colleagues, 1968) could have occurred as a result of either incubation of fear or development of freezing.

The question of incubation, unlike the questions of amnesia, aversion and competing responses, has not been incorporated into the "mainstream" of thought about the effects of convulsions because the work is still relatively new. But incubation-type effects may prove very useful to a more complete explanation of the effects of electroconvulsive shock.

In the meantime, three issues have been raised about the retrograde amnesia induced by a convulsion. These

issues are (1) the length of the so-called consolidation period, (2) the possibility of "recovery" from ECS-induced amnesia, and (3) the apparently different effects of convulsions upon tasks requiring immobility and tasks requiring movement. Since these issues are related to the more general question of retrograde amnesia, the experiments cited involve one-trial, passive-avoidance tasks unless specified otherwise.

How long is the Consolidation Period?

Even casual perusal of the published experiments on ECS-induced amnesia reveals that the quantitative results vary considerably even though the amnesic phenomena may be generally similar. There are particularly large differences among the reported training-ECS, or ECS-delay, intervals at which an amnesic effect is obtained. Since it has become customary to estimate the length of the consolidation period as the longest amnesia-producing ECS-delay interval, these differences present serious difficulties of interpretation. Reports of the longest amnesia-producing ECS-delay intervals range from a few seconds (Quartermain and colleagues, 1965; Chorover & Schiller, 1965) through many minutes (Thompson & Dean, 1955; Heriot & Coleman, 1962), and up to several hours (Kopp, Bohdanecky & Jarvik, 1966).

For the most part, these quantitative discrepancies have been overlooked in the heat of the more exciting controversies outlined earlier. In a series of experiments, however, Chorover and Schiller (1965, 1966) addressed themselves to the reported discrepancies in estimates of the consolidation period. Since this line of investigation has had a profound effect on ECS experimentation and interpretation and, since it is germane to the experimental reports following, it is worthwhile to consider the experiments and conclusions of Chorover and Schiller in some detail.

Using a Jarvik or step-down passive-avoidance task for three daily trials, Chorover and Schiller (1965) reported three major findings. First, if a convulsion occurred 10 sec. after the first trial, passive-avoidance performance in the second trial was impaired but if the convulsion occurred 30 sec. after the first trial, passive-avoidance performance in the second trial was not significantly different than if no convulsion was induced on the first trial. Thus, they concluded that a convulsion more than 10 sec. after a training trial did not produce retrograde amnesia. Second, some animals received only a convulsion and no grid shock when they stepped off the platform. With this treatment, step-down latencies were the same in the second trial (after one convulsion) as in the first trial (before any convulsions) but, after three trials, the step-down laten-

cies were longer than before treatment. In other words, three convulsions alone, without other passive-avoidance training, produced apparent passive avoidance while a single convulsion did not have this effect. Third, for the animals which received only convulsions (no grid shock), the step-down latencies in the third trial formed an inverted-U-shaped function with respect to the ECS-delay interval. If convulsions were induced 0, 5 or 10 sec. after stepping down, there were no statistically significant differences in latencies on the third trial whether grid shock was also given or not; with an ECS-delay interval of 30 sec., however, grid shock followed by a convulsion produced much longer latencies in the third trial than convulsions alone.

To account for these results, Chorover and Schiller (1965) suggested that (1) a single convulsion produced a true retrograde amnesia only for events up to about 10 sec. beforehand, and (2) convulsions had aversive effects which gradually became conditioned to the testing situation with the usual delay-of-punishment gradient (Kamin, 1959), which is, the sooner the punishment the greater its effect. However, with convulsions as the punishment, the empirically obtained gradient assumed an inverted-U shape with respect to the ECS-delay interval because the memory for the testing situation, and hence the strength of conditioning, improved as the ECS-delay interval was lengthened, up to about

10 sec. The punishment gradient (inversely proportional to the ECS-delay interval) and the memory gradient (directly proportional to the ECS-delay interval) combined to produce an inverted-U gradient. Chorover and Schiller's (1965) suggestions became widely respected but, as the authors noted, their explanation did not adequately explain why other investigators had produced impaired retention on similar tasks with much greater ECS-delay intervals.

Chorover and Schiller (1966) subsequently made an attempt to reconcile their finding that a convulsion exerts an amnesic effect only if it occurs within a few seconds after training with other results which showed "prolonged retrograde amnesia." They suggested that convulsions produced amnesia for discriminated tasks only if it occurred shortly after training but that a convulsion also reduced generalized inhibition or suppression of movement. That is to say, a convulsion produces true amnesia only for a period of a few seconds beforehand and any effect upon a longer period beforehand is really the disruption of generalized suppression of movement. This idea is, of course, the logical culmination of using the longest effective ECS-delay interval as the estimate for the consolidation period so that Chorover and Schiller (1965, 1966) essentially estimate the consolidation period at about 10 sec.

One way to test this idea is to see if an ECS-in-

duced response decrement is dependent upon the ECS-delay interval or not. If there are two types of tasks, discriminated and non-discriminated, and if ECS induces amnesic effects only upon discriminated tasks, then it follows that discriminated tasks will be those which show decreasing "graded" decrements as the ECS-delay interval increases. Effects of ECS which do not change as the ECS-delay interval changes will be non-amnesic effects, and the task will involve non-discriminated learning.

Although the reasoning involves some circularity, it has had a great heuristic effect. Chorover and Schiller (1966) reported that, in a two-compartment apparatus designed by Bures and Buresova (1963), the amount of time an animal spent in the compartment where it previously received gridshock remained the same whether ECS occurred 1 min. or 1 hr. after gridshock--although this time was still more than if no ECS was given. Thus, they concluded that the ECS induced a reduction in generalized inhibition of movement, not an amnesia, and that the task did not involve discriminated learning. In the same type of experiment, however, other investigators have reported that ECS given about 1 min. after training produced effectively shorter latencies than ECS given about 1 hr. after training (Spevack, Rabedeau & Spevack, 1967). In this task, short latencies mean impairment in passive-avoidance performance. There-

fore, Chorover and Schiller (1966) refer to the ECS effect as ungraded and conclude that their results show a non-amnesic effect while Spevack and colleagues (1967) refer to the ECS effect as graded and conclude that their results show retrograde amnesia.

This particular discrepancy must be considered unresolved since Chorover and Schiller (1966) measured elapsed time in the previously-charged compartment while Spevack and colleagues (1967) measured latency of entrance. Moreover, different statistical techniques were used and neither group investigated the effects at ECS-day intervals between 1 min. and 1 hr.

Kopp and colleagues (1966) designed an experiment specifically to test Chorover and Schiller's (1966) ideas. They used a task requiring the passive-avoidance of one compartment in a two-compartment apparatus, and they showed separately that this task involved discriminative learning. A single convulsion after the passive-avoidance training produced a gradient of latencies related to the ECS-delay interval, and significantly shorter latencies were found even when the convulsion occurred as long as 6 hr. after training. Thus, they concluded that Chorover and Schiller's (1966) proposal was not experimentally supported.

Just the same, with a similar apparatus, Chorover and Schiller's (1966) interpretation was supported in prin-

ciple, although not in detail, by the results of another experiment (Suboski, Black, Litner, Greener & Spevack, 1968). With a discriminative passive-avoidance task, temporally graded effects were found from a convulsion occurring up to 100 sec. after training, but not beyond. This result is, of course, much closer to Chorover and Schiller's (1965) results than to Kopp and colleagues' (1966) results. But the absolute differences in the maximum effective ECS-delay intervals found by Chorover and Schiller (1965) and Suboski and colleagues is still considerable.

Like Kopp and colleagues, Schneider and Sherman (1968) induced retrograde amnesia with an ECS-delay interval of 6 hr., but they used a very different procedure. In a step-down task, rats received footshock when they stepped down and they were then returned to the home cage for 6 hr. Then they were placed on the grid floor of the apparatus, received another foot shock, and ECS was delivered 0.5 sec. later. When the animals were tested 24 hr. after the passive-avoidance training, they had very short step-down latencies, indicating amnesia for the footshock. By way of control, ECS delivered 6 hr. after passive-avoidance training (that is, without the second footshock) did not produce retrograde amnesia.

There are certain problems with these results, however. Schneider and Sherman (1968) used footshock and ECS

parameters similar to those used by Chorover and Schiller (1965). But when animals in Schneider and Sherman's experiment received a footshock and no ECS, their latencies 24 hr. later were shorter than those of the animals similarly treated in Chorover and Schiller's experiment. In Chorover and Schiller's (1965) experiment, this procedure produced a maximum median latency (30 sec.) while in Schneider and Sherman's (1968) experiment, the reported mean latency was only 12.5 sec.

In any case, it seems most likely that Schneider and Sherman's findings were a special case, resulting from a "two shock" procedure, and not directly related to the length of the consolidation period in the simpler situations which have been used more frequently. With a somewhat similar procedure, for example, it was reported that a single convulsion could produce retrograde amnesia even if it occurred the day after learning (Misanin, Miller and Lewis, 1968). In this latter case, the trained response was the suppression of drinking and training involved a specific conditioned stimulus. The day after training, animals were presented again with the conditioned stimulus and some were convulsed immediately afterwards. When tested the following day (two days after training), the animals which had been convulsed after re-presentation of the conditioned stimulus drank faster than the animals which had not been convulsed after

the re-presentation. Although these results are very curious, it is difficult to relate them systematically to more conventional results pertinent to estimating the length of the consolidation period. One possibility is that foot-shock (Schneider & Sherman, 1968), or even a stimulus conditioned to foot shock (Misanin and colleagues, 1968), sensitizes the nervous system to electroconvulsive shock and thus increases the likelihood of retrograde amnesia.

Another attempt to resolve the question of the length of the consolidation period was made by Cherkin (1966), who reanalyzed the published data from the experiments of Chorover and Schiller (1965), Quartermain and colleagues (1965), King (1965) and Weissman (1964). Using probit analysis, Cherkin (1966) concluded that the slopes of the data he analyzed were similar but that the strength of the response at the start of testing differed among the experiments. That is to say, applying the formula, $y = a + b \log t$, to the post-ECS learning curves, he suggested that the major difference among the four experiments was in the parameter, a. Therefore, he concluded, a similar consolidation process was involved in these four cases but it was superimposed upon different strengths of original learning.

However, the strength of original learning is necessarily counfounded with the amount of experience in the apparatus before the convulsion, and it has also been re-

ported that simple exploratory experience in the apparatus determined whether a convulsion induced retrograde amnesia (Lewis, Miller and Misanin, 1968). Perhaps, then, familiarity with the apparatus rather than particular training is a determinant of the length of the consolidation period. Using a step-down task, Lewis and colleagues (1968) found that a convulsion immediately after passive-avoidance training induced retrograde amnesia only in animals which were unfamiliar with the apparatus. The passive-avoidance performance of animals which had explored the apparatus before training was relatively unaffected by a convulsion immediately after training.

Thus, the question of the length of the consolidation period has been reformulated in part into a number of subquestions about discrimination, familiarity or prior training, and so forth. This may indicate that there is no absolute consolidation period or, at least, one that can be inferred from behavioral results, because of other complications arising from training or testing conditions, or from other effects of a convulsion. Another of these complications is the possibility that animals "recover" from retrograde amnesia induced by convulsions.

Is there Recovery from Retrograde Amnesia?

If the retrograde amnesia induced by a convulsion

spontaneously decreases over time, this suggests that the effect of convulsions is upon "retrieval" rather than "storage" of memory. This is obviously a crucial question and one recognized many years ago (Ribot, 1882). Largely because of physiological conceptions (Hebb, 1949), most authors have assumed that the amnesia involved a storage deficit rather than a retrieval deficit although this assumption is not required by the behavioral data. There is a strong impression that the retrograde amnesia seen clinically spontaneously decreases over time (Williams, 1966)--it is usually considered "shrinking"--but this impression if often attributed to decreasing disorientation and confusion. Recovery from the effects of convulsions has also been seen in animals: conditioned suppression which disappeared after 21 convulsions was present again when tested 30 days later (Brady, 1951). In this case, however, it was not believed that amnesia was involved.

The first claim that animals recovered from amnesia induced by a convulsion was made by Cooper and Koppenaal (1964) when they reported that, after animals received two shocks for drinking shortly followed by a convulsion, drinking was greater 1 hr. after the convulsion than 24 hr. after the convulsion. This result was interpreted as "suppression and recovery of a ... (passive) avoidance response after a single ECS."

Similarly, when ECS was given to mice within 10 sec. after shock-induced suppression of drinking, decreasing durations of drinking were found in independent groups tested 3, 24 and 48 hr. afterwards (Kohlenberg & Trabasso, 1968).

However, when shock was used to suppress movement (Chevalier, 1965) or induce passive avoidance of moving through a hole into a dark box (Luttges & McGaugh, 1967; Greenough, Schwitzgebel & Fulcher, 1968), no recovery was found from the retrograde amnesia induced by a post-training convulsion. In these experiments, independent groups were tested 1, 7 and 30 days afterwards (Chevalier, 1965) or 12 hr., 7 days and 32 days afterwards (Luttges & McGaugh, 1967), or 2, 2.5, 3, 24 and 72 hr. afterwards (Greenough and colleagues, 1968). In the latter two experiments, no recovery from the ECS-induced retrograde amnesia was observed in repeated tests either.

The reason why there is concern whether independent groups or groups tested repeatedly were used relates to a finding of recovery by Zinkin and Miller (1967). In a step-down, passive-avoidance task, these investigators found recovery from ECS-induced amnesia when they tested the same animals daily over a 3-day period. The experiment was criticized by Herz and Peeke (1967), however, because they thought that the experience involved in the re-exposure to the test situation may have been a factor in the recovery. In their

own experiments on mice, for example, Herz and Peeke (1967, 1968) reported no recovery from ECS-induced retrograde amnesia for passive avoidance of drinking if independent groups were tested 24 hr. and 72 hr. after training. However, "recovery" from the amnesia seen 24 hr. after training occurred if the same animals were retested 48 hr. and 72 hr. after training. Therefore, Herz and Peeke (1967, 1968) concluded, the amnesia is permanent and so-called recovery requires extinction, habituation or some other learning involved in the re-exposure to the test situation. However, Zinkin and Miller's results were essentially confirmed in an experiment in which independent groups of rats were tested 24 hr. or 96 hr. after step-down passive-avoidance training immediately followed by a convulsion (Nielson, 1968). In this case, recovery occurred only if the animals were tested with the earclips (through which the convulsion has been induced) attached; Zinkin and Miller (1967) also attached the earclips when they tested their animals.

It is possible that the earclips somehow served as a "reminder" of the passive-avoidance training. Certain aids help in clinical cases of retrograde amnesia (p. 10) and, in an animal experiment, a "reminder" footshock which was itself too weak to train passive avoidance induced dramatic recovery from retrograde amnesia for passive avoidance of drinking (Koppenaal, Jagoda & Cruce, 1967). In

this experiment, two groups of animals which had been pre-trained to drink in an apparatus were then shocked once for drinking in the apparatus and, 5 to 10 sec. later, were convulsed. Latencies of drinking were tested 24 hr. later but, 40 min. before testing, one of these groups received a weak footshock in the apparatus. The animals which had received the "reminder" footshock had much longer latencies than the animals which had not received this treatment. Since retrograde amnesia in this case was indicated by short latencies, the "reminder" produced apparent recovery from retrograde amnesia, and the authors concluded that a convulsion may inhibit or suppress the expression of a fully formed memory but does not make memory formation impossible.

Schneider and Sherman (1968) also observed recovery with a "two shock" procedure. Animals received shock when they stepped off a platform and then received another shock 27.5 sec. later. A single convulsion 0.5 sec. after the second shock produced the short latencies indicating retrograde amnesia in a test 24 hr. later. Retest 48 hr. after the passive-avoidance training showed the increased latencies indicating recovery from the amnesia. Unlike Zinkin and Miller (1967), these investigators observed no recovery, however, if animals received only a single shock followed by a convulsion 0.5 sec. later. Tests after 24 hr. and 48 hr. both showed short latencies, even though

the same groups were tested twice in Schneider and Sherman's experiment.

Thus, there appears to be recovery from amnesia. Whether this phenomenon is due to recovery of memory or to something else affecting performance is unclear. Unfortunately, something of a controversy seems to be deveoping over the existence of recovery or recovery-like phenomena. It is likely that a real effect has occurred in the reports of recovery, which warrants non-partisan investigation. For instance, Riddell (1969) was also able to replicate Zinkin and Miller's (1967) observations of recovery, although he found a weaker effect, which later disappeared itself. Moreover, Riddell (1969) reported that the recovery was not seen if animals were tested 24 hr. and 72 hr. after training, omitting the 48-hr. test interval. Nor was recovery observed if animals were tested every 6 hr. after training for the first 24 hr. and then tested at four more 24-hr. intervals. In these latter procedures, short latencies were seen continuously, as a result of a 0.5 sec. ECS-delay interval after a single passive-avoidance trial.

Riddell (1969) suggested that the convulsed animals actually showed fairly constant latencies, but the non-convulsed animals had gradually decreasing latencies over certain retest intervals. Thus, comparisons of performance between control and convulsed animals allow a recovery-from-

amnesia interpretation. In Zinkin and Miller's (1967) report, for instance, the latencies of animals which had a convulsion, but no passive-avoidance training, also increased spontaneously over the 3-day test period. In two other reports of recovery from amnesia the recovery was inferred from comparisons with a first test administered 3 hr. (Kohlenberg & Trabasso, 1968) or 1 hr. (Cooper & Koppenaal, 1964) after passive-avoidance training. According to Greenough and colleagues (1968), mice show increased latencies (that is, no apparent amnesia) following either a convulsion alone or passive-avoidance training followed by a convulsion, if they are tested within 3 hr.

These results are not easy to integrate systematically but, in general, they suggest that a variety of responses, involving immobility or movement, develop in shock-avoidance procedures and that some understanding of this "base-line" performance is probably necessary in order to interpret the reports of recovery from amnesia. Recovery from amnesia, or some similar spontaneous effect, seems clearly to occur over time under some conditions. These conditions are not well understood now, but they probably pertain to the sorts of responses which develop over time following aversive training and are, therefore, related to the question of incubation.

Does ECS induce Amnesia for Active Tasks?

The third question about ECS-induced retrograde amnesia relates to the fact that, except for the early reports (Duncan, 1949; Thompson & Dean, 1955), most of the experiments addressed to the question of amnesia have involved a passive-avoidance task. This situation has occurred because the passive-avoidance response can easily be trained in a single trial, and the time of learning can be specified. Therefore, the "time since learning" can be determined, and quantitative estimates of the consolidation period can supposedly be made. In order for the amnesia interpretation to have any generality, however, it is crucial to show that a convulsion induces amnesia for tasks requiring movement, as well as tasks requiring immobility. Furthermore, tasks requiring movement involve somewhat different neurological systems than tasks requiring immobility. (McCleary, 1966), so that a phenomenon which applies only to passive tasks requires different neurological conceptualization than one which applies equally to both active and passive tasks.

Convulsions seem to have a weaker, or less clearly seen, amnesic effect upon active tasks than upon passive tasks, although there are very few reports on active tasks which appear analogous to the one-trial passive tasks. In a shock-motivated T-maze, a single convulsion did not induce

retrograde amnesia if it occurred either 20 sec. or 3 min. after the end of four training trials (Gerbrandt, Buresova & Bures, 1968) and, with a similar task, a single convulsion 30 sec. after training to a 90% criterion induced retrograde amnesia for a brightness-discrimination habit, but not for a spatial-discrimination habit (Corson, 1965). Thus, ECS seemed to induce amnesia for visually-guided tasks requiring movement but not for spatially-guided tasks requiring movement. The shortest ECS-delay interval in the experiments involving spatial tasks, however, was 20 sec. and this is longer than the longest amnesia-producing interval for certain passive-avoidance tasks (Chorover & Schiller, 1965). Moreover, while only a single ECS was used, several training trials were used in these experiments and Corson (1965) reported that there was no amnesia for brightness-discrimination training if the initial training trials involved a pattern discrimination, rather than a brightness discrimination.

Attempts have also been made to induce amnesia for appetitive tasks with convulsions. A convulsion induced amnesia for learning in the Hebb-Williams maze, but the degree of amnesia was much less severe than that produced when a convulsion followed training in a passive-avoidance task (Schiller & Chorover, 1967). It had been reported earlier that ECS induced amnesia for learning to drink in

a box, but this amnesic effect was shown only by a decreased number of entries into the area where water was available (Tenen, 1965). ECS produced no amnesic effect as measured by latency of entrance into the area or by the amount of time spent in the area.

Thus, ECS may induce some degree of retrograde amnesia for tasks requiring active movement but the amnesic phenomenon does not appear as clearly as it does in tasks requiring immobility. Although none of the experimental situations requiring movement have been very similar to the situations in which passive-avoidance training was used, there seems to be a difference between the effect of convulsions upon the two types of task, in terms of the apparent degree of retrograde amnesia induced.

An attempt to resolve the different effects of ECS upon passive tasks and tasks requiring movement necessarily raises the question of ECS effects upon movement itself. This is a question to which few previous investigators have specifically addressed themselves, but one which may be crucial in understanding the effects of ECS upon learning and memory.

The Question of Movement

Apart from the retrograde effects of convulsions outlined so far, a series of convulsions given beforehand

impairs the acquisition of a passive-avoidance response (Poschel, 1957; Delprato, 1966). A series of convulsions also facilitates the acquisition of an active-avoidance response requiring either moving back and forth in a shuttle box (Vanderwolf, 1963b), or reversal in a T-maze (Casseday, 1966). Since these results arise from anterograde effects of the convulsions, they cannot be explained as amnesia; since the results are opposite for active and passive tasks, they cannot be explained as general learning or motivational deficits. Since a series of convulsions usually decreases spontaneous activity (Munn, 1950, p. 443), it is unlikely that these results occurred because the convulsions simply increased the tendency to move. The results can be explained, however, on the basis that convulsions reduce the tendency to freeze or inhibit movement in frightening situations.

There have also been inferences that a single convulsion increases movement (Routtenberg & Kay, 1965) or reduces fear-induced inhibition of movement (Chorover & Schiller, 1966). In the context of retrograde amnesia experiments, the important point is whether a single convulsion effectively increases movement, or reduces freezing, because the impressive evidence that ECS induces retrograde amnesia derives from the results of only a single convulsion. The only systematic investigation of this point

was reported by Kopp, Bohdanecky and Jarvik (1967). These investigators reported that a single convulsion decreased spontaneous latencies in a two-compartment apparatus, but only if the convulsion was induced no longer than 1 hr. before testing. In the same apparatus, Kopp and colleagues (1967) also gave a single passive-avoidance trial first, and then induced a single convulsion at various intervals before the testing 24 hr. after training. The convulsion produced shorter latencies only if it occurred 8 hr. or less before testing.

From these results, Kopp and colleagues (1967) concluded that a 24-hr. training-testing interval successfully separated the prograde effects of a single convulsion from the retrograde effects. Nonetheless, it might prove useful if more information pertinent to this conclusion was obtained because, if a single convulsion can affect movement tendencies up to 24 hr. afterwards, this has serious implications for results purported to demonstrate retrograde amnesia. In particular, such information might resolve the differences between the amnesic effects for tasks requiring immobility and tasks requiring movement.

III. STATEMENT OF PURPOSE

The experimental results discussed this far may be summarized as follows. Convulsions induce some retrograde amnesia, which is shown clearly in one-trial, passive-avoidance situations, but convulsions also have other effects which are powerful enough to seriously complicate attempts to investigate the hypothetical consolidation process. One particular problem is that convulsions may exert a stronger, more clearly seen, amnesia effect upon tasks which require movement inhibition than upon tasks which require active movement.

This difference in effect between active and passive tasks suggests that a single convulsion may affect the tendency to make movements itself. If a single convulsion exerts a powerful effect upon the tendency to move or freeze, this may make it impossible to draw quantitative conclusions about the consolidation process from behavioral results alone because such results invariably involve testing an animal's movements.

Since amnesic interpretations of ECS effects on behaviour, such as quantitative estimates of the consolidation period, have implications for the physiology of

memory, understanding these "complications" is of some importance. Therefore, the purpose of the present investigation was to study certain effects of convulsions, especially those effects which may seriously affect conclusions about amnesia or consolidation. A major part of the investigation is more specifically addressed to the effect of convulsions upon the tendency to inhibit movements.

EXPERIMENTAL METHODS AND RESULTS

Subjects

The subjects of these experiments were 915 naive male hooded rats which weighed from 200 to 300 gm. at the start of the experiments. They were housed 5 or 6 to a cage during the experiments. Food and water were continuously available to them in the home cages except that no water was available in the home cages of animals being trained to drink in an experimental apparatus.

Procedure for Producing Convulsions

Convulsions were produced with a device made by connecting small alligator clips through light wire leads across the secondary coil of a 1500-v. transformer. A Decade Interval Timer (Hunter, Model 111-B, Series D) in the primary circuit of the transformer limited the flow of the current to selected durations. Cotton packs were tied to the alligator clips with suture thread, and the cotton-packed clips were dipped in saline prior to producing each convulsion in order to assure good electrical contact. To produce convulsions, the clips were attached to the animals' pinnae; for pseudo-convulsions the clips were

also attached to the pinnae but the transformer was disconnected.* Unless specified otherwise, convulsions were produced with an ECS of 0.3 sec. duration.

If convulsions were produced 10 sec. or less after learning, the earclips were attached just prior to training and kept on during training; otherwise the earclips were attached after training or at the appropriate time before training. Since Weissman (1963) found that convulsions produced retrograde amnesia most clearly in animals which showed complete tonic extension, any animal which showed incomplete tonic extension during the seizure was discarded. Animals which suffered spinal injuries from the convulsion were also discarded. The numbers of animals discarded from each experiment are shown in Table 13.

Statistical Analysis

Statistical comparisons between independent groups were made with the Mann-Whitney U test and comparisons between two tests of the same group (paired comparisons) were made with the Wilcoxon matched-pairs signed-ranks test (Siegel, 1956). In both cases, levels of statistical

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*It is said procedurally, for instance, that an animals "received a pseudo-convulsion 30 sec. after training." This means that the animal was treated just as if a convulsion were to be induced and the earclips were removed about 30 sec. after training.

significance cited in this document are for two-tailed tests. Comparisons among several groups of animals were made with the Kruskal-Wallis one-way analysis of variance (Siegel, 1956). The individual results used for these analyses are given in the appendices at the end of the thesis.

I. ACTIVE-AVOIDANCE EXPERIMENTS

Apparatus. The avoidance apparatus was a plywood box, 36 in. long by 10 in. wide and 18 in. high. A sliding door located half way along the box divided it into two equal-sized compartments. One comparment was painted white and the other compartment was painted black. The floor of the box was a grid of 1/8-in. stainless steel rods set about 1/2 in. apart. The grid floor of the black compartment could be charged by the secondary coil of an 1100-v. transformer operating through a variable series resistance. This resistance was set so that, with an animal on the grid, the current was about 1.0 mA.

Avoidance Procedure. The avoidance response required an animal to move from the black compartment to the white compartment within 5 sec. The animal was placed on the grid floor of the black compartment, facing away from the white compartment and, 5 sec. later, continuous electric shock was delivered to the grid floor. After an intertrial interval of 20 sec., the animal was replaced at the starting point and another trial was administered. This procedure was continued for 25 trials or until the animal made 9 avoidance responses in 10 consecutive trials,

whichever occurred first (90% criterion). For the first 3 trials, the sliding door was closed when the animal entered the white compartment; on the later trials, this door remained open. The number of shocks received (i.e., escape responses) in reaching the 90% criterion was recorded for each animal. This procedure was used throughout the avoidance experiments.

Part 1. Facilitation of avoidance learning.

As pointed out earlier (p. 49), there is evidence that a series of convulsions reduces the tendency to freeze in frightening situations (Pöschel, 1957; Delprato, 1966; Vanderwolf, 1963; Casseday, 1966). There is, however, relatively little information on similar effects from a single convulsion (Kopp and colleagues, 1967). Since the impressive evidence demonstrating retrograde amnesia comes from the results of only a single convulsion, it is important to know if a single convulsion can also reduce the tendency to freeze. Such knowledge would have obvious implications for interpreting results purported to show retrograde amnesia produced by a single convulsion.

One criterion used by previous investigators was that, if convulsions reduce the tendency to inhibit movement in frightening situations, they should produce faster acquisition of a fear-motivated response which requires

active movement. In the present instance, an attempt was made to see if a single convulsion would facilitate acquisition of a one-way active-avoidance response.

Vanderwolf (1963b) indicated that a series of convulsions did not facilitate one-way avoidance learning. However, the animals in that experiment were placed in the apparatus for 5 min. before training and allowed to explore the uncharged apparatus. Such an opportunity to explore can itself improve active-avoidance performance in rats with medial-thalamic damage, and it was suggested that this effect occurred because the exploratory period also reduced the tendency to freeze in frightening situations (Vanderwolf, 1966). Since an opportunity to explore for a few minutes beforehand is a common feature of aversive training, it seemed worthwhile to study the effects of this treatment, as well as the effect of a convulsion.

Accordingly, in the present experiment, animals received one-way, active-avoidance training after a single convulsion, or after a 5-min. exploratory period, or after both or after neither.

Procedure. Four groups of animals (N=56) received avoidance training, and each group was treated differently before training. The animals in the Convulsion/Exploration Group received a single ECS, 24 hr. before training, and

were placed in the uncharged avoidance apparatus for 5 min. just before training. The Convulsion/No Exploration Group also received a single ECS 24 hr. before training but were not allowed to explore the apparatus before training. The No Convulsion/Exploration Group received a pseudo-convulsion 24 hr. before training, and the 5-min. exploratory period just before training. The No Convulsion/No Exploration Group received a pseudo-convulsion 24 hr. before training and no exploratory period before training.

Results. The mean numbers of shocks received by the 4 groups in reaching the avoidance criterion are shown in Table 1*. It can be seen from Table 1 that the No Convulsion/No Exploration Group took more shocks in acquiring the active-avoidance response than any of the other groups, and that there were no statistically significant differences in rate of acquisition among these other three groups.

Thus, either an opportunity to explore the apparatus for 5 min. before training, or a single convulsion 24 hr. before training, facilitated one-way active-avoidance learning by about the same amount. Combining the convulsion and the exploratory period did not produce signifi-

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*Individual results of the experiment are shown in Appendix A.

Table 1

Mean number of shocks received in reaching 90% criterion in the active-avoidance task after a convulsion or exploratory period.

		CONVULSION	NO CONVULSION
EXPLORATION	n	11	15
		5.0*	5.1*
NO EXPLORATION	n	16	14
		5.3*	8.9

Analysis of Variance: $H = 20.4$, $df = 3$, $p < .001$.

* less than the No Convulsion/No Exploration Group ($p < .02$).

cantly faster learning than either treatment alone.

Part 2. Retrograde effects of a convulsion.

Despite the finding in Part 1 that a single convulsion can facilitate avoidance learning, there are several reasons for predicting that a single convulsion will also induce retrograde amnesia for the avoidance task. First, several convulsions reportedly induce amnesia for the task (Duncan, 1949). Second, a single convulsion given after the 10 or 15 min. which it takes to learn the task can induce retrograde amnesia (Thompson, 1955; Heriot and Coleman, 1962). Third, it has been concluded in a previous report (Meyer, 1968) that a single convulsion exerted an amnesic effect upon this particular avoidance task. Since it is obviously crucial to find out if a single convulsion will induce retrograde amnesia for learning the same task that it facilitates, several attempts were made to induce retrograde amnesia for the kind of avoidance training given in Part 1.

Procedure. Using the avoidance training procedure described earlier, 102 animals each received two sessions of avoidance training. Just prior to the first session, each animal was put in the apparatus for 5 min. After the first session the animal was lifted out, earclips were put on and, 30 sec. after the last trial, either a convulsion

or a pseudo-convulsion was produced. In the case of the convulsion, the ECS was either 0.3 sec., or 0.6 sec. or 2.0 sec. in duration. Using the same avoidance procedure, with the omission of any exploratory period beforehand, some groups were tested 24 hr. later and the others were tested 50 hr. later, as shown in column 2 of Table 2.

Results. Twenty of the 87 convulsed animals were discarded, as shown in Table 13. For the remaining 67 convulsed animals and 15 pseudo-convulsed animals, the mean numbers of shocks received in reaching the 90% avoidance criterion are shown in Table 2*. Although each group improved significantly between the first and the second sessions, there were no statistically significant differences among the improvements shown by each group. Each group of animals improved about the same amount between sessions, and no convulsed group's performance was statistically different from the pseudo-convulsed group's performance in either session. Even when all the results of the convulsed animals were pooled, the change between the first and second session was not statistically different from that of the pseudo-convulsion group ($U = 467$; $z = 0.8$; $p > .21$). Thus, all the groups performed about the same in

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*Individual results of the experiment are shown in Appendix B.

Table 2

Mean number of shocks received in reaching 90% criterion in the active-avoidance task as a result of convulsions after the first session.

Duration of ECS	Intersession Interval	n	First Session	Second Session	Change*
Pseudo-Convulsion	24 hr.	15	5.9	2.8	-3.1**
0.3 sec.	24 hr.	18	6.1	3.1	-2.9**
0.3 sec.	50 hr.	20	5.9	3.6	-2.3**
0.6 sec.	24 hr.	18	5.6	2.8	-2.7**
2.0 sec.	50 hr.	11	5.0	2.7	-2.3**

* Analysis of Variance (of the changes between session): $H = 3.9$, $df = 4$, $p > .30$.

**significant change between sessions ($p < .01$)

both the first and second sessions, and the convulsion induced no detectable amnesia for the avoidance training of the first session.

Part 3. Amnesia and reduced freezing.

A convulsion might have failed to affect performance in the second session of Part 2 either because it failed to affect the memory of the first day's training or because, even though it induced some amnesia, this was offset by the reduced freezing suggested by the results of Part 1. If an animal spent the same amount of time in the apparatus that it took to learn the avoidance response, and if a convulsion immediately afterward induced a loss of memory for the experience, then whether an animal received training or just explored for the equivalent amount of time before having a convulsion should make no difference in subsequent avoidance training in the apparatus. Animals which explored and animals which had training would effectively be learning the response for the first time. However, under these conditions, if the convulsion actually failed to affect memory, then, in subsequent avoidance training, animals which received prior training should perform better than animals which were merely allowed to explore the apparatus for an equivalent amount of time. In the present experiment, therefore, animals were first placed in the apparatus, then received a single convulsion

and then, 24 hr. later, received avoidance training. The results from this procedure can also be compared with appropriate results from the preceding parts.

Procedure. First, 27 animals were each placed in the avoidance apparatus for 11 min. (the mean time taken to learn the response in Part 2). Then 13 animals received a single ECS 30 sec. after being lifted out of the apparatus; the other 14 animals were simply returned to the home cages without ECS. Finally, 24 hr. later, each animal received avoidance training as previously described, with no exploratory period beforehand.

Results. Three of the 13 convulsed animals were discarded, as shown in Table 13. The mean numbers of shocks received in reaching the 90% avoidance criterion are shown in Table 3*. It can be seen in Table 3 that the convulsed animals learned the avoidance response with fewer shocks than the non-convulsed animals. Thus, a single convulsion again facilitated one-way active-avoidance learning.

In order to interpret these results, several comparisons must be made with the results in Parts 1 and 2. The non-convulsed animals in the present experiment received more shocks than the No Convulsion/Exploration Group in

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*Individual results of the experiment are shown in Appendix C.

Table 3

Mean number of shocks received in reaching 90% criterion in the active-avoidance task after the exploratory period followed by a convulsion or no convulsion.

TREATMENT	n	SHOCKS
Convulsion	10	3.8*
No Convulsion	14	8.6

*$p < .02$

Part 1 ($p < .02$) and did not differ significantly from the No Convulsion/No Exploration Group in Part 1. This is to say, the 11 min. of exploration 24 hr. before training did not facilitate the avoidance learning, although 5 min. of exploration immediately before training had facilitated avoidance learning.

Comparisons should also be made between the convulsed animals in Part 3 and the group in Part 2 which received the 0.3-sec. ECS followed by a 24-hr. intersession interval. These two groups were treated the same except that the animals in Part 2 received avoidance training in the first session while the animals in the present experiment simply explored the apparatus for an equivalent amount of time. A comparison would therefore indicate the effect of training, as opposed to exploratory experience, when convulsive effects are controlled. The animals which only explored received significantly more shocks in reaching the avoidance criterion than the animals which received previous avoidance training ($p < .02$). A statistically significant difference between these groups may not seen likely since the means are very close (3.8 shocks for the animals which explored and 3.1 for the animals which were trained). However, one of the trained animals received 18 shocks (see Appendix C), an atypically high number in the second session, so that the mean value for

the trained animals does not describe the group performance very well. The median number of shocks received by the trained animals in the second session, for example, was only 2.1 compared with the median of 3.5 for the animals which only explored. Thus, prior training followed by a convulsion produced better avoidance performance than the temporal equivalent spent in exploration followed by a convulsion.

Finally, the 11 min. of exploration followed by a convulsion produced better avoidance performance than a convulsion alone. The Convulsed Group in Part 3 received significantly fewer shocks in reaching the avoidance criterion than the Convulsion/No Exploration Group in Part 1 ($p < .02$). Thus, although the 11-min. exploratory period the day before training did not itself facilitate avoidance learning, this period in conjunction with a convulsion facilitated avoidance learning more than a convulsion by itself. That is, the effect of the exploration the day before training was manifested only in conjunction with a subsequent convulsion.

Summary of Active-Avoidance Experiments

The major results of the Active-Avoidance Experiments can be summarized as follows. Compared with animals which receive no convulsion and have no experience in

the apparatus before training, the mean number of shocks received in reaching a 90% avoidance criterion in a given session was decreased by about 68% after one session of training, by about 40% after a single convulsion 24 hr. before training, by about 42% after 5 min. of exploration just before training, and by about 57% after 11 min. of exploration 24 hr, before training followed by a single convulsion.

However, avoidance performance was unaffected by exploration of the apparatus 24 hr. before training when no convulsion was induced after the exploratory period. There was also no evidence that a convulsion 30 sec. after the completion of active-avoidance training induced any retrograde amnesia for the training.

II. ESCAPE EXPERIMENTS

The conclusion that a convulsion given shortly after avoidance training produced little or no amnesia for the training is somewhat at odds with the conclusions of most previous investigators. Since it took about 6 min. to learn the avoidance response used in the first experiments (plus the 5-min. exploratory period in Part 2), it was decided to search for an aversively-motivated task which required active movement but which could be learned in a single trial. A one-trial task was considered essential because the point in time when learning occurred (and presumably, consolidation began) could then be established. This would mean that the interval between learning and a subsequent convulsion (the ECS-delay interval) could be specified and thus the standards conventionally used to demonstrate convulsion-induced retrograde amnesia would be fully met.

One task which was investigated required running through a hole in the wall of a box in order to escape electric shock. Animals were placed on the charged grid floor of a square box, and could escape by running through a 2-in. square hole in one wall at floor level. Altogether

75 animals were tested on this task following various training, convulsive and exploratory treatments. Although a convulsion did not induce detectable retrograde amnesia for escape training, other results made it doubtful that the changes between two trials met conventional standards used to establish "learning." It was found, for example, that if animals received no escape training but were allowed to explore a large wooden stand for 20 min., they performed as well on the escape task the next day as the animals which had escape training after exploring the stand for 20 min. (see Table 4).

Since this hole-in-the-wall task did not appear to be a satisfactory learning task, it was abandoned. Group mean latencies on this task are shown in Table 4 for comparison with the results obtained with the next task investigated. This next task required animals to jump out of a box in order to escape electric shock.

Apparatus. The escape apparatus (jump box) was a shellacked plywood box, 12 in. by 12 in. with walls 11 in. high. A platform 2 1/2 in. wide ran around the outside of the box, 1/2 in. from the top. The floor of the box was a grid of 1/8-in. stainless steel rods set about 1/2 in. apart. The grid floor was connected to a Physiological Electronics Four-Line Scrambler (Model 3201) set at about 130 v. A large wooden stand with sawdust on the floor was

Table 4

Group mean escape latencies for the "hole-in-the-wall" task.

TREATMENT	n	FIRST TRIAL	SECOND TRIAL
20-min. exploration on wooden stand followed by first escape trial; no convulsion; second trial 24 hr. later.	16	17.9	7.5*
Treated like first group, but 0.3-sec. ECS given 30 sec. after first trial	16	17.8	6.9*
Treated like first group, but spent 18 sec. in box with no grid shock, instead of escape trial on first day	13	--	5.9
Explored wooden stand for 20 min., like groups above, but then directly returned to home cage without escape trial or exploration in the apparatus	19	--	8.5
Treated like first group, but without any exploration before first escape trial	11	25.4	9.1*

*shorter than latency on first trial ($p < .02$)

Note: no statistically significant differences among the groups within either trial.

also used.

Escape Procedure. The escape response required an animal to jump out of the box onto the platform which ran around the outside. On each escape trial, the animal was simply placed on the already-charged grid floor, allowed to jump out, and returned to the home cage. The time taken to jump out of the box was recorded with a stopwatch for each animal, and any animal which failed to jump out within 45 sec. was discarded. Escape latencies were considered the time taken until both forepaws grasped the top of the box; invariably the animal then pulled itself over the top and onto the platform.

There were two escape trials for certain groups of animals in the present experiments. In this case, the second trial occurred 24 hr. after the first trial and, immediately before the first trial, a group of 5 or 6 animals (from a single cage) was put on the wooden stand for 20 min. This exploratory period did not occur prior to the second trial, nor where only a single trial was given; in these cases, the animals from a single cage were placed on the wooden stand but remained there only until any preceding animal had received its escape trial.

Part 1. Learning the Escape Response.

The purposes of this experiment were three. First,

the experiment was undertaken to see if the jump-escape training produced reliable improvements in performance and, thus, to ascertain whether this task could be used to measure aversively-motivated, one-trial learning which required active movement. Second, the experiment was intended to demonstrate the factors necessary to any such learning. Third, it was designed to show whether a convulsion or exploratory period facilitated any such learning.

Procedure. Six groups of animals (N=97) received escape training. The Standard-Training Group received two escape trials as described in the Escape Procedure. The animals in the No-Shock Group were first placed on the wooden stand for 20 min. and then placed in the apparatus for 15 sec. with the floor uncharged (15 sec. was about the mean time taken by the Standard-Training Group to escape on the first trial). Then these animals were removed from the apparatus, returned to the home cage and received a single escape trial 24 hr. later.

Animals in the Wood-Top Group were placed on the wooden stand for 20 min. and then placed in the apparatus for 15 sec. with the grid floor charged and a plywood lid over the box in order to prevent escape. Then these animals were removed from the apparatus, returned to the home

cage and received a single escape trial 24 hr. later. The Plastic-Top Group was treated just like the Wood-Top Group except that a plexiglas lid was used to prevent escape rather than a plywood lid. These latter two groups thus received 15 sec. of inescapable shock in the apparatus, but the animals in the Plastic-Top Group could see out during the shock while the animals in the Wood-Top Group could not.

The animals in the Convulsed Group received a single ECS and, 24 hr. later, were placed on the wooden stand for 20 min. and then received a single escape trial. The standard escape procedure (used for the Standard-Training Group) included a 20-min. exploratory period on the wooden stand immediately prior to the first trial. In order to assess the effect of this exploratory period, there was also a No-Exploration Group which was treated just like the Standard-Training Group on the first trial, with the omission of the exploratory period preceding escape training. The No-Exploration Group received only a single trial.

Results. Ten animals were discarded (Table 13). Group mean escape latencies for the remaining 87 animals are shown in Table 5* It is clear from Table 5 that the

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* Individual results of the experiment are shown in Appendix D.

Table 5

Group mean latencies in the Jump-Escape Task as a result of different training treatments.

GROUP	n	FIRST TRIAL	SECOND TRIAL
Standard-Training Group	16	13.7	2.1*
No-Shock Group	15	17.6	--
Wood-Top Group	13	16.4	--
Plastic-Top Group	15	17.6	--
Convulsed Group	13	14.7	--
No-Exploration Group	15	15.1	--

*shorter than latency on first trial ($p < .01$)

Analysis of Variance (of latencies listed under "First Trial"): $H = 0.74$, $df = 5$, $p > .98$

escape latencies decreased markedly and reliably from the first to the second trial for the Standard-Training Group. Only one animal in 16 failed to show improvement in escape performance (Appendix D). This improvement in escape performance appeared to be due entirely to the act of escaping in the first trial. Neither exploring the box nor being shocked in the box, for an equivalent period of time, produced significantly different escape latencies than those of the Standard-Training Group in the first trial. This latter result is crucial to the subsequent experiments on retrograde amnesia, since it indicates that the learning occurs (and presumably consolidation begins) at the moment of escape, rather than at the onset of shock or some other point in time.

The results in Table 5 also demonstrate that an opportunity to explore for 20 min. immediately before training did not affect escape learning. Neither was escape learning affected by a convulsion 24 hr. before training. Thus exploration did not facilitate escape learning, although it facilitated active-avoidance learning and learning the hole-in-the-wall task. Furthermore, exploration plus a convulsion together did not facilitate escape learning, although either of these treatments (and both together) facilitated active-avoidance learning. These findings are important because they indicate that

freezing or inhibition of movement is not an important component in acquisition of the escape response.

When animals in the Plastic-Top Group were receiving inescapable shock, they behaved somewhat differently than the animals in the Standard-Training Group in the first trial. Animals in both groups showed the usual unconditioned reactions to electric shock on the grid (squealing, urinating, biting the bars, jumping about and so forth) but the animals which could escape tended more often to rear against one wall or make abortive jumps before actually jumping out of the box. Under the plastic top, the animals tended more often to remain on all four paws, and none was observed trying to jump out. Animals in both groups ran around the grid near the walls but, after a while some of the animals in the Plastic-Top Group crouched and remained still; this rarely happened with the Standard-Training Group. Animals in the Plastic-Top Group also seemed to move backwards more frequently than animals in the Standard-Training Group. When the plastic top was removed, only one or two animals tried to jump out and this occurred after an attempt was first made to pick them up from the grid floor.

Thus the animals which received inescapable shock showed no evidence that they had learned the escape response but were prevented from performing it. Similarly,

freezing was not observed to be a major component act in learning the jump-escape response. These observations therefore support the numerical results presented before.

Part 2. Retrograde effects of a convulsion.

A second experiment was undertaken to investigate the retrograde effects of a single convulsion on jump-escape learning. Convulsions were induced 1 sec. or 30 sec. after a single training trial, and the effects of the convulsion was tested in a second trial by comparisons between convulsed and non-convulsed animals.

Procedure. Four groups of animals (N=86) received two escape trials as described in the Escape Procedure. The 30-sec. Convulsion Group received a single ECS 30 sec. after jumping out of the box on the first trial; the 30-sec. Control Group received a pseudo-convulsion 30 sec. after escaping on the first trial. The 1-sec. Convulsion Group received a single ECS 1 sec. after jumping out of the box on the first trial; because this group had earclips put on before the first trial, there was also a 1-sec. Control Group which had earclips put on before the first trial and received a pseudo-convulsion 1 sec. after escaping.

Results. Twenty-one of the 86 animals were discarded (Table 13). The group mean escape latencies for

the other 65 animals are shown in Table 6*. As shown in Table 6, each group improved significantly from the first to the second trial, and there were no statistically significant differences in escape performance among the groups on either the first trial or the second trial. Thus, each group showed evidence of learning the escape response, and the convulsions induced no detectable retrograde amnesia for the jump-escape training.

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*Individual results of the experiment are shown in Appendix E.

Table 6

Group mean latencies in the Jump-Escape Task as a result of a convulsion after the first trial.

GROUP	n	FIRST TRIAL	SECOND TRIAL	CHANGE
30-sec. Convulsion Group	18	13.8	2.8*	-11.0
30-sec. Control Group	15	13.5	2.9*	-10.6
1-sec. Convulsion Group	16	14.7	3.8*	-10.9
1-sec. Control Group	16	14.3	3.3*	-10.9

*shorter than latency on first trial ($p < .01$)

Analysis of Variance (among the changes between trials): $H = 0.29$, $df = 3$, $p > .95$

III. DRINKING EXPERIMENTS

In view of the finding that a convulsion induced no detectable retrograde amnesia for escape training, the purpose of the Drinking Experiments was to ascertain if there was some factor other than the type of response required which might have been responsible for the failure to induce retrograde amnesia. An investigation was therefore made of the retrograde effect of a convulsion upon passive-avoidance training in a task in which the training and convulsive treatments were as similar as possible to those used in the Escape Experiments. An investigation was also made of the retrograde effects of a convulsion upon escape training which was preceded by the same treatment given before the passive-avoidance task.

Apparatus. The apparatus from the Escape Experiments was used but the jump box was modified to hold a water bottle on the outside. A curved spout projected into the box through a 1/2-in. hole drilled in the midline of one wall, 2 in. above the grid floor.

Drinking Procedure. Each animal (N=70) was deprived of water in the home cage and was allowed to drink

only in the apparatus for 10 min. each day. After three days of this treatment, the time taken to start licking the water spout was recorded with a stopwatch. Training to drink continued until each animal had a latency of 4 sec. or less and the over-all mean latency was less than 3 sec. It took about 12 days of training to reach this criterion. The day after the criterion was reached, either passive-avoidance or jump-escape training was administered*.

Part 1. Drink-avoidance task.

The purpose of this experiment was to test the retrograde effect of a convulsion upon passive-avoidance training in which the duration of grid shock, the apparatus and the convulsive parameters were the same as those used in Part 2 of the Escape Experiments for the 30-sec. groups.

Procedure. After the drinking procedure, two groups of animals (N=26) received passive-avoidance training. Animals from both groups were placed in the apparatus and the grid floor was charged for 15 sec. when the

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*By the end of the drinking procedure, 8 animals had contracted paratyphoid disease and one animal had a latency longer than 4 sec. after 12 days of training; these 9 animals were discarded (they are not recorded in Table 13), which left 61 animals in the experiments.

animals started to drink (15 sec. was about the mean jump-escape latency in the first trial of the Escape Experiments). A plywood lid was placed over the apparatus while the grid was charged in order to prevent escape.

At the end of the 15-sec. grid shock, the animals were removed from the apparatus and the Convulsed Group (n=14) received a single ECS 30 sec. later. The Pseudo-Convulsed Group (n=12) received a pseudo-convulsion after 30 sec. The animals were returned to the home cages and drinking latencies were recorded again 24 hr. later, to a maximum latency of 30 sec.

Results. Three convulsed animals were discarded from the experiment (Table 13). The group mean drinking latencies for the other 23 animals are shown in Table 7*. It can be seen from Table 7 that the passive-avoidance training increased the drinking latencies whether or not a convulsion was induced after the training. However, the pseudo-convulsed animals had significantly greater increases in latencies after the passive-avoidance training than the convulsed animals. That is, the convulsion impaired retention of the passive-avoidance training, thereby indicating that it had induced retrograde amnesia

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*Individual results of the experiment are shown in Appendix F.

Table 7

Group mean drinking latencies before and after passive-avoidance training.

	n	BEFORE AVOIDANCE TRAINING	AFTER AVOIDANCE TRAINING	CHANGE
Convulsed Group	11	2.4	7.9	5.6*
Pseudo-Convulsed Group	12	2.3	30.0***	25.2**

*$p < .05$

** $p < .01$

***longer than mean latency of the convulsed animals after avoidance training ($p < .002$)

for the drink-avoidance training.

Part 2. Jump-Escape Task.

A convulsion induced retrograde amnesia for passive-avoidance training but not for escape training. It is possible that this difference was somehow due to the experience of drinking in the apparatus before passive-avoidance training, or perhaps the water deprivation changed the impedance of the brain and thus altered the effectiveness of the ECS. In order to check possibilities like these, animals were first trained to drink in the apparatus and were then trained to jump out of the apparatus in order to escape electric shock.

Procedure. Two groups of animals (N=35) were first trained to drink in the apparatus, as described in the Drinking Procedure above. Then they received two escape trials, as described in the Escape Procedure (p. 73). The Convulsed Group received a single ECS, and the Pseudo-Convulsed Group received a pseudo-convulsion, 1 sec. after the jump-escape response in the first trial.

Results. Three animals were discarded from the experiment (Table 13). Group mean escape latencies for the remaining 32 animals are shown in Table 8*. Both

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*Individual results of the experiment are shown in Appendix G.

Table 8

Group mean latencies in the Jump-Escape Task after training to drink in the escape apparatus.

	n	FIRST TRIAL	SECOND TRIAL	CHANGE
Convulsed Group	16	21.5	7.2	-14.3*
Pseudo-Convulsed Group	16	23.3	7.3	-16.1*

*$p < .01$.

groups showed significant decreases in escape latency between the first and second trial, and there were no statistically significant differences between the two groups in either trial. Thus a convulsion 1 sec. after training induced no retrograde amnesia for the escape learning.

The latencies in the first trial of this experiment were pooled and compared with the pooled latencies in the first trial in Part 2 of the Escape Experiments (Table 6). The animals in the present experiment had significantly longer escape latencies in the first trial than the animals in Part 2 of the Escape Experiments ($p < .02$). Thus the water deprivation and drinking procedure made animals escape from the jump box more slowly, but the failure of a convulsion to induce retrograde amnesia for escape training cannot be attributed to these treatments.

The results of the Drinking Experiments, that a single convulsion induced retrograde amnesia for passive-avoidance training but not for escape training, indicated that the appearance of retrograde amnesia depends upon the type of response required. In the two Drinking Experiments, the apparatus and most experimental parameters were the same; factors such as amount of handling, degree of water deprivation, experience in the apparatus, intensity of gridshock and electroconvulsive shock and so forth

were also the same for the two tasks. The main differences between the experiments were the type of response required and the ECS-delay interval. The shorter ECS-delay interval used in the jump-escape task should have been more conducive to retrograde amnesia than the longer interval used in the drink-avoidance task. Therefore, the observation of retrograde amnesia in the passive-avoidance task but not in the escape task indicated that the appearance of retrograde amnesia was due to the different types of response required in the two tasks.

IV. PASSIVE-AVOIDANCE EXPERIMENTS

There have been, as pointed out earlier (p. 30), large differences in estimates of the length of the hypothetical consolidation period. For the most part, these estimates have been based upon the longest ECS-delay interval which produced apparent retrograde amnesia in a particular experimental task--usually a passive-avoidance task--but each published report has included only one such task. The first of the present experiments was therefore an attempt to confirm the different results obtained by Weissman (1964) and by Chorover and Schiller (1965). Weissman (1964) observed retrograde effects from a single convulsion which occurred several minutes after passive-avoidance training, while Chorover and Schiller (1965) observed retrograde effects only if the convulsion occurred within a few seconds after passive-avoidance training.

In the second of the present experiments, certain assumptions which Chorover and Schiller (1965, 1966) made about the task they used were investigated.

Apparatus. The drinking apparatus was a hardware-cloth box, 12 in. long, 7 1/2 in. wide and 8 in. high, built around a supporting wooden frame. A water bottle

could be fitted to the front of the apparatus with a curved spout projecting inside through a hardware cloth.

The step-down apparatus was a grey plywood box, 15 1/2 in. by 15 1/2 in. with walls 11 3/4 in. high. The floor of the box was a grid of 1/8-in. stainless-steel rods set about 1/2 in. apart. An insulated grey wooden platform, 5 1/8 in. square and 2 1/8 in. high, was located in the centre of the grid floor.

The water spout in the drinking apparatus, and the grid floor in the step-down apparatus, could be connected through a variable resistance to the secondary coil of an 1100-v. transformer. The resistance was adjusted so that, when an animal completed the circuit, a milliammeter in series indicated a current of about 1 mA.

Part 1. Comparison of the two tasks.

In the present experiment, two passive-avoidance tasks were compared with respect to the retrograde effects of a single convulsion which occurred at various intervals after training. The step-down task was the same as the task used by Chorover and Schiller (1965). The other task involved the passive avoidance of drinking and was therefore similar to the task used by Weissman (1964). For purposes of interpretation, performance on both tasks was measured in terms of latencies and the maximum lat-

ency after passive-avoidance training was 30 sec. for both tasks.

Passive-avoidance procedures. For the drinking task (n=112) animals were put in the apparatus daily and allowed to drink for 15 min. The time taken to start drinking was recorded with a stopwatch. After 10 days, any animal with a latency greater than 4 sec. was discarded. For passive-avoidance training, each animal except those in the No-Training Group was put in the apparatus for 15 sec. with the water spout charged relative to the hardware-cloth floor. For the No-Training Group, the spout was disconnected from the transformer. In general, animals received 2 to 5 brief shocks through the mouth with the transformer connected. At the end of the passive-avoidance training trial, the animals underwent the Convulsion Procedure and were tested 24 hr. later to a maximum latency of 30 sec.

For the step-down task (n=133), animals were put in the apparatus for 15 sec. per day for four days. The animal was placed on the wooden platform and the time taken to place both forepaws on the grid floor was recorded with a stopwatch. Any animal with a latency greater than 4 sec. was discarded. For passive-avoidance training, the grid floor was charged and, after stepping down, the animals except those in the No-Training Group received

grid shock for about 2 sec. For the No-Training Group, the grid floor was not charged. At the end of the passive-avoidance training trial, the animals underwent the Convulsion Procedure and were tested 24 hr. later to a maximum latency of 30 sec.

Convulsion Procedure. For the drinking task, a single ECS was given 1 sec., 30 sec., 60 sec., or 300 sec. after training (15 sec. in the apparatus). For the step-down task, a single ECS was given 1 sec., 10 sec., 30 sec., or 60 sec. after training (termination of grid shock). For both tasks, the No-Shock Groups received a single ECS 30 sec. after training and the Pseudo-Convulsion Groups received a pseudo-convulsion 30 sec. after training.

Earclips were put on the animals in the 1-sec. and 10-sec. groups before the passive-avoidance training trial, and the convulsion occurred in the apparatus. Earclips were put on the other animals after they were removed from the apparatus and the convulsion occurred just beside the apparatus. The animals in the 60-sec. and 300-sec. groups spent the ECS-delay interval on the wooden stand used in the Escape Experiments and the animals in the 30-sec. groups spent the interval beside the apparatus.

Results. Forty-two animals were discarded (Table

13). The results for the remaining 203 animals are shown in Table 9*. It is clear that the ECS-delay intervals which produced the shorter latencies indicative of retrograde amnesia differed greatly between the two passive-avoidance tasks. For example, the 60-sec. group in the drinking task had a mean latency which was significantly shorter than its pseudo-convulsed counterpart, whereas the 30 sec. group in the step-down task had a mean latency which was not significantly different than that of its pseudo-convulsed counterpart. It should also be noted that the results from the step-down procedure are similar to the results of Chorover and Schiller (1965). The 10-sec. group, but not the 30-sec. group, had latencies which were significantly shorter than the comparable pseudo-convulsion group.

Thus, the ECS exerted no amnesia for the step-down task at ECS-delay intervals longer than 10 sec. while the ECS apparently had an amnesic effect on the drinking task at ECS-delay intervals up to 60 sec. It is also clear from this analysis that neither group showed constant effects from different ECS-delay intervals. That is, a gradient of mean latencies relative to increasing

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*Individual results of the experiment are shown in Appendix H.

Table 9

Group mean passive-avoidance latencies as a result of various ECS-delay intervals after the first trial.

	STEP-DOWN TASK				DRINKING TASK			
	n	Before Avoidance Training	After Avoidance Training	Change*	n	Before Avoidance Training	After Avoidance Training	Change*
No-Training Groups	17	0.7	0.9	0.2**	17	1.6	1.6	0.0**
1-sec. Groups	20	0.9	2.5	1.6**	17	2.1	2.6	0.4**
10-sec. Group	17	0.8	8.1	7.3**				
30-sec. Groups	15	0.9	28.3	27.5	18	1.8	8.7	6.9**
60-sec. Groups	12	0.7	28.5	27.8	17	1.8	20.4	18.5**
300-sec. Group					17	1.7	25.4	23.7
Pseudo-Convulsion Group	18	0.9	28.3	27.4	18	1.8	28.7	26.9

*Analysis of Variance (of the changes from avoidance training): $H = 19.3$, $df = 5$, $p < .01$. for the Step-Down Task; $H = 14.4$, $df = 5$, $p < .02$ for the Drinking Task.

**significantly less than the comparable Pseudo-Convulsion Group ($p < .002$).

NOTE: No significant differences among the Before-Training Latencies in either task.

ECS-delay intervals occurred with both passive-avoidance tasks.

If one faced the front of the drinking apparatus, the spout of the water bottle projected into the box on the right side, and the animals were placed on the left side, facing the back wall. Before passive-avoidance training, the animals would run quickly to the spout followed a U-shaped course. In the 15-sec. passive-avoidance training trial, the animals naturally also followed this course, and then jumped backwards when the mouth contacted the spout. Animals then tended to "slink" back to the spout, and jump back again when shocked. Different animals repeated this pattern 2 to 5 times, although there was no formal quantification of this sort of observation. Finally, the animals tended to go to the back of the box (usually the left back corner if one faced the apparatus) and stand facing in the direction of the spout. After the passive-avoidance training, the animals persisted in running quickly over the U-shaped route, but animals in the Pseudo-Convulsion Group would stop short of the spout, "oscillate" there for a while, and then usually retreat once again to the back of the box. The major difference among the groups seemed to be the length of this "oscillation" and whether the animals retreated to the back of the box or extended their

necks to drink. So a distinct impression was given that the animals moved about the apparatus a great deal. This was, of course, particularly true of the convulsed animals since the pseduo-convulsed animals tended more often to remain either at the back of the cage or else "frozen" in front of the spout.

Similar behaviour was observed in the step-down apparatus, although the differences between convulsed and pseudo-convulsed animals was not so apparent here. The pseudo-convulsed animals seemed to be more "limp" than the convulsed animals and tended to lie down on the platform. The convulsed animals seemed to have more muscle tone and would stand up on the platform more often. Indeed, the convulsed animals often rotated about the platform and one animal actually jumped out of the apparatus. Another extended its body so that it leaned against the wall with its forepaws while its hindpaws remained on the platform. Again, the impression was that the convulsed animals had difficulty remaining still.

Part 2. Discrimination in the Step-Down Task

The results of the preceding experiment indicated that the longest ECS-delay interval which produces apparent retrograde amnesia depends, in part, on the passive-avoidance task used. In attempting to explain such results,

Chorover and Schiller (1966) suggested that a convulsion induces retrograde amnesia only in tasks which "depend upon retention of a specific discrimination of cues." A fundamental assumption implicit in Chorover and Schiller's (1966) theory of retrograde amnesia is, therefore, that the step-down task requires discriminated learning. This assumption was tested in the present experiment.

Procedure. Three groups of animals were trained and tested in the step-down apparatus (N=36). Group 1 underwent the same treatment as the Pseudo-Convulsion Group in Part 1 except that no earclips were used and the grid shock lasted 4 sec. rather than 2 sec. Group 2 was treated the same way as Group 1 except that, for testing 24 hr. after passive-avoidance training, the animals were placed on the grid floor beside the platform. An area of grid the same size as the platform was drawn with a "magic marker" and the time taken to move both forepaws outside this area was recorded. Training for Group 3 comprised placing the animal in the apparatus with the platform removed and, 1 sec. later, applying grid shock for 4 sec. For testing Group 3, 24 hr. later, the platform was put back in the apparatus, the animals were placed on it, and the step-down latencies were recorded.

Results. One animal was discarded from the ex-

periment (Table 13). The group mean latencies in testing for the other 35 animals are shown in Table 10*. There were no significant differences in test latencies among the three groups. Thus, the animals performed about the same no matter which of the three treatments they received, and this result constitutes evidence that the step-down task does not require discriminated learning. Rather, the task appears to measure a fairly general suppression of movement.

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*Individual results of the experiment are shown in Appendix I.

Table 10

Group mean latencies in the Step-Down Task after different training treatments.

GROUP	LATENCY
Group 1	27.0
Group 2	26.6
Group 3	28.3

Analysis of Variance: $H = 1.12$, $df = 2$, $p > .50$

V. MULTIPLE-CONVULSIONS EXPERIMENTS

All the present experiments this far have involved the use of only a single convulsion. The Multiple-Convulsions Experiments were carried out to investigate the effect of a series of convulsions before training upon the retrograde effect of a single convulsion after training. This effect was investigated with both step-down passive-avoidance training (Part 1) and jump-escape training (Part 2).

Part 1. Passive-Avoidance Training

Procedure. Two groups of animals (N=29) underwent the same treatment in the step-down apparatus as the 10-sec. group in Part 1 of the Passive-Avoidance Experiments, except that the ECS-delay interval was actually 12 sec. rather than 10 sec. During the four days preceding passive-avoidance training, the animals in the Convulsed Group (n=17) also received 5 ECSs and the animals in the Pseudo-Convulsed Group (n=12) received 5 pseudo-convulsions. The convulsions and pseudo-convulsions were spaced at least 5 hr. apart.

Results. Nine animals were discarded from the experiment (Table 13). The group mean step-down latencies

for the other 20 animals are shown in Table 11*. The latencies of the two groups were not significantly different before passive-avoidance training but, following passive-avoidance training and a convulsion 12 sec. later, the Convulsed Group had significantly shorter latencies than the Pseudo-Convulsed Group. Thus, the series of 5 convulsions beforehand had no detected effect upon "spontaneous" step-down latencies, but this treatment greatly inflated the apparent retrograde amnesia induced by a single convulsion after passive-avoidance training.

Part 2. Jump-Escape Task.

A series of convulsions can impair maze learning (Braun, Russell and Patton, 1949) and it has been suggested that such results might indicate that a series of convulsions slows down the consolidation process (Thompson, Harvey, Pennington, Smith, Gannon and Stockwell, 1958). In order to investigate whether the results of Part 1 of the present experiments were produced by slower consolidation or impaired learning, the same convulsive procedure used in Part 1 was applied to the jump-escape task used in the Escape Experiments.

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*Individual results of the experiment are shown in Appendix J.

Table 11

Group mean step-down latencies as a result of five convulsions before training.

	n	Before Avoidance Training	After Avoidance Training	Change
CONVULSED GROUP	10	0.6	5.2	4.6***
PSEUDO-CONVULSED GROUP	10	1.1	16.2*	15.1**

*longer than the Convulsed Group ($p < .02$)

**$p < .01$

***$p < .05$

Procedure. Two groups of animals (N=49) underwent the same treatment in the escape apparatus as the 1-sec. Convulsion Group in Part 2 of the Escape Experiments, except that there was no exploratory period before the first trial. The Convulsed Group (n=31) and the Pseudo-Convulsed Group (n=18) received the same convulsive treatment as their respective namesakes in Part 1 of the present experiments, except that the ECS after the first trial occurred 1 sec. after the response rather than 12 sec. afterwards.

Results. Twenty-two of the animals were discarded (see Table 13). The results for the other 27 animals are shown in Table 12*. The latencies of the two groups were not significantly different on either trial. Thus, the 5 convulsions beforehand affected neither initial escape performance nor the ability to learn the escape response and consolidate this learning.

Both groups had somewhat longer latencies on the first trial than the 1-sec. groups in Part 2 of the Escape Experiments (Table 5); the animals in the present experiments had latencies on the first trial like the animals which had been trained to drink in the box before escape training (Drinking Experiments, Part 2; Table 7). Thus, it seems

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*Individual results of the experiment are given in Appendix K.

Table 12

Group mean jump-escape latencies as a result of five convulsions before training.

TREATMENT	n	FIRST TRIAL	SECOND TRIAL	CHANGE
Convulsed Group	12	20.2	3.7	-16.5*
Pseudo-Convulsed Group	15	20.1	5.8	-14.3*

*$p < .01$

that handling and the like somehow increase jump-escape latencies initially. This effect, however, does not alter the finding that the convulsive treatments used with the escape task failed to increase susceptibility to ECS-induced retrograde amnesia.

On the second trial, the animals in the present experiments had latencies about the same as the animals in the 1-sec. groups in Part 2 of the Escape Experiments. The somewhat longer mean latency of the Pseudo-Convulsed Group in the present experiment was produced by two animals (see Appendix K). One animal took quite high jumps in the apparatus, but did not actually reach the top of the wall for about 13.5 sec. This animal actually seemed to have some problem co-ordinating his front limbs, although it was able to pull itself over the top of the wall onto the platform on the apparatus. The other animal (15.5 sec.) leaned against one wall, and seemed to place both hind paws on the same steel rod in the floor, thus averting the scrambled shock. Then it lowered its forepaws to the grid floor, received a shock, and jumped out shortly thereafter. Despite these two animals, there was no statistically significant difference between the Convulsed and Pseudo-Convulsed Groups on the second trial.

Table 13

Numbers of animals discarded from each experiment.

EXPERIMENTS	REASONS FOR DISCARDING: Did not meet behavioral criterion	Incomplete Tonic Extension	Suffered Spinal Injury
ACTIVE-AVOIDANCE EXPERIMENTS			
Part 1	--	--	--
Part 2 (retrograde effects)	--	9	11
Part 3 (from convulsed group)	--	2	1
ESCAPE EXPERIMENTS			
Part 1			
Standard-Training Group	1	--	--
No-Shock Group	1	--	--
Wood-Top Group	2	--	--
Plastic-Top Group	2	--	--
Convulsed Group	1	1	--
No-Exploration Group	2	--	--
Part 2	2	7	12
DRINKING EXPERIMENTS			
Passive-Avoidance Task	--	2	1
Jump-Escape Task	2	1	--

(table continues)

Table 13 (continued)

Number of animals discarded from each experiment.

	REASONS FOR DISCARDING		
	Did not meet behavioral criterion	Incomplete Tonic Extension	Suffered Spinal Injury
PASSIVE-AVOIDANCE EXPERIMENTS			
Part 1			
Drinking Task	5	3	1
Step-Down Task	5	9	19
Part 2 (From Group 2)	1	--	--
MULTIPLE-CONVULSIONS EXPERIMENTS			
Step-Down Task			
Convulsed Group	1	2	4
Pseudo-Convulsed Group	1	--	1*
Escape Task			
Convulsed Group	2	4	13
Pseudo-Convulsed Group	1	--	2*

*Injuries suffered as a result of the single convulsion following the first trial.

DISCUSSION

The results of inducing a convulsion at various intervals after passive-avoidance training on the step-down task (Passive-Avoidance Experiments, Part 1) confirmed the empirical findings of Chorover and Schiller (1965) that retrograde amnesia occurs in this situation only if the convulsion follows the training trial within about 10 sec. The results from the same convulsive procedure with the drinking task (Passive-Avoidance Experiments, Part 1) confirmed other empirical findings (e.g., Weissman, 1964) that retrograde amnesia occurs in some tasks when a convulsion follows a training trial by a minute or more. Thus, the longest ECS-delay interval which produced amnesia was different for the drinking and step-down tasks.

This difference was most likely produced by a difference in motivational strength between the two tasks. The motivation for leaving the platform in the step-down task was presumably a mild aversion to open, well-lit places or a weak thigmotaxic tendency (or both), whereas the motivation in the drinking task was thirst induced by 24 hr. of water deprivation. Quartermain and colleagues (1965) suggested that the strength of motivation, upon which

passive-avoidance training followed by a convulsion is superimposed, can affect the apparent consolidation period, and this interpretation seems to best account for the results of the Passive-Avoidance Experiments. Further evidence for this interpretation was obtained in an unreported experiment, the results of which indicated that the maximum amnesia-producing ECS-delay interval in the drinking task could be extended even further by water deprivation for 48 hr. rather than 24 hr.

There are, however, a number of alternative interpretations which should also be considered. One alternative is to infer different consolidation periods for different tasks and, while that is reasonable, considerable analysis of the present and previous reports is possible without this inference.

Another alternative is Cherkin's (1966) proposal (p. 38) that different consolidation periods represent different strengths of original learning. According to this proposal, it would be predicted, where performance on two tasks was the same before training, that any particular ECS-delay interval should have the same amnesic effect on the two tasks. This prediction obtains because, on the basis of previous reports, Cherkin (1966) inferred that the rate of consolidation was the same for all learning tasks.

In Part 1 of the Passive-Avoidance Experiments, the performance before training was quite similar on the two tasks: the step-down latencies were about 1 sec. and the drinking latencies were about 2 sec. However, a convulsion at the ECS-delay intervals tested produced apparently greater amnesia for the drinking task than for the step-down task. Thus, Cherkin's (1966) proposal does not easily account for these results.

It is also unlikely that Chorover and Schiller's (1966) proposal (p. 33) can account for the results. These investigators proposed that only tasks which involve discriminated learning show a time-dependent effect of lengthening the ECS-delay interval. According to this proposal, lengthening the ECS delay interval should have a constant effect upon tasks which do not require discriminated learning since "increased exploratory activity, rather than retrograde amnesia, causes the apparent 'memory impairment'" in such tasks. However, the results of Part 2 of the Passive-Avoidance Experiments indicated that the step-down task does not necessarily involve discriminated learning, although a graded effect was shown on this task with increasing ECS-delay intervals. Since the assumption that the step-down task requires discriminated learning underlies Chorover and Schiller's (1966) proposal, these results also

cast serious doubts upon the proposal as a whole.

Although the apparent consolidation periods were different for the drinking and step-down tasks, both passive-avoidance tasks showed clear retrograde amnesia from a convulsion shortly after training. In sharp contrast to these results, a convulsion induced no detectable retrograde amnesia for the escape task, even when the convulsion occurred only 1 sec. after the jump on the first trial (Escape Experiments, Part 2). Since this jump was shown to be responsible for learning the escape response (Escape Experiments, Part 1), it can be concluded that a convulsion 1 sec. after learning induced no detectable amnesia for the escape response. Note that in the escape procedure, any aversive effect of the convulsion would increase jumping latencies. Therefore, the finding that the latencies in the escape task were the same on the second trial whether or not a convulsion occurred after the first trial indicates no detectable aversive effect from the convulsion, as well as no detectable amnesic effect. Thus the lack of apparent amnesia for escape learning cannot be attributed either to aversive effects of the convulsion or to competing conditioned responses.

The results of the Drinking Experiments also indicate that the amnesic differences between convulsions after

escape training and passive-avoidance training cannot be attributed to the particular experimental conditions used, nor to factors like handling, thirst, brain impedance and so forth. Where animals were treated the same before training, and trained in the same apparatus, a convulsion after training induced apparent retrograde amnesia for passive-avoidance training and no detectable amnesia for escape training. Depriving animals of water except in the apparatus, and handling them otherwise as if they were to receive passive-avoidance training, produced longer latencies on the first escape trial (Drinking Experiments, Part 2), but these treatments did not cause the convulsion to induce any detected amnesia for the escape training. Thus, the different procedures before training were unimportant in accounting for the amnesic differences between convulsions used in the escape task and the passive-avoidance task.

Movement and Freezing

The joint observations of retrograde amnesia in the passive-avoidance tasks and no retrograde amnesia in the escape or active-avoidance tasks probably reflect the different movement requirements in the two kinds of tasks. The escape and active-avoidance tasks required an animal to make an active movement, while the passive-avoidance tasks required an animal to freeze or refrain from movement.

A convulsion induced clearly observed retrograde amnesia only in situations which required refraining from movement, and not in situations which required an active movement to be made. This difference between active and passive situations seems to be a general rule (p. 46) and an attempt to resolve or explain the difference is clearly necessary for understanding the behavioral effects of electroconvulsive shock. Unless it is assumed that different tasks have different consolidation periods and, in particular, that no consolidation period is required for escape and active-avoidance learning, this difference means that retrograde amnesia alone cannot account for the convulsive effect in the present experiments. A convulsion must have another effect in addition to the amnesic effect.

For several reasons, it is unlikely that this other effect is simply an increased tendency to make active movements after a convulsion, although such an inference would resolve the differences in amnesic effects between active and passive situations. First, a series of 5 convulsions induced neither shorter spontaneous latencies in the first trial of the step-down task (Multiple-Convulsions Experiments, Part 1) nor shorter jumping latencies in the escape task (Multiple-Convulsions Experiments, Part 2). Second, ECS usually decreases, not increases, the tendency to make active movements (Munn, 1950). Third, a single convulsion may in-

duce shorter spontaneous step-down latencies, but only for about 1 hr. afterwards (Kopp and colleagues, 1967).

A more likely resolution of the amnesic differences between convulsions after passive-avoidance training and escape or active avoidance training is the conclusion that a single convulsion reduces the tendency to freeze or refrain from movement in frightening situations.

When an animal is placed in an aversive situation and becomes frightened, it has two tendencies. One tendency is to remain motionless or to freeze (what Hebb (1955) called the "paralysis of terror"), and the other tendency is to run away from the aversive situation. The most common aversive motivation for laboratory purposes is electric shock. Electric shock, however, does not readily indicate to an animal where to move in order to escape or avoid, so the freezing tendency predominates at first in most laboratory experiments, whether the "correct" response is movement or immobility. Under "natural conditions", this may not be true of course but, in laboratory tasks, the predominant freezing tendency allows fear-motivated tasks which require immobility to be learned in a single trial and with a brief duration of shock. The freezing tendency, however, means that several trials or much longer durations of shock, or both, are needed for an animal to learn an

escape or active-avoidance response. In the present experiments, for instance, a passive-avoidance response was acquired as the result of only 2 sec. of shock while the escape response required a mean shock of about 15 sec. duration.

The results of several previous experiments, considered collectively, indicate that convulsions generally reduce or impair freezing tendencies. A series of convulsions, for instance, facilitated active-avoidance learning requiring either reversal in a T-maze (Casseday, 1966) or running back and forth in a shuttle box (Vanderwolf, 1963b). It takes even more trials to learn these tasks than to learn a one-way active-avoidance task because even more freezing is involved. In learning the bidirectional tasks, an animal must overcome the tendency to avoid previously aversive places, as well as the tendency to freeze after being shocked. A series of convulsions also impaired passive-avoidance learning (Poschel, 1957; Delprato, 1966) as well as performance (Hunt & Brady, 1951) and acquisition (Brady & Hunt, 1951) of a conditioned suppression response, or CER. This latter task simply requires freezing. The effect of the convulsions upon freezing evidently disappears within 30 days (Brady, 1951), but the effect of the convulsions is the same whether electric shock occurs between

or immediately after responses, that is, whether the task is operationally defined as conditioned suppression or passive avoidance (Spevack & Suboski, 1967).

These results can best be understood collectively if a series of convulsions reduces or impairs the tendency to freeze. If a single convulsion has the same effect, it becomes clearer why a convulsion induced apparent retrograde amnesia for the passive-avoidance training, but not for the escape training, in the present experiments. Evidence that a single convulsion reduces freezing was provided by the finding that a single convulsion facilitated one-way active-avoidance learning (Active-Avoidance Experiments, Part 1). As explained before, the active-avoidance training procedure probably induced a certain amount of freezing, which interfered with learning the active-avoidance response. The convulsion reduced this freezing tendency and thus allowed the convulsed animals to learn the avoidance response more quickly than non-convulsed animals. Similarly, an exploratory period before training also reduced the freezing tendency, or served as a "warm-up" period, and thereby facilitated active-avoidance learning (Active-Avoidance Experiments, Part 1). However, neither exploration nor a single convulsion before training facilitated escape learning (Escape Experiments, Part 3). Nor did a series of 5 convulsions facilitate escape learning (Multi-

ple-Convulsions Experiments, Part 2). In the escape procedure, the continuous presence of grid shock in the apparatus produced vigorous movements, and thus probably overcame any tendency to freeze. This meant that there was little or no freezing for the convulsion to overcome. In other words, the convulsive effect depended in part upon the amount of freezing produced by the task involved. For example, in a bidirectional active-avoidance task, a series of convulsions facilitated learning even when an exploratory period occurred before training (Vanderwolf, 1963b) while in a one-way active-avoidance task, a convulsion facilitated learning only in the absence of an exploratory period before training (Active-Avoidance Experiments, Part 1). This difference in efficacy between the convulsive treatments probably occurred because the bidirectional task produced a greater tendency to freeze than the one-way task, which was also why animals took more trials to learn the bidirectional task.

Although a single convulsion facilitated active-avoidance learning 24 hr. later, a single convulsion impaired passive-avoidance learning only up to 8 hr. afterwards (Kopp and colleagues, 1967). Since the effect of the convulsion in both cases was presumably reduced freezing, it seems that a single convulsion reduces freezing but,

24 hr. afterwards, this effect is fairly weak and not behaviorally obvious in a passive-avoidance situation. The reduced freezing did not seem to just gradually dissipate over time: in the passive-avoidance task, the latencies abruptly increased between 8 hr. and 12 hr. after the convulsion, rather than gradually increasing over the post-convulsion intervals tested (Kopp and colleagues, 1967). An analogous result might also occur with active-avoidance tasks, but there is no evidence on this point yet. In any case, a convulsion evidently reduces the tendency to freeze in frightening situations and, after 24 hr., this effect may be manifested only under certain circumstances. One circumstance is when movement is a requirement of the task, but another circumstance may be when there is also a slight amnesia.

Degree of Amnesia

Certain results in the present report suggest that the retrograde amnesia produced by a single convulsion is actually quite slight, although this is not easily quantifiable. In the Active-Avoidance Experiments, it was found that 11 min. of exploration followed by a convulsion produced better avoidance performance 24 hr. later than a convulsion alone. The reduced freezing was presumably the same in the two procedures, since both involved a single

convulsion. The effect of the 11 min. of exploration was itself sufficiently weak that, without a convulsion afterwards, it had no effect upon subsequent avoidance performance (Active-Avoidance Experiments, Part 3). However, in conjunction with the reduced freezing induced by the convulsion this exploratory period facilitated avoidance learning. Thus the convulsion afterwards did not induce amnesia for the exploratory period even though the effect of the exploratory period per se was quite weak. This result is consistent with a weak retrograde-amnesia effect from a single convulsion, but difficult to understand if a single convulsion induced a strong retrograde amnesia.

Another possible explanation for these results is that both the exploratory period and the convulsion alone produced a small reduction in freezing but acted together synergistically. This possibility seems unlikely, however, since a convulsion 24 hr. before training did not act synergistically with an exploratory period immediately before training, even though this exploratory period had a strong enough effect by itself to facilitate active-avoidance learning (Active-Avoidance Experiments, Part 1). The fact that a convulsion 1 sec. after training had no observed effect upon subsequent escape performance also suggests that the actual loss of memory caused by a convulsion is quite slight or incomplete. Finally, it has

been recently reported that, in a passive-avoidance task, the amount of retrograde amnesia induced by a single convulsion can be increased if a flashing light is presented during the ECS-delay interval (Miller, Misanin & Lewis, 1969). This result implies that the convulsion itself does not produce a maximum amnesia and is therefore also compatible with the idea that the amnesia produced by a single convulsion is quite weak.

An Explanatory Proposal

Thus, it seems reasonable to propose that a single convulsion produces a slightly reduced tendency to freeze in frightening situations, plus a weak retrograde amnesia, and that both effects are operative 24 hr. after the convulsion. Neither effect alone can account for the results outlined so far. If the convulsion induced only a reduction in freezing, then there is no particular reason why the latencies in the passive-avoidance tasks were a function of the ECS-delay interval; the dependence of the latencies in the passive-avoidance tasks upon the ECS-delay interval (Passive-Avoidance Experiments, Part 1) indicates retrograde amnesia as an effect of a single convulsion. On the other hand, if retrograde amnesia were the sole effect of the convulsion, then there would have been no facilitation of active-avoidance learning, and if the amnesic effect

were much stronger than the reduced-freezing effect, then some retrograde amnesia should have been observed in the tasks which required active movement. However, if a convulsion slightly reduced the tendency to freeze and also induced a weak retrograde amnesia, it becomes possible to account for the results so far, as follows:

First, consider the reduced-freezing effect. The reduction of the freezing tendency does not mean that a convulsed animal is incapable of sitting still. It means only that it is less likely to refrain from movement when frightened. Therefore, in a test of "spontaneous movement" 24 hr. after a single convulsion (Kopp and colleagues, 1967) or even after several convulsions (Multiple-Convulsions Experiments, Part 1), no effect of the convulsion was observed. However, when active movement is required or reinforced, as in active-avoidance learning, the reduced freezing will be manifested, as faster learning in this case (Active-Avoidance Experiments, Part 1). Where freezing is already at a minimum or nearly so, as in an escape task, no further facilitation of learning was possible from a convulsion (Escape Experiments, Part 3) or even from a series of convulsions (Multiple-Convulsions Experiments, Part 2). Similarly, the facilitation of active-avoidance learning induced by a convulsion can be masked by a "warm-up" period before training (Active-Avoidance Experiments, Part

1) because both the convulsion and the "warm-up" period served to reduce freezing, but either treatment alone was probably capable of overcoming all the freezing induced by this task. In tasks which produce more freezing, such as bidirectional active-avoidance tasks, convulsions plus a "warm-up" period facilitated learning even more than a "warm-up" period alone (Vanderwolf, 1963b). In other words, the manifestation of the convulsive effect depended in part upon the amount of freezing induced by the pertinent task.

As a more general proposition, the manifestation of the effects of a convulsion probably depends upon the number and strength of factors effectively conducive to movement in fear-producing situations. From this viewpoint, the reduced freezing produced by a convulsion is one factor effectively conducive to movement (in fear-producing situations but not necessarily otherwise). Any retrograde amnesia produced by a convulsion is effectively conducive to movement only in a task which requires immobility; in a task which requires active movement, any such retrograde amnesia is a factor effectively conducive to immobility. In practice, the manifestation of convulsive effects also depends upon how stringently actual movements are reflected by numerical data, such as latencies or percentages of correct responses and so forth. These two propositions naturally apply to both the manifestation of retrograde am-

nesia and the manifestation of reduced freezing.

In view of these propositions, consider a fear-motivated task which requires immobility, such as a passive-avoidance task. In such a task, both the retrograde amnesia and the reduced freezing produced by a convulsion are effectively conducive to movement. These two convulsive effects interact, probably in a synergistic manner, to produce movements which are reflected in the experimental results as, for instance, shorter step-down latencies. Thus a clearly observed apparent retrograde amnesia occurred in the passive-avoidance tasks when a convulsion occurred shortly after training because both convulsive effects were conducive to movement. In an aversively-motivated task which requires movement, however, any retrograde amnesia is conducive to immobility, so that this effect interacts antagonistically with the reduced freezing. These two convulsive effects tend to cancel one another, so that no apparent retrograde amnesia was seen in the escape or active-avoidance tasks. This proposal thus accounts for the different amnesic effects seen in fear-motivated tasks which require movement and tasks which require immobility.

The proposal also accounts for the other results in the present investigation, and can account, in general terms, for the apparent discrepancies among certain previous findings related to the question of the length of the consoli-

dation period. For example, the proposal allows the different consolidation periods obtained with the drinking task and the step-down task (Passive-Avoidance Experiments, Part 1) to be interpreted in terms of factors conducive to movement: the thirst or water-deprivation, or both, in the drinking task were more conducive to movement than the aversion to open, well-lit places or the thigmotaxic tendency in the step-down task. Increased movement is commonly observed following water deprivation. Since there were more factors conducive to movement in the drinking task, there was a tendency towards shorter latencies and, thus, an apparently longer consolidation period.

When Cherkin (1966) reanalyzed some prior results pertinent to estimating the length of the consolidation period (Chorover & Schiller, 1965; King, 1965; Quartermain and colleagues, 1965; Weissman, 1964), he concluded that the principal procedural differences among these experiments was in the strength of the learning before passive-avoidance training. However, two of these experiments involved a drinking task (King, 1965; Weissman, 1964) while two involved a step-down task. Therefore, Cherkin's conclusions can be reinterpreted like the results of Part 1 of the Passive-Avoidance Experiments: different apparent consolidation periods were observed because of differences in the number or strength of factors conducive to movement in the test situation. Similarly, the observation that apparent retro-

grade amnesia was observed only in animals which were unfamiliar with the experimental apparatus (Lewis and colleagues, 1968) can be reinterpreted according to the present proposal. Animals which are unfamiliar with an experimental apparatus may have a greater tendency to move than animals which are familiar with the apparatus (Claus & Bindra, 1960; Bindra & Claus, 1960). Therefore the combination of retrograde amnesia and non-familiarity, which are conducive to movement separately may have induced faster movement; in animals which were familiar with the experimental situation, the retrograde amnesia was too weak by itself to have this effect.

It is also possible to interpret certain procedures which apparently produced recovery from convulsion-induced retrograde amnesia as factors effectively conducive to immobility. These recovery-producing or immobility-conducive procedures have included a weak "reminder" footshock (Koppenaal and colleagues, 1967), testing with earclips on (Nielson, 1968), retesting the same animals (Herz & Peeke, 1967, 1968) and two footshocks rather than a single footshock (Schneider & Sherman, 1968). It is intuitively plausible that some of these treatments may have served to increase the freezing tendency in the test situations.

The practical proposal about convulsive effects, that these effects may be seen only when numerical results

stringently reflect actual movements, is more important than it may seem at first. For instance, in an unreported experiment on mice, the size and location of the platform in a step-down task determined in part the length of the step-down latencies observed. Larger platforms, and platforms placed against one wall or in a corner, produced much longer "spontaneous" step-down latencies than smaller platforms located in the middle of the grid floor. Larger platforms allowed movement to occur without actually affecting step-down latencies, and platforms not in the middle of the floor were simply more conducive to remaining still. A convulsion shortly after passive-avoidance training did not produce an apparent retrograde amnesia, except with a fairly small platform located in the middle of the grid floor. During the 30 sec. maximum latency used, both convulsed and non-convulsed animals remained on the larger or eccentrically-placed platforms. Such results are not unexpected according to the present proposal. With larger platforms, the step-down latencies simply failed to reflect the larger number of movements made by the convulsed animals. With the eccentrically-placed platforms, the apparatus was not conducive to movement even for the convulsed animals; in this case, more factors conducive to movement were required for any amnesia or reduced freezing to be manifested. From this analysis, it seems likely that the

differences between convulsed and non-convulsed animals would not have been masked in this experiment had the maximum latency been increased well beyond 30 sec. Extending the maximum latency may not just be a minor procedural variation, for long maximum latencies are more conducive to movement than short maximum latencies.

In this context, consider the two extremes in previous reports of the longest ECS-delay interval which induced retrograde amnesia. These extremes were the 10-sec. maximum effective interval found by Chorover and Schiller (1965) and the finding by Kopp and colleagues (1966) that a convulsion 6 hr. after passive-avoidance training in a two-compartment box induced retrograde amnesia. In this case, motivational differences cannot readily account for the difference in results since the two tasks appear to be similarly motivated. Kopp and colleagues (1966) felt that perhaps they found a long temporal gradient while Chorover and Schiller (1965) found only a short gradient because they used mice instead of rats or because they used a less intense punishing shock. Decreasing the intensity of punishing shock in training a passive-avoidance response allows retrograde amnesia to be produced at longer ECS-delay intervals (Ray & Bivens, 1968), and Kopp and colleagues (1966) used a punishing shock of about 0.32 mA

while Chorover and Schiller's (1965) shock was 0.75 mA.

The procedural difference pointed up by Kopp and colleagues (1966) may be a factor, but there is another procedural difference relevant to the present interpretation. Kopp and colleagues (1966) allowed a maximum latency in the test trial of 300 sec. whereas Chorover and Schiller (1965) allowed a maximum latency of only 30 sec. Moreover, if a 30-sec. maximum latency is applied to the published results of Kopp and colleagues (1966), then these results become very similar to those of Chorover and Schiller (1965). That is, if any group latency longer than 30 sec. reported by Kopp and colleagues is set at 30 sec., then it appears that the longest ECS-delay interval which would have produced latencies significantly shorter than the control latencies would have been less than 20 sec., rather than 6 hr.

This means that the convulsed animals may have had latencies which were shorter than those of non-convulsed animals in both experiments, but when the ECS-delay interval was greater than 10 or 15 sec., the convulsed animals still had latencies greater than 30 sec. Thus the combination of retrograde amnesia plus reduced freezing probably produces an apparent retrograde amnesia even at quite long ECS-delay intervals, but this effect can be masked by the use of shorter maximum latencies. Longer maximum latencies

allow relatively smaller differences between convulsed and non-convulsed animals to become evident, because a long maximum latency is conducive to movement, and short maximum latencies allow only very great differences to be shown.

It is difficult to relate Chorover and Schiller's (1965) procedure in the same way to other pertinent results (e.g., Heriot & Coleman, 1962; Weissman, 1964; Quartermain and colleagues, 1965) because these other results were not reported in terms of latencies. King's (1965) results are reported in terms of latencies but his task required the passive avoidance of drinking. Therefore, it is not surprising that he obtained longer amnesia-producing ECS-delay intervals than Chorover and Schiller (1965) obtained with a step-down passive-avoidance task, in the light of the results from Part 1 of the Passive-Avoidance Experiments. Even so, if King's (1965) reported latencies are set to a maximum of 30 sec., the longest ECS-delay intervals which might have produced latencies significantly different than control latencies would have been 75 sec. rather than the 300 sec. which he reported.

Therefore, Chorover and Schiller's (1965) results that a convulsion more than 10 sec. after training had no retrograde effect seems more a reflection of the 30-sec. maximum latency they used than of the "true" consolidation

process. It also seems likely that previous estimates of the consolidation period in general have been determined in part by the number and strength of experimental factors effectively conducive to movement and by the stringency with which the reported results reflected such movement.

Thus it may be possible to resolve many of the apparent differences among previous reports on retrograde amnesia in terms of the present proposal that the occurrence or apparent magnitude of retrograde amnesia depends upon the number of factors conducive to movement. This proposal was also supported by the results of the Multiple-Convulsions Experiments. In these experiments, animals first received either 5 convulsions or 5 pseudo-convulsions. Then they received a single training trial on either the escape or passive-avoidance tasks, followed by a single convulsion. A second trial was given 24 hr. after the first. The ECS-delay interval used with the passive-avoidance task was selected judiciously: with this ECS-delay interval of 12 sec., the animals which received pseudo-convulsions before training had latencies in the second trial which fell about midway between the latencies produced in this task by training but no convulsion and by convulsion but no training (see Table 8). It is likely that the impairment in freezing induced by a series of convulsions is cumulative:

at least, 16 convulsions impaired learning to inhibit an active-avoidance response while 8 convulsions did not have this effect (Delprato, 1966). If the freezing impairment and the retrograde amnesia induced by convulsions interact, then the 5 convulsions before training should inflate the apparent retrograde amnesia induced by the single convulsion after training. That is, according to the present proposal, animals which had convulsions before passive-avoidance training should have shorter latencies in the second trial than animals which had pseudo-convulsions before training. That was the result of Part 1 of the Multiple-Convulsions Experiments.

In the escape procedure used in Part 2, there was very little opportunity for freezing to occur, as pointed out before. The 5 convulsions therefore could not reduce freezing and, according to the present proposal, do not actually increase the retrograde amnesia. Thus the 5 convulsions before training should have no effect upon original escape learning, nor upon retention of this learning after a single convulsion. This was the result of Part 2 of the Multiple-Convulsions Experiments.

Previous work has demonstrated that a single convulsion after discrimination training can impair subsequent performance, and this impairment was greater in young

rats and rats with some neocortex removed (Thompson and colleagues, 1958). The investigators proposed that these results occurred because there were fewer functional neurons within the brains of either young or brain-damaged rats. This decreased the number of neurons modified during learning and thus effectively slowed down the consolidation process. Therefore, a convulsion at a particular ECS-delay interval produced a greater retrograde effect in young or cortically-damaged rats than in old or unoperated animals because fewer cells had been consolidated.

In the Multiple-Convulsions Experiments, it could be argued according to the proposal of Thompson and colleagues (1958) that the 5 convulsions before training likewise effectively slowed down the consolidation process and, for that reason, increased the retrograde effect of a single convulsion upon passive-avoidance training. However, the convulsions before training in the Multiple-Convulsions Experiments did not alter the negligible retrograde effect of a single convulsion upon escape training. This result seems to rule out the possibility that the convulsions beforehand generally retarded learning or slowed down the consolidation process. Rather, the results offer further support for the present proposal that observed convulsive effects reflect the joint outcome of factors in the experi-

mental situation conducive to movement and factors conducive to immobility or freezing.

Disinhibition

When Gellhorn (1943) found that convulsions tended to restore an active-avoidance response which had been experimentally extinguished, he attributed this effect to disinhibition in the Pavlovian sense of the term. Pavlov (1927) believed that novel stimuli were inhibitory since the presentation of novel stimuli during training, or trained performance, attenuated a conditioned salivary response. Pavlov called this phenomenon external inhibition. He also thought that experimental extinction occurred because the extinction procedure set up cortical process which he called internal inhibition. Therefore, when he found that the presentation of novel stimuli produced recovery of an extinguished response, Pavlov concluded that the novel stimuli must be inhibiting the internal inhibition. That is to say, he concluded that the novel stimuli produced what he called disinhibition.

Gellhorn (1943, 1945) concluded that convulsions disinhibited the active-avoidance response for similar reasons, but he believed that the disinhibitory effect was specific to the extinguished response, and was not a general effect upon movement. He suggested that the disinhibi-

tion was "due to increased hypothalamic discharges to the cortex, which ... may make subthreshold cortical processes supraliminal" (Gellhorn, 1946, p. 221).

Leaving the anatomy aside for the moment, Gellhorn's conclusion that convulsions exert a disinhibitory effect is extremely useful but, according to the present analysis, the convulsions disinhibit fear-induced immobility in general, not just a particular response. Gellhorn concluded that the disinhibition was specific to the extinguished avoidance response because the convulsions did not increase spontaneous activity, even though they restored the active-avoidance response. As present observations indicated (Multiple-Convulsions Experiments, Part 1) a series of convulsions may also have no detected effect upon spontaneous movements (latencies before passive-avoidance training) but still exert a profound effect on a trained passive-avoidance response. Similarly, in Delprato's (1966) report, a series of 16 convulsions had no observed effect upon one-way, active-avoidance learning but still seriously impaired learning to inhibit that response in a subsequent punishment-extinction procedure. All this does not mean that the convulsion's disinhibitory effect is specific to a particular response under investigation. It means, rather, that the effect of the convulsion is to decrease

fear-induced freezing or suppression of movement--no matter how the freezing or movement suppression is induced--without increasing the frequency or latency of movements in general.

This reinterpetation was also supported by the results of an attempt to confirm Gellhorn's results using a conditioned-suppression response (Geller & Brady, 1960). After conditioned suppression of drinking was extinguished, a series of 21 convulsions did not reinstate the conditioned suppression. This result indicates that convulsions do not simply reinstate extinguished responses. The result is quite understandable within the present proposal, however, since there was no freezing after extinction of the conditioned suppression and, therefore, the convulsions could not exert a disinhibitory effect.

Discrimination

The same general point that a convulsion has a general disinhibitory effect, must also be made with respect to Chorover and Schiller's (1966) proposals. As outlined in the Introduction, (p. 33) these investigators distinguished between convulsive effects upon discriminated responses and upon non-discriminated responses. They suggested that a convulsion produced retrograde amnesia only for discriminated learning. For other learning, "increased

exploratory activity, rather than retrograde amnesia, causes the apparent 'memory impairment'" which results from the convulsion after training. The logical prediction from this proposal is that discriminated responses should show effects from a convulsion after training which are graded relative to the ECS-delay interval while non-discriminated responses should show convulsive effects which are constant, or unrelated to the ECS-delay interval. Apart from the failure to experimentally validate this proposal (Passive-Avoidance Experiments; Kopp and colleagues, 1966), it is suggested in the present proposal that the distinction between discriminated (i.e. passive-avoidance) responses and generalized (i.e., conditioned-suppression) responses is irrelevant, at least to understanding the effects of electroconvulsive shock. Both types of responses involve the inhibition of movement, and part of the present proposal is that a convulsion reduces fear-induced inhibition of movement regardless of how it is produced.

The distinction between the passive-avoidance response and the conditioned-suppression response is defined operationally: for the passive-avoidance response, punishment is contingent upon a response while for the conditioned-suppression response, punishment is independent of response. In Spevack and Suboski's (1967) experiment, how-

ever, grid shock inhibited bar-pressing for water whether the shock was contingent upon bar-pressing or not, and a series of convulsions increased (disinhibited) bar-pressing in either case by about the same amount. In the present experiments, shock suppressed drinking whether it was administered through a grid floor (Drinking Experiments, Part 1) or through a water-bottle spout (Passive-Avoidance Experiments, Part 1) and, in both cases, a single convulsion afterwards disinhibited drinking in subsequent tests. In other previously published reports, the same amnesic effect was observed when a convulsion occurred shortly after training no matter whether the authors defined the trained response as "movement suppression" (e.g., Chevalier, 1965) or "passive avoidance" (e.g., Luttges & McGaugh, 1967) or "conditioned emotional response" (e.g., Kohlenberg & Trabasso, 1968). Thus, these operational distinctions do not seem to affect the results of a convulsion. From the animal's point of view, so to speak, the operational distinction between passive avoidance and conditioned suppression may not make much sense.

A more likely interpretation of these results is that a convulsion directly impairs mechanisms involved in withholding or inhibiting movements, and does not impair discrimination mechanisms. In any frightening situation

where an animal freezes, no matter whether the freezing is induced in a task operationally defined as "passive avoidance" or "active avoidance" or "conditioned suppression", a convulsion disinhibits the freezing in general. This seems the best systematic way to account for both prograde and retrograde effects of a convulsion. This may seem to be belaboring an obvious point. But the notion that a convulsion exerts a disinhibitory effect upon non-discriminated tasks and an amnesic effect upon discriminated tasks has had a great heuristic effect, and appears to be widely accepted at present. The present proposal precludes this distinction.

At the same time, there may be an element of truth in the suggestion that certain kinds of tasks, often involving discrimination learning, demonstrate retrograde amnesia more clearly. For instance, a convulsion induced retrograde amnesia for aversively-motivated brightness-discrimination training but not for aversively-motivated spatial-discrimination training (Thompson, 1958; Corson, 1965) nor for training in a shock-motivated T-maze (Gerbrandt and colleagues, 1968). These results probably mean that the weak retrograde amnesia induced by a single convulsion is manifested on complex tasks, since complex tasks would be expected to demonstrate even mild impairments

more readily than relatively simple tasks. A series of convulsions also produces a greater impairment for complex tasks than for simple tasks (Munn, 1950, p. 445). It is not that convulsions disrupt the memory for discriminated tasks but that the retrograde amnesia is more evidently manifested in tasks which are difficult for an animal to learn, and visual discriminations are difficult for a rat to learn. It is not contended in the present proposal that a single convulsion induces no retrograde amnesia, but only that in most tasks requiring active movement, this effect is masked by the concomitant disinhibitory effects of the convulsion. In passive-avoidance tasks, on the other hand, the observed retrograde amnesia has an "inflated" value because of this disinhibitory effect.

Estimating the Consolidation Period

The suggestion that the retrograde amnesia for passive-avoidance training appears inflated, however, does not necessarily mean that there is no retrograde amnesia when convulsed animals show poorer retention than non-convulsed animals. There seems to be a principle that, where two reports differ with respect to the longest ECS-delay interval which produces apparent amnesia, the shorter interval reported is a better reflection of the actual consolidation process. After Chorover and Schiller's (1965)

report, for instance, there was a tendency to conclude that the consolidation process was completed in a few seconds, and that reports of longer apparent retrograde amnesias were somehow due to experimental artifacts. Since it has been suggested that these "artifacts" may be fully as important in demonstrating convulsive effects as the convulsion itself, there seems no particularly sound reason for the principle that the "shorter report" is more valid. If anything, the contrary seems true.

Where the amnesic and disinhibitory effects are pitted against one another, as in active-avoidance tasks, little or no amnesia is observed; where these two effects work in concert with one another, as in passive-avoidance tasks, retrograde amnesia is prevalent. Similarly, a single convulsion does not produce an observed disinhibitory effect in a passive-avoidance task a day later, where it is virtually the only factor conducive to movement. But such a disinhibitory effect is readily apparent in active-avoidance learning a day later, where there are also other factors strongly conducive to movement. A disinhibitory effect can also be seen in a passive-avoidance task where other movement-conducive factors are present (such as greater disinhibition plus some amnesia: Multiple-Convulsions Experiments, Part 1).

As indicated before, these results considered collectively suggest that neither the amnesic nor the disinhibitory effect of a single convulsion is very significant by itself. A single convulsion therefore probably does not produce observable retrograde amnesia unless accompanied by another factor conducive to movement (except perhaps in very complex tasks). However, since the disinhibitory effect of a single convulsion also seems weak by itself, any latency decreases induced by a convulsion after passive-avoidance training may well reflect actual amnesia, as well as disinhibition. Both convulsive effects may be required since the disinhibition induced by a single convulsion is too weak by itself to produce apparent retrograde amnesia a day later. Nor can the occurrence of disinhibition easily account for latency effects which are graded with respect to increasing ECS-delay intervals.

There is, then, no particular reason to accept a short ECS-delay gradient as more "correct" or "true" than a long gradient. Where two reports differ on the estimated length of the consolidation period based upon the effects of increasing ECS-delay intervals, it makes just as much sense to conclude that the longer estimate is valid, since the shorter estimate may be based upon a procedure too insensitive to demonstrate long-term retrograde amnesia.

Less tortuous reasoning, however, suggests that it is impossible to make quantitative estimates of the length of the consolidation period solely from behavioral results of electroconvulsive shock. The number and variety of concomitant behavioral effects from convulsions seriously complicate interpretations of behavioral results. These complications probably make quantitative estimates of amnesic parameters so hazardous that they are not useful to an understanding of memory functions.

It may be possible, though, to experimentally separate amnesic effects from disinhibitory effects with procedures involving direct electrical stimulation of specific loci in the brain. The evidence on this point is still slight, and some other results must first be noted to make the point at all.

Neurological Considerations

The disinhibitory effect of convulsions may be due to damage to septal or hippocampal areas. Both convulsions (Vanderwolf, 1963b) and hippocampal damage (Isaacson, Douglas & Moore, 1961; Olton & Isaacson, 1968) facilitate active-avoidance learning in a shuttle box. Hippocampal damage, like electroconvulsive shock, also impairs passive avoidance (Kimura, 1958; Isaacson & Wickelgren, 1962;

McNew & Thompson, 1966) and learning of conditioned movement suppression (Brady, 1958). McCleary (1966) has also presented evidence that active and passive fear-motivated behaviours are controlled, in part, by different functional areas in the limbic system. This and other evidence (e.g., Vanderwolf, 1962, 1963a, 1964; Posluns, 1962) suggests that there are separate mechanisms in the brain for initiating movement and for inhibiting movement. The behavioral results of hippocampal damage just outlined, and other behavioral results of hippocampal damage (Brady, 1958; Teitelbaum & Milner, 1963), suggests that the hippocampal area is part of a system for inhibiting movement, so that hippocampal damage produces less inhibition in fearful situations.

On similar grounds, the medial thalamus has been implicated as part of a system for initiating movement (Vanderwolf, 1962, 1963a), and electroconvulsive shock partially offsets the effects of medial-thalamic damage (Vanderwolf, 1968). Medial thalamic damage seriously impairs performance in a one-way active-avoidance task, but this effect can be attenuated by a longer pre-shock interval or by a series of convulsions before training. Therefore, the disinhibitory effect of convulsions probably occurs because electroconvulsive shock damages neural structures involved

in a system for inhibiting movement. Such damage offsets, to some degree, more directly inflicted damage to neural structures involved in initiating movement, thereby producing the results observed by Vanderwolf (1968).

In particular, convulsions may induce disinhibitory effects by producing especially serious dysfunction of the hippocampal area, thereby mimicking the effects of surgically-produced hippocampal damage to some degree. The hippocampal area is extremely susceptible to seizure discharges (Gastaut & Fisher-Williams, 1959), and it has been suggested that such structures may be selectively depressed by electroconvulsive shock (French, Gernandt & Livingston, 1956) since they would be more reliably and thoroughly convulsed than other parts of the brain when electroconvulsive shock is given (Vanderwolf, 1963b).

The hippocampus has also been implicated in movement-producing mechanisms by observations that when an animal starts to make a trained (Vanderwolf & Heron, 1964) or "spontaneous" (Vanderwolf, 1969) movement, slow, rhythmical patterns of electrical activity occur in the hippocampus.

However, hippocampal damage also impairs consolidation mechanisms (Milner, 1966),as convulsions are reputed to do. For example, as pointed out earlier (p. 8), Russell

(1948) felt that penetrating brain wounds produced retrograde amnesia only when the temporal lobes were injured, and Williams (1966) had the clinical impression that the amnesia produced by electroconvulsive therapy was very much like that seen in cases of brain disease involving the hippocampal region. Thus, the similar amnesic effects of convulsions and hippocampal damage suggests that the amnesic effect of convulsions, as well as the disinhibitory effect, occurs through hippocampal dysfunction.

This interpretation may be accurate for humans, but there is no very convincing evidence that hippocampal damage in the rat produces retrograde amnesia. However, to explain two sets of results, the suggestion has been made that hippocampal damage does produce retrograde amnesia in the rat. First, hippocampal damage reduced spontaneous alternation and increased exploration in the rat (Dember, Brodwick & Roberts, 1960) and, second, hippocampal damage prevented rats from reversal learning in a shock-motivated T-maze if the intertrial interval was 30 min. but not if the intertrial interval was 30 sec. (Thompson, Langer & Rich, 1964). These results, however, are open to other interpretation. First, no operated control animals were tested for comparison with the hippocampal damage, so that attributing any deficits specifically to hippocampal damage

is highly speculative. In fact, Thompson and colleagues (1964) apparently observed no differences in T-maze reversal performance among rats with hippocampal damage, damage to the mammillary bodies and damage to the mammillio-thalamic tract. Second, these results can also be interpreted as a result of increased perseveration rather than impaired consolidation or short-term memory, as Kimble (1963, p. 282) suggested to explain his similar observations.

Therefore, the suggestion that hippocampal damage in rats produces impairment of memory is not very persuasive. There is, at the same time, evidence that direct electrical stimulation of the amygdala produces retrograde amnesia in the rat (Goddard, 1964a, 1964b) and the cat (Kesner & Doty, 1968). In the rat, Goddard (1964a) reported that continuous low-intensity stimulation of the amygdala interfered with active-avoidance learning and with learning conditioned movement suppression, if the stimulation occurred just after the presentation of the unconditioned stimulus. Similar electrical stimulation did not affect food-motivated learning in a Lashley III maze.

Kesner and Doty (1968) trained cats to eat in a box, and then established a passive-avoidance response with a single shock through the mouth. Four sec. later, afterdischarges were elicited in some animals by direct stimulation of loci in the limbic system. Afterdischarges

from stimulation of the amygdala abolished the passive-avoidance response, although afterdischarges from stimulation in the ventral hippocampus, fornix or septum had no effect upon subsequent passive-avoidance performance, even if the afterdischarges spread to the dorsal hippocampus. Afterdischarges from stimulation of the dorsal hippocampus itself produced retrograde amnesia in only 8 of 14 animals, and then only if intense stimulation was used. The investigators believed, partly on empirical grounds, that amnesia occurred because these afterdischarges spread to the amygdala.

Incidentally, Kesner and Doty (1968) also confirmed previous observations (e.g., McGaugh & Alpern, 1966) that motor convulsions were not necessary to produce retrograde amnesia since the direct stimulation of the limbic system produced no motor seizures, and since electroconvulsive shock applied across the frontal bone produced motor convulsions but no retrograde amnesia. From their observations, Kesner and Doty (1968) naturally concluded that the amygdala seems to have a critical role in memory, at least for the passive-avoidance response, but the hippocampus probably does not.

Therefore, although the amnesic and disinhibitory effects of electroconvulsive shock may not be separable

with behavioral tests, these effects might be separable with procedures involving direct electrical stimulation of selected loci in the limbic system.

Conclusions

A single convulsion slightly weakens or impairs the tendency for frightened animals to freeze or inhibit movement. This conclusion was supported by the finding that a single convulsion facilitated the acquisition of an active-avoidance response.

A single convulsion also induces a weak retrograde amnesia, or slight loss of memory, for events preceding the convulsion. A result favoring this conclusion was that an exploratory period the day before training followed by a convulsion facilitated active-avoidance learning more than a convulsion alone, even though the exploratory period by itself had an effect which was too weak to affect active-avoidance learning. Therefore, the amnesic effect of the convulsion seemed slight. Moreover, a convulsion immediately after escape training induced no detectable retrograde amnesia for that experience.

The period of time preceding the convulsion for which actual retrograde amnesia is induced is difficult to estimate with behavioral tests because the concomitant impairment of freezing produced by the convulsion determines,

in part, the manifestation of any retrograde amnesia. This proposal was supported by the finding that a single convulsion shortly after learning induced apparent retrograde amnesia for passive-avoidance training, but not for escape training or active-avoidance training.

As a more general principle, the manifestation of either the reduced freezing (disinhibition) or retrograde amnesia produced by a convulsion probably depends upon the balance between the number and strength of other factors conducive to movement or conducive to immobility in a test situation. This proposal was supported by two major results. First, a convulsion induced apparent retrograde amnesia for passive-avoidance of drinking after longer ECS-delay intervals than for passive-avoidance of stepping off a small platform. This difference can be understood if the water deprivation used in the drinking task is seen as a factor conducive to movement. Second, a series of convulsions before training increased the retrograde effect of a convulsion after training in a passive-avoidance task, but not in an escape task. This was interpreted to mean that the convulsions before training effectively increased the factors conducive to movement in the passive-avoidance situation, but these factors were already maximal in the escape task (where the weak amnesia was the only factor not effectively conducive to movement).

As a practical consideration, the behavioral manifestation of convulsions also depends upon how stringently the numerical results of behavioral tests reflect the actual movement in the test situation. Thus, apparently minor alterations in experimental apparatus and test parameters can alter the occurrence and apparent severity of amnesic and disinhibitory effects of a convulsion. This consideration explains several observations in the present investigation and also accounts for certain discrepancies among previous reports on the longest ECS-delay interval which produced retrograde amnesia.

Summary

Previous results have indicated that electroconvulsive shock which occurs after an experience produces a loss of memory, or induces retrograde amnesia, for the experience. However, other concomitant effects of electroconvulsive shock seriously complicate the interpretation of behavioral results observed after convulsions. The results of the present investigation indicate in particular that the retrograde amnesia is probably slight in animals but appears enhanced in passive-avoidance tasks and diminished in aversively-motivated tasks which require movement because of a concomitant impairment in movement-inhibiting or freezing mechanisms. This latter effect is called

"disinhibition." Evidence supporting this interpretation can be found in clinical reports, and in previous reports of animal experiments, as well as in the results of the present investigation. If this interpretation is correct, then it is extremely difficult to make reasonable quantitative estimates of the severity or temporal extent of the retrograde amnesia induced by electroconvulsive shock on the basis of behavioral tests alone. Such tests invariably involve observation or measurement of movement, and thus reflect the disinhibitory effect, as well as the amnesic effect, of electroconvulsive shock. However, the results of other experiments allow the retrograde amnesia to be interpreted as a result of amygdaloid dysfunction, and the disinhibition to be interpreted as a result of hippocampal and, perhaps, septal dysfunction. Therefore, it may prove possible to separate memory functions and movement-inhibiting functions in animals with procedures involving more precisely localized effects upon the brain.

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APPENDICES

The appendices contain the individual results obtained in the present experiments, which are summarized in Tables 1-12.

APPENDIX A

Number of shocks received in reaching 90% criterion in Part 1 of the Avoidance Experiments.

No Convulsion/ No Exploration (n=14)	Convulsion/ No Exploration (n=16)	No Convulsion/ Exploration (n=15)	Convulsion/ Exploration (n=11)
5	3	3	3
6	4	3	3
6	4	3	4
8	4	4	4
8	4	4	4
8	5	4	4
9	5	4	5
9	5	5	6
9	5	5	6
9	6	6	7
10	6	6	9
10	6	6	
11	6	7	
16	7	8	
	7	9	
	7		
124.	84	77.	55.
$\bar{X}$ 8.86	5.25	5.13	5.00

APPENDIX B

Number of shocks received in reaching 90% criterion in Part 2 of the Avoidance Experiments.

PSEUDO-CONVULSION

24 hr. between sessions (n=15)

First Session	Second Session	Change
3	1	-2
3	2	-1
4	1	-3
5	1	-4
5	1	-4
5	2	-3
5	3	-2
6	2	-4
7	2	-5
7	3	-4
7	5	-2
7	5	-2
8	2	-6
8	10	2
9	2	-7
89	42	-47
$\bar{X}$ 5.93	2.80	-3.13

(appendix continues)

APPENDIX B (continued)

Number of shocks received in reaching 90% criterion in Part 2 of the Avoidance Experiments.

0.3-SEC. CONVULSION

24 hr. between sessions (n=18)

First Session	Second Session	Change
3	1	-2
3	3	0
4	5	1
4	5	1
5	2	-3
5	2	-3
5	3	-2
6	1	-5
6	1	-5
7	1	-6
7	2	-5
7	2	-5
7	2	-5
7	18	11
8	1	-7
8	2	-6

(appendix continues)

APPENDIX B (continued)

Number of shocks received in reaching 90% criterion in Part 2 of the Avoidance Experiments.

0.3-SEC. CONVULSION

24 hr. between sessions (n=18)

First Session	Second Session	Change
8	3	-5
9	2	-7
109	56	-53
$\bar{X}$ 6.06	3.11	-2.94

0.6-SEC. CONVULSION

24 hr. between sessions (n=18)

First Session	Second Session	Change
3	2	-1
3	3	0
4	2	-2
4	2	-2
5	2	-3
5	2	-3
5	2	-3
5	3	-2
5	3	-2
5	3	-2

(appendix continues)

APPENDIX B (continued)

Number of shocks received in reaching 90% criterion in Part 2 of the Avoidance Experiments.

0.6-SEC. CONVULSION

24 hr. between sessions (n=18)

First Session	Second Session	Change
6	2	-4
6	2	-4
6	3	-3
7	3	-4
7	4	-3
7	4	-3
8	4	-4
9	5	-4
100	51	-49
$\bar{X}$ 5.55	2.83	-2.72

(appendix continues)

APPENDIX B (continued)

Number of shocks received in reaching 90% criterion in Part 2 of the Avoidance Experiments.

0.3-SEC. CONVULSION

50 hr. between sessions (n=20)

First Session	Second Session	Change
2	1	-1
3	1	-2
3	2	-1
3	3	0
4	5	1
5	1	-4
5	2	-3
5	2	-3
5	3	-2
6	2	-4
6	3	-3
6	4	-2
6	4	-2
6	7	1
7	11	4
8	5	-3

(appendix continues)

APPENDIX B (continued)

Number of shocks received in reaching 90% criterion in Part 2 of the Avoidance Experiments.

0.3-SEC. CONVULSION

50 hr. between sessions (n=20)

First Session	Second Session	Change
9	2	-7
9	4	-5
10	7	-3
10	3	-7
118	72	-46
$\bar{X}$ 5.90	3.60	-2.30

2.0-SEC. CONVULSION

50 hr. between sessions (n=11)

First Session	Second Session	Change
2	2	0
3	2	-1
3	4	1
3	8	5
4	1	-3
5	1	-4

(appendix continues)

APPENDIX B (continued)

Number of shocks received in reaching 90% criterion in Part 2 of the Avoidance Experiments.

2.0-SEC. CONVULSION

50 hr. between sessions (n=11)

First Session	Second Session	Change
5	3	-2
6	2	-4
6	2	-4
8	2	-6
10	3	-7
55	30	-25
$\bar{X}$ 5.00	2.73	-2.27

APPENDIX C

Number of shocks received in reaching 90% criterion in Part 3 of the Avoidance Experiments.

CONVULSED (n=10)	NONCONVULSED (n=14)
2	2
3	5
3	6
3	6
3	7
4	8
4	9
4	9
6	9
6	10
	10
	12
	14
	14
38	121
$\bar{X}$ 3.80	8.64

APPENDIX D

Jump latencies in Part 1 of the Escape Experiments.

SPECIFIC-TRAINING GROUP
(n=16)

First Trial	Second Trial	Change
1.5	7.5	6.0
5.5	4.0	-1.5
5.5	1.0	-4.5
7.0	1.5	-5.5
7.0	1.0	-6.0
10.0	2.0	-8.0
11.0	0.5	-10.5
11.5	3.5	-8.0
12.5	2.0	-10.5
13.0	2.0	-11.0
13.0	0.5	-12.5
15.0	1.5	-13.5
19.0	1.5	-17.5
20.0	1.0	-19.0
25.0	2.0	-23.0
43.0	2.5	-41.5
219.5	34.0	-185.5
$\bar{X}$ 13.72	2.13	-11.59

(appendix continues)

APPENDIX D (continued)

Jump latencies in Part 1 of the Escape Experiments.

NO-SHOCK GROUP (n=15)	WOOD-TOP GROUP (n=13)	PLASTIC-TOP GROUP (n=15)
2.0	1.5	3.5
4.5	2.0	5.0
5.0	3.0	8.0
6.0	4.0	9.0
7.0	5.5	10.0
7.0	11.0	10.5
10.0	12.0	13.0
11.0	17.0	14.0
19.0	18.0	17.0
25.0	18.5	18.0
26.0	35.0	21.0
29.0	42.0	26.0
35.0	44.0	28.0
37.0		38.0
41.0		43.0
264.5	213.5	264.0
$\bar{X}$ 17.63	16.42	17.60

(appendix continues)

APPENDIX D (continued)

Jump latencies in Part 1 of the Escape Experiments.

CONVULSED GROUP (n=13)	NO-EXPLORATION GROUP (n=15)
3.5	2.0
4.0	2.0
9.0	7.0
9.0	7.0
9.5	8.0
11.0	8.5
13.0	9.0
13.0	10.0
14.5	17.0
16.5	17.5
18.0	21.0
30.0	25.0
40.5	26.0
	32.0
	35.0
191.5	227.0
$\bar{X}$ 14.73	15.13

APPENDIX E

Jump latencies in Part 2 of the Escape Experiments.

30-SEC. CONVULSION GROUP (n=18)			30-SEC. CONTROL GROUP (n=15)		
First Trial	Second Trial	Change	First Trial	Second Trial	Change
3.0	1.0	-2.0	3.0	1.0	-2.0
3.5	1.5	-2.0	4.0	2.5	-1.5
4.0	3.0	-1.0	5.0	3.0	-2.0
4.5	3.0	-1.5	6.5	2.0	-4.5
6.0	1.0	-5.0	7.0	2.0	-5.0
6.0	2.0	-4.0	7.5	4.0	-3.5
6.5	2.0	-4.5	11.0	2.0	-9.0
7.5	4.0	-3.5	11.0	5.0	-6.0
10.0	3.0	-7.0	13.0	3.0	-10.0
10.5	3.5	-7.0	15.5	3.5	-12.0
11.0	2.0	-9.0	16.0	2.0	-14.0
16.0	6.0	-10.0	20.0	2.5	-17.5
17.5	3.0	-14.5	24.5	1.5	-23.0
19.0	2.0	-17.0	25.0	9.0	-16.0
23.0	6.0	-17.0	33.5	1.0	-32.5
26.5	2.0	-24.5			
33.5	4.0	-29.5			
40.0	1.5	-38.5			
248.0	50.5	-197.5	202.5	44.0	-158.5
$\bar{X}$ 13.78	2.81	-10.97	13.50	2.93	-10.57

(appendix continues)

APPENDIX E (continued)

Jump latencies in Part 2 of the Escape Experiments.

1-SEC. CONVULSION GROUP (n=16)			1-SEC. CONTROL GROUP (n=16)		
First Trial	Second Trial	Change	First Trial	Second Trial	Change
5.0	5.0	0.0	3.5	1.0	-2.5
6.0	3.0	-3.0	3.5	2.0	-1.5
6.0	5.0	-1.0	5.0	1.0	-4.0
8.0	3.5	-4.5	6.0	5.5	-0.5
9.0	3.0	-6.0	6.5	4.5	-2.0
10.0	3.5	-6.5	7.0	6.0	-1.0
11.0	3.0	-8.0	9.0	2.0	-7.0
13.5	4.0	-9.5	12.0	1.5	-10.5
15.0	4.0	-11.0	13.0	2.0	-11.0
17.5	3.0	-14.5	13.0	2.0	-11.0
18.0	4.0	-14.0	14.0	1.5	-12.5
18.5	3.0	-15.5	15.0	4.0	-11.0
21.0	4.0	-17.0	18.0	5.0	-13.0
21.0	6.5	-14.5	31.0	9.0	-22.0
27.5	2.5	-25.0	34.0	2.5	-31.5
28.0	3.0	-25.0	38.0	4.0	-34.0
235.0	60.0	-175.0	228.5	53.5	-175.0
$\bar{X}$ 14.69	3.75	-10.94	14.28	3.34	-10.94

APPENDIX F

Latencies of drinking in Part 1 of the Drinking Experiments.

	CONVULSED GROUP (n=11)			PSEUDO-CONVULSED GROUP (n=12)		
	Before Shock	After Shock	Change	Before Shock	After Shock	Change
	1.0	1.0	0.0	1.0	30.0	29.0
	1.0	1.5	0.5	1.5	30.0	28.5
	1.0	3.0	2.0	1.5	30.0	28.5
	2.0	1.0	-1.0	1.5	30.0	28.5
	2.0	4.0	2.0	2.0	30.0	28.0
	2.0	30.0	28.0	2.0	30.0	28.0
	2.5	2.0	-0.5	2.0	30.0	28.0
	3.0	5.5	2.5	2.0	30.0	28.0
	3.5	4.0	0.5	3.0	30.0	27.0
	4.0	5.0	1.0	3.0	30.0	27.0
	4.0	30.0	26.0	4.0	30.0	26.0
				4.0	30.0	26.0
	26.0	87.0	61.0	27.5	330.0	302.5
$\bar{X}$	2.36	7.91	5.55	2.29	30.0	27.71

APPENDIX G

Jump latencies after drinking training in Part 2 of the Drinking Experiments.

CONVULSED GROUP (n=16)			PSEUDO-CONVULSED GROUP (n=16)		
First Trial	Second Trial	Change	First Trial	Second Trial	Change
4.0	1.0	-3.0	5.0	3.0	-2.0
7.0	6.0	-1.0	10.0	3.0	-7.0
11.0	3.0	-8.0	12.0	3.5	-8.5
15.0	4.0	-11.0	12.0	18.0	6.0
15.0	4.5	-10.5	14.5	1.5	-13.0
15.5	4.0	-11.5	19.0	3.0	-16.0
19.0	3.5	-15.5	19.5	16.5	-3.0
19.0	14.0	-5.0	21.5	12.0	-9.5
19.0	26.0	7.0	21.5	16.5	-5.0
22.0	8.0	-14.0	23.5	2.5	-21.0
22.5	11.0	-11.5	24.0	6.5	-17.5
23.5	4.0	-19.5	27.0	3.0	-24.0
29.5	1.5	-28.0	32.5	4.0	-28.5
36.0	2.5	-33.5	43.0	13.5	-29.5
41.0	16.5	-24.5	44.0	4.0	-40.0
44.5	5.0	-39.5	44.0	5.5	-38.5
343.5	114.5	-229.0	373.0	116.0	-257.0
$\bar{X}$ 21.47	7.16	-14.31	23.31	7.25	-16.06

APPENDIX H

Latencies in the step-down and drinking passive-avoidance tasks in Part 1 of the Passive-Avoidance Experiments.

STEP-DOWN TASK

No-Training Group
(n=17)

Before Shock	After Shock	Change
1.0	0.5	-0.5
1.0	0.5	-0.5
1.0	1.0	0.0
1.0	1.0	0.0
1.0	1.0	0.0
1.0	1.0	0.0
0.5	0.5	0.0
0.5	0.5	0.0
0.5	0.5	0.0
0.5	0.5	0.0
0.5	0.5	0.0
0.5	0.5	0.0
0.5	0.5	0.0
0.5	0.5	0.0
0.5	0.5	0.0
0.5	1.0	0.5
1.0	5.5	4.5
12.0	16.0	4.0
$\bar{X}$ 0.71	0.94	.23

(appendix continues)

APPENDIX H (continued)

Latencies in the step-down and drinking passive-avoidance tasks in Part 1 of the Passive-Avoidance Experiments.

STEP-DOWN TASK

1-sec. Group
(n=20)

Before Shock	After Shock	Change
3.0	0.5	-2.5
1.0	0.5	-0.5
1.0	0.5	-0.5
1.0	1.0	0.0
1.0	1.0	0.0
1.0	1.0	0.0
1.0	1.0	0.0
0.5	0.5	0.0
0.5	0.5	0.0
0.5	0.5	0.0
0.5	0.5	0.0
0.5	0.5	0.0
0.5	0.5	0.0
0.5	0.5	0.0
0.5	0.5	0.0
1.0	1.5	0.5
0.5	3.0	2.5
1.0	4.0	3.0
0.5	9.0	8.5
1.0	22.5	21.5
17.0	49.5	32.5
$\bar{X}$ 0.85	2.48	1.63

(appendix continues)

APPENDIX H (continued)

Latencies in the step-down and drinking passive-avoidance tasks in Part 1 of the Passive-Avoidance Experiments.

STEP-DOWN TASK

10-sec. Group
(n=17)

Before Shock	After Shock	Change
1.0	1.0	0.0
0.5	0.5	0.0
0.5	0.5	0.0
1.0	1.5	0.5
0.5	1.0	0.5
0.5	1.0	0.5
1.0	2.0	1.0
0.5	1.5	1.0
0.5	2.5	2.0
0.5	3.0	2.5
0.5	5.0	4.5
0.5	5.5	5.0
0.5	9.0	8.5
1.5	18.0	16.5
1.0	25.0	24.0
1.5	30.0	28.5
1.5	30.0	28.5
13.5	137.0	123.5
$\bar{X}$ 0.79	8.06	7.27

(appendix continues)

APPENDIX H (continued)

Latencies in the step-down and drinking passive-avoidance tasks in Part 1 of the Passive-Avoidance Experiments.

STEP-DOWN TASK

30-sec. Group
(n=15)

	Before Shock	After Shock	Change
	4.0	10.0	6.0
	0.5	25.0	24.5
	1.0	30.0	29.0
	1.0	30.0	29.0
	1.0	30.0	29.0
	1.0	30.0	29.0
	0.5	30.0	29.5
	0.5	30.0	29.5
	0.5	30.0	29.5
	0.5	30.0	29.5
	0.5	30.0	29.5
	0.5	30.0	29.5
	0.5	30.0	29.5
	0.5	30.0	29.5
	0.5	30.0	29.5
	13.0	425.0	412.0
$\bar{X}$	0.87	28.33	27.47

(appendix continues)

APPENDIX H (continued)

Latencies in the step-down a nd drinking passive-avoidance tasks in Part 1 of the Passive-Avoidance Experiments.

STEP-DOWN TASK

60-sec. Group
(n=12)

	Before Shock	After Shock	Change
	0.5	12.0	11.5
	2.0	30.0	28.0
	1.0	30.0	29.0
	1.0	30.0	29.0
	0.5	30.0	29.5
	0.5	30.0	29.5
	0.5	30.0	29.5
	0.5	30.0	29.5
	0.5	30.0	29.5
	0.5	30.0	29.5
	0.5	30.0	29.5
	0.5	30.0	29.5
	8.5	342.0	333.5
$\bar{X}$	0.71	28.5	27.79

(appendix continues)

APPENDIX H (continued)

Latencies in the step-down and drinking passive-avoidance tasks in Part 1 of the Passive-Avoidance Experiments.

STEP-DOWN TASK

Pseudo-Convulsion Group
(n=18)

	Before Shock	After Shock	Change
	0.5	16.0	15.5
	1.0	17.5	16.5
	0.5	26.5	26.0
	3.0	30.0	27.0
	2.0	30.0	28.0
	1.0	30.0	29.0
	1.0	30.0	29.0
	1.0	30.0	29.0
	1.0	30.0	29.0
	1.0	30.0	29.0
	1.0	30.0	29.0
	0.5	30.0	29.5
	0.5	30.0	29.5
	0.5	30.0	29.5
	0.5	30.0	29.5
	0.5	30.0	29.5
	0.5	30.0	29.5
	0.5	30.0	29.5
	16.5	510.0	493.5
$\overline{X}$	0.92	28.33	27.42

(appendix continues)

APPENDIX H (continued)

Latencies in the step-down and drinking passive-avoidance tasks in Part 1 of the Passive-Avoidance Experiments.

DRINKING TASK

No-Training Group
(n=17)

	Before Shock	After Shock	Change
	3.0	1.5	-1.5
	2.0	1.5	-0.5
	1.5	1.0	-0.5
	1.0	0.5	-0.5
	3.0	3.0	0.0
	2.0	2.0	0.0
	2.0	2.0	0.0
	1.5	1.5	0.0
	1.5	1.5	0.0
	1.0	1.0	0.0
	1.0	1.0	0.0
	1.0	1.0	0.0
	1.0	1.0	0.0
	1.0	1.0	0.0
	1.5	2.0	0.5
	1.5	2.0	0.5
	1.5	3.0	1.5
	27.0	26.5	-0.5
$\bar{X}$	1.59	1.56	-0.03

(appendix continues)

APPENDIX H (continued)

Latencies in the step-down and drinking passive-avoidance tasks in Part 1 of the Passive-Avoidance Experiments.

DRINKING TASK

1-sec. Group
(n=17)

Before Shock	After Shock	Change
3.0	2.0	-1.0
2.0	1.0	-1.0
2.5	2.0	-0.5
2.5	2.0	-0.5
2.0	1.5	-0.5
1.5	1.0	-0.5
3.0	3.0	0.0
2.5	2.5	0.0
2.0	2.0	0.0
2.0	2.0	0.0
2.0	2.0	0.0
1.5	2.0	0.5
2.5	4.5	2.0
1.0	3.0	2.0
1.0	3.0	2.0
2.5	5.0	2.5
2.5	5.0	2.5
36.0	43.5	7.5
$\bar{X}$ 2.12	2.56	0.44

(appendix continues)

APPENDIX H (continued)

Latencies in the step-down and drinking passive-avoidance tasks in Part 1 of the Passive-Avoidance Experiments.

DRINKING TASK

30-sec. Group
(n=18)

Before Shock	After Shock	Change
2.0	1.0	-1.0
3.0	2.5	-0.5
2.5	2.0	-0.5
1.5	1.0	-0.5
3.0	3.0	0.0
2.0	2.0	0.0
1.0	1.0	0.0
2.0	2.5	0.5
1.5	2.0	0.5
1.5	2.0	0.5
1.0	3.0	2.0
2.5	5.0	2.5
1.5	8.0	6.5
1.0	16.0	15.0
1.0	20.0	19.0
1.0	25.0	24.0
3.5	30.0	26.5
1.0	30.0	29.0
32.5	156.0	123.5
$\bar{X}$ 1.81	8.67	6.86

(appendix continues)

APPENDIX H (continued)

Latencies in the step-down and drinking passive-avoidance tasks in Part 1 of the Passive-Avoidance Experiments.

DRINKING TASK

60-sec. Group
(n=17)

Before Shock	After Shock	Change
1.5	3.0	1.5
2.5	5.0	2.5
1.0	9.0	8.0
2.0	11.0	9.0
1.0	14.0	13.0
2.0	16.5	14.5
1.5	18.0	16.5
2.0	19.0	17.0
1.5	20.0	18.5
1.5	24.0	22.5
2.0	26.5	24.5
3.0	30.0	27.0
2.5	30.0	27.5
2.5	30.0	27.5
2.0	30.0	28.0
1.5	30.0	28.5
1.0	30.0	29.0
31.0	326.0	315.0
$\bar{X}$ 1.82	20.35	18.53

(appendix continues)

APPENDIX H (continued)

Latencies in the step-down and drinking passive-avoidance tasks in Part 1 of the Passive-Avoidance Experiments.

DRINKING TASK

300-sec. Group
(n=17)

Before Shock	After Shock	Change
1.0	3.5	2.5
1.0	5.0	4.0
2.0	16.0	14.0
1.0	23.0	22.0
1.5	27.0	25.5
2.0	28.0	26.0
4.0	30.0	26.0
3.0	30.0	27.0
2.5	30.0	27.5
2.0	30.0	28.0
2.0	30.0	28.0
1.5	30.0	28.5
1.5	30.0	28.5
1.0	30.0	29.0
1.0	30.0	29.0
1.0	30.0	29.0
1.0	30.0	29.0
29.0	432.5	403.5
$\bar{X}$ 1.71	25.44	23.74

(appendix continues)

APPENDIX H (continued)

Latencies in the step-down and drinking passive-avoidance tasks in Part 1 of the Passive-Avoidance Experiments.

DRINKING TASK

Pseduo-Convulsion Group
(n=18)

Before Shock	After Shock	Change
1.0	16.5	15.5
2.0	21.0	19.0
2.5	29.0	26.5
3.0	30.0	27.0
3.0	30.0	27.0
2.5	30.0	27.5
2.0	30.0	28.0
2.0	30.0	28.0
2.0	30.0	28.0
2.0	30.0	28.0
2.0	30.0	28.0
1.5	30.0	28.5
1.5	30.0	28.5
1.0	30.0	29.0
1.0	30.0	29.0
1.0	30.0	29.0
1.0	30.0	29.0
1.0	30.0	29.0
32.0	516.5	484.5
$\bar{X}$ 1.78	28.69	26.92

APPENDIX I

Test latencies for the three groups in Part 2 of the Passive-Avoidance Experiments.

Group 1 (n=12)	Group 2 (n=11)	Group 3 (n=12)
14.0	16.0	22.0
24.0	18.0	24.0
25.0	23.5	26.0
25.0	24.5	28.0
26.0	30.0	30.0
30.0	30.0	30.0
30.0	30.0	30.0
30.0	30.0	30.0
30.0	30.0	30.0
30.0	30.0	30.0
30.0	30.0	30.0
30.0		30.0
324.0	292.0	340.0
$\bar{X}$ 27.0	26.6	28.3

APPENDIX J

Step-down latencies in Part 1 of the Multiple-Convulsions Experiments.

CONVULSED GROUP (n=10)			PSEUDO-CONVULSED GROUP (n=10)		
Before Avoidance Training	After Avoidance Training	Change	Before Avoidance Training	After Avoidance Training	Change
0.5	0.5	0.0	0.5	1.5	1.0
0.5	0.5	0.0	0.5	1.5	1.0
0.5	0.5	0.0	0.5	4.0	3.5
1.0	1.0	0.0	1.0	6.5	5.5
0.5	1.0	0.5	0.5	10.0	9.5
0.5	1.0	0.5	3.0	19.0	16.0
1.0	3.0	2.0	3.0	30.0	27.0
0.5	5.5	5.0	1.0	30.0	29.0
0.5	9.0	8.5	0.5	30.0	29.5
0.5	30.0	29.5	0.5	30.0	29.5
6.0	52.0	46.0	11.0	162.5	151.5
$\bar{X}$ 0.60	5.20	4.60	1.10	16.25	15.15

APPENDIX K

Jump-Escape Latencies in Part 2 of the Multiple-Convulsions Experiment.

CONVULSED GROUP (n=12)			PSEUDO-CONVULSED GROUP (n=15)		
First Trial	Second Trial	Change	First Trial	Second Trial	Change
1.0	4.0	3.0	5.0	4.0	-1.0
4.0	2.0	-2.0	4.0	3.0	-1.0
5.5	1.0	-4.5	5.0	3.5	-1.5
5.5	0.5	-5.0	9.0	6.5	-2.5
15.5	9.5	-6.0	9.0	2.5	-6.5
12.0	1.5	-10.5	12.5	4.0	-8.5
14.5	3.5	-11.0	16.5	7.0	-9.5
24.0	8.0	-16.0	18.0	6.0	-12.0
31.5	4.5	-27.0	25.5	8.0	-17.5
43.0	5.5	-37.5	19.0	1.0	-18.0
41.5	2.0	-39.5	19.5	1.5	-18.0
44.0	2.0	-42.0	36.0	15.5	-20.5
			41.0	13.5	-27.5
			37.0	3.5	-33.5
			44.5	8.0	-36.5
242.0	44.0	-198.0	301.5	87.5	-214.0
$\bar{X}$ 20.11	3.67	-16.50	20.10	5.83	-14.27