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THE EFFECT OF ELECTROSHOCK CONVULSIONS ON LEARNING AND RETENTION IN THE RAT.

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I. Introduction

The treatment of mental disorder by the methods known as 'shock therapies' has had widespread adoption since the methods were first introduced. In 1928 Sakel began to treat patients with hypoglycemia produced by insulin injection. In 1934 von Meduna produced convulsions by injecting metrazol. In 1938 Cerletti and Bini employed the passage of electricity through the head to induce the convulsive attack (48). Other methods, such as the use of coramine, ammonium chloride, nitrogen inhalation, electronarcosis, etc., have been used occasionally, but insulin, metrazol, and electroshock remain by far the most widely accepted methods of shock treatment.

In spite of the generic name shock therapies, there are some differences among the three methods. Insulin and metrazol are pharmacological agents administered by injection, while electricity is a physical stimulus applied by placing electrodes on the head. Experience with a large number of patients indicates that insulin is somewhat more effective for schizophrenia, while metrazol and electricity have shown better results with the affective disorders (14). Many therapists believe that it is not necessary to prolong the insulin coma until the patient has convulsions. Nearly all workers are agreed, however, that the major motor attack must accompany the administration of metrazol or electricity for best therapeutic results. The somatic and therapeutic effects of metrazol and electricity seem to be very similar. Since electricity has a number of advantages over metrazol, it has rather generally replaced the drug. In some hospitals it has also replaced insulin. Electroshock, therefore, is now the most widely

used organic therapy in psychiatry (14).

The results with shock therapies have been encouraging, but there are some disadvantages to their use. Some patients show fear of the treatment and others have had bones fractured by the convulsion. Memory impairment is often observed as a complication of the treatment. In this paper we have chosen this memory complication for study. Psychiatrists concerned with shock therapy have often assumed that the mechanism (still largely unknown) whereby shock treatment produces relief from symptoms bears little relation to the disturbance of the memory function. As the following review of the literature will show, however, some writers hold that improvement through shock therapy may be basically an amnesia for recent events. In view of this hypothesis it seems valuable to investigate the memory impairment produced by one of the major types of shock treatment. Because of its widespread adoption, cerebral electroshock was chosen for study. Specifically, the present experiments are concerned with the severity of the amnesia for a learned response when the shock is applied to rats at different times following the response.

Two groups of experiments will be reported. In the first group it will be shown that when two conflicting maze habits are successively established in rats, a single cerebral electroshock will disrupt the more recently learned habit, thereby permitting the old habit to regain dominance, only when the shock is administered within a certain time after training on the recent habit. In the second group it will be shown that there is a direct relation between impairment of learning and the interval between each trial and the ensuing cerebral shock in the conditioned avoidance situation.

Before reporting these experiments the work on shock-induced impairment in intellectual capacity will be reviewed. The clinical aspects of shock therapy will be omitted since they have been reviewed by Stainbrook (48), and by Kalinowsky and Hoch (14).

II. Changes in intellectual functions as a result of shock: review of the literature

A. Studies on human patients

In this section we have made an attempt to classify the papers into several categories. Because grouping of this material is difficult and often arbitrary, many of the studies overlap. For the sake of clarity, however, we have divided this literature into (1) qualitative reports, (2) the use of quantitative tests, (3) the use of batteries of tests, and (4) theoretical papers.

1. Qualitative reports

In clinical discussions of shock therapy many therapists report the occurrence of memory loss only as an incidental observation. We shall include only those papers in which the memory defect was emphasized.

Wortis (53) wrote that memory difficulties may persist for many weeks if ten or more treatments are given.

Smith, Hughes, Hastings, and Alpers (44) reported that almost all patients experienced some memory defect during treatment and that the patients considered it an unpleasant symptom. Occasionally the defect was prominent and in some patients it persisted for several months. The authors reported no patients in whom the loss was permanent and indicated that it was usually a spotty defect, ordinarily applying to proper names, places, and dates.

Levy, Serota, and Grinker (17) treated twelve patients with electroshock and eleven others with metrazol. They found that impairment in intellectual functions occurred more frequently in the electrically treated patients. Impairment in electroshocked patients lasted from one to several weeks, and patients with more shocks showed greater defect.

Plattner (29) treated seven patients with combined insulin-metrazol therapy. At the end of the treatment three of these patients showed slight disturbances of comprehension. The other four showed a marked amnesic syndrome that for the time being seriously interfered with their ability to work. In an analogous study with five patients receiving only metrazol, no memory loss appeared.

Birois (1) found that 20 patients out of a total of 41 treated with electroshock showed disturbances of memory. All types of amnesias were observed, 7 cases of pure retrograde, 11 cases of retro-anterograde, and 2 cases of pure anterograde. All defects were fragmentary rather than global in character. The anterograde amnesia was transitory, lasting 15 days at the maximum after termination of treatment. The retrograde amnesia persisted longer. The restoration of memory, beginning with the end of treatment, was gradual.

Smith, Hastings, and Hughes (45) reported on 312 patients treated with shock. All developed some degree of memory loss. This ranged from slight impairment lasting a few hours to more serious defects lasting as long as nine months. No permanent defects were seen. The memory defect increased with the number of treatments. There was no apparent relationship between the age of the patient and the degree of impairment, but in the older patients the defect lasted longer than in the younger patients.

Most shock therapists believe that the memory loss is always reversible, except perhaps in old patients. Brody (3), however, reported five cases in which the loss seemed to be permanent. These cases were all completely recovered from their symptoms and had been out of the hospital at least a year at the time of writing. All patients had reported some recovery from the memory impairment, but it had not cleared up entirely. The author wrote

that the memory disturbance seemed to be chiefly a loss of long-known familiar material, particularly names of persons and places and habits of work. Brody believed that his findings implied permanent or semi-permanent damage to the brain. He therefore believed his results contraindicated shock therapy for those individuals whose work required them to remember names of persons and places.

Qualitative though they are, these reports afford evidence that shock therapy does induce disturbances of memory. Various workers disagree, however, as to the seriousness of this complication. Since nearly all therapists believe that the memory loss is reversible, it is not considered serious enough to be a contraindication to therapy.

2. The use of quantitative tests

There are still too few studies of a quantitative nature on memory loss from shock therapy. Besides the papers reported in this section the reader may find that some of the experiments reviewed in sections 3 and 4 below are relevant.

Zubin and Barrera (56) taught several patients paired word associates under different conditions. Some learned before taking electric treatment, some during the course of treatment, and others after a series of shocks. Recall, recognition, and relearning were tested under the same conditions. Control groups were utilized throughout. They report that when no shock intervened between learning and relearning, the number of trials saved was significant when compared to its standard error. However, when a shock was interpolated between learning and relearning there was no saving, and indeed there was a slight but non-significant loss. They emphasize that learning ability was no poorer after shock than before shock. Recall after shock was found to be significantly less than recall after the control and non-shock

periods. The highest recall was found when both learning and recall occurred in the post-treatment period. Concerning recognition the authors report that the patients were able to recognize correctly both paired associates nearly 94% of the time under control conditions. Under shock conditions this dropped to 81.5%. They note, however, that shock interfered only slightly with recognition since the post-shock recognition was 87% as strong as the post-control recognition. Some of the patients had been taught the material some time before treatment, while others had been taught it immediately before treatment. The authors were therefore able to report on the differential effect of shock on recent and remote learning. They found that the shock affected the material learned immediately before shock more than the older material.

From a paper by Sherman, Mergener, and Levitin (41) we obtained the results of some investigators whose work was not available to us in the original. These authors reported that Tooth and Blackburn tested sixteen metrazol-treated patients with a modified form of the Babcock test. The results on this test were compared with the pre-psychotic intelligence level as estimated from a vocabulary score. Nine of the subjects complained of memory difficulties and the test results showed evidence of an impairment in eight of them. Sherman, et al, reported, however, that Wittman, and later Wittman and Russell, found no evidence of memory defect from shock therapy.

Sherman, Mergener, and Levitin's own experiment consisted of administering a series of six tests before, during, and after a course of grand mal seizures in ten patients. Four patients received electric shocks and six received chemical ones. They found an increase in the average scores on four standard memory tests when the responses during the course of therapy were compared with those preceding the experiment. The increase, however, was not significant

except in one test (report of a paragraph heard) but the fact that there was a slight improvement in all tests suggested that the treatment had a favorable effect. This effect was attributed to improvement in attention and concentration, compared with the efficacy of these processes before treatment.

Zubin (57) studied the 'interference' effect as influenced by electroshock. Paired associates (household commodities paired with pseudo-brand names) were taught before shock and again after shock. Although it is not clear what he means, Zubin says that direct interference was introduced by utilizing the same commodities each time. Under control conditions the interference effect was marked and Zubin had predicted that when shock was introduced between learning and relearning the interference would disappear. Instead it was accentuated. The author's tentative conclusion was that electroshock disorganizes but does not destroy the memory trace. The maze experiments to be reported in our paper tend to bear out this conclusion.

Stone (51) was interested both in memory loss attributable to shock and in the recovery from this impairment. He used two distinct groups of patients. Group 1 received the Wechsler Memory Scale, Form I, one day before the first convulsive shock and Form II one day after the last shock. Group 2 received the same test, Form I, one day after their last shock and Form II approximately 2 weeks later. For Group 1 the loss was 16% of the original score; for Group 2 the gain was 27% of the first score. In both cases the changes in performance were statistically significant. This carefully planned experiment affords clinching evidence both of the memory loss due to shock and of the gradual improvement in memory following treatment.

Purcell (32) performed a number of psychometric experiments on patients receiving electroshock. We present a summary of the results.

1. When shock preceded learning the effect on memory was more deleterious

than when shock was interposed between learning and retention.

2. Relearning scores for the group where shock preceded learning did not differ significantly from the group where shock occurred between learning and relearning.
3. Recall was significantly better for learning on non-shock days than for learning on shock days.
4. Recognition scores for material learned after shock did not differ significantly from scores when learning preceded shock.
5. The results of Zubin and Barrera, see above (56), on savings and recognition were not confirmed.

Although the experimenters whose work has been reviewed in this section used quantitative tests, these tests were not always standardized. This increases the difficulties in summarizing this material and may account for some of the disagreement. Nevertheless it is obvious that nearly all workers found some memory impairment from shock. The main source of disagreement, disregarding differences in method, lies in which functions of the memory process are most seriously affected by shock.

3. The use of batteries of tests

Although the main emphasis in the study of intellectual impairment from shock therapy has been on memory, some experimenters have studied the effect of shock on global intelligence, or on an array of psychological functions. We shall omit the papers in which personality tests were utilized and review only those experiments which employed the more 'intellectual' tests.

Stone (50) administered five forms of the Army Alpha intelligence test to 15 hospital patients during and after a course of electroshock therapy. With but few exceptions the test scores showed an appreciable decline from the first to the last of the test series and a corresponding rise in scores

during the three weeks following the last treatment. The mean of the final test scores was approximately 10% higher than the mean of the best previous scores (usually the initial test scores).

McNeel, Dewan, Myers, Proctor, and Goodwin (22) compared the psychiatric rating of 33 schizophrenic patients with the results from a psychological test battery given before and during insulin treatment. Their results showed that if the psychiatric rating approached normality after shock, so did the psychometric rating.

In the first of a series of two papers Schnack, Shakow, and Lively (39) administered the 1916 Stanford-Binet, the Kent-Rosanoff Word Association Test, and a test of Level of Aspiration to 70 male schizophrenic patients. Their interest was in finding test items which would serve as prognostic indicators of long-run clinical improvement from insulin and metrazol therapy. In a second study reported in the same paper the authors reexamined the test results to secure any information regarding the differential effect of the shock treatment on psychological functions. The general result was that considerable changes in the direction of improvement were noted in most of the measures. To test the significance of the test changes comparison was made with individually matched control patients who had had neither form of shock therapy, but who had been given two tests while under routine hospital care. The results indicated that approximately two-thirds of the improvement could be attributed to the ordinary hospital regime and to familiarity with the test situation.

Wechsler, Halpern, and Jaros (52) used Wechsler's vocational interest blank, a test of counting by threes, naming words in three minutes, and a similarities and directions test. These tests were administered to schizophrenic patients before and after insulin treatment. A clinical appraisal of

the patients, made 6 to 18 months after treatment, was compared with the test results. Although it would seem difficult to give an exact figure, the authors report a correspondence of 87% between the clinical judgment and the test results. Their analysis further suggested that certain patients may be harmed by the shock treatment insofar as test performance after treatment is concerned.

Luborsky (19) gave a battery of 22 psychometric tests to 12 patients before, during, and after treatment by electroshock. He made two comparisons: between scores before shock and scores during shock, and between scores before shock and scores after shock. The schizophrenic patients showed a general decrease in test scores in the before-during comparison and a less marked decrease in the before-after comparison. The depressive patients, however, showed large score increases in the before-during comparison and further increases in the before-after comparison.

Perlson (28) reported the unusual case of a 27 year old male patient who received 248 shock treatments. Of these, 94 were metrazol injections, 152 were electroshock convulsions, and 2 were by electronarcosis. Several days after the last shock the following tests were administered: the Otis Employment Test 1, the American Council on Education Psychological Examination 1940 College Edition, the Ohio State University Psychological Examination Form 21, the Bennett Test of Mechanical Comprehension Form AA, the Likert and Quasha Revised Minnesota Paper Form Board Test Series AA, and the Kuder Preference Record Test Form BB. In all of these tests the patient did surprisingly well; no intellectual or emotional deterioration was disclosed.

Evidently there is greater disagreement when batteries of tests are used than when simple memory tests are employed. Particularly in this group of studies, wide differences in method and in tests employed preclude adequate comparison of results. Nevertheless, when the experiment is designed to allow

for accurate comparison of test results, either on the basis of control groups or standard scores, a depression of intellectual ability has usually been found.

4. Theoretical papers

A minority of writers has attached theoretical significance to the occurrence of memory impairment from shock therapy. In some cases these theoretical interpretations which may or may not be based upon research, are little more than the author's conclusions on the problem.

Myerson (26) believes that the basis of improvement and recovery through shock therapy lies in the depression of the higher activities of the brain. Thus the memory is impaired and the most recent acquisitions, which include the pathological state, are forgotten. As the brain recovers, the well-established trends, those which are relatively normal, come back.

The latest conclusions of Zubin are reported in an abstract of a paper given before the Midwestern Psychological Association in 1946 (58). He believes that learning and retention, when measured by recall and relearning, are adversely affected by electric shock therapy. Recognition suffers only insofar as a 'jamais vu' phenomenon is observed. Retention is present for materials learned on shock days. General intelligence declines during the treatment period.

Ziskind (54) has presented a very complete discussion of the memory defect occurring under shock treatment. He has also formulated a theory relating the memory loss to symptom relief. We shall summarize his paper in some detail.

In considering the nature of the memory defect Ziskind says that the persistent amnesia resulting from metrazol therapy resembles the memory impairment noted in organic psychoses. In its mildest form it is a lacunar

disturbance for isolated events of recent origin. With greater severity, remote memory is also affected. Still more pronounced forms present the Korsakoff syndrome.

Ziskind warns that memory defects may be overlooked, particularly in patients with various degrees of inaccessibility. On the other hand, the memory impairment may give the impression that the patient is getting worse and lead to discontinuance of the treatment. Such practice will give rise to therapeutic failures and has probably colored statistics unfavorably. For satisfactory continuance of treatment the physician judiciously spaces subsequent treatments farther apart. In fact, Ziskind says that shock treatment is highly quantitative. Success or failure may depend on adequate spacing of convulsions with reference to the favorable effect on symptoms on the one hand, and the adverse effects on memory on the other.

Concerning the genesis of the memory defect, Ziskind says that there is pronounced impairment of memory after each metrazol convulsion. It is brief and transient and is probably entirely reversible for one or more of the early seizures. Some workers give the duration as up to two hours, others up to eight hours. It is to be expected that the duration will become progressively longer as the injections become more numerous and more frequent. The postconvulsive amnesia is chiefly for events of recent origin. When the patient wakens from the postconvulsive stupor he shows loss of memory principally for the occurrences of the same morning. Recovery takes place progressively so that the earliest happenings of the day are recalled first, and then subsequent events in order up to the time of the injection. The injection itself is recalled last.

After the later convulsions minor residual memory defects may persist and finally cumulate to produce the more enduring amnesias. This development may

be visualized as the resultant of two forces. The first is the primary destructive or depressive effects of metrazol on neural functions; the second is the recuperative, nutritive, or anabolic functions of the brain. The primary action of each metrazol convulsion is shown as a precipitate diminution or depression of memory. This corresponds with the period of unconsciousness and subsequent confusion. The recuperative phase then sets in and the loss of function is shown to be reversible. With repeated injections the functional loss is regained more slowly and incompletely, and residual impairment persists at the end of the interval between treatments. This residual defect increases with repeated treatments and tends to become more pronounced and of longer duration. Finally it becomes permanent. Ziskind believes that there is probably actual injury to nerve cells. This damage parallels the loss, recovery, and residual defect in memory. If the proffered interpretation of the memory loss is correct, the persistent amnesias are but the cumulative, more enduring, residuals of the acute postconvulsive memory impairments.

Ziskind goes on to present his theory of the relation of the amnesia to the mechanism of shock therapy. The similarities in the loss of symptoms and the loss of memory under treatment suggest a common underlying mechanism. The clinical similarities are:

1. Both symptoms and memory tend to become effaced as therapy progresses. The effect on symptoms is greater in degree and earlier in occurrence than that on memory. This is in conformity with the complete loss of symptoms after therapy, at which time memory, as a rule, is but little affected.
2. The fluctuation in symptoms and in memory is essentially similar after individual treatments. As with memory, there is an early transient postconvulsive loss of symptoms. The acute temporary disappearance

of the depression, and even displacement by elation, is not infrequently noted after a metrazol seizure. With each subsequent treatment the relief from symptoms, like the arrest of memory, is progressively longer and greater. Soon the symptoms are gone for the inter-treatment interval of from one to four days. Cure is suspected but the depression may recur if the treatment is terminated. Finally the symptoms disappear completely and permanently. The parallel memory defect would be the persistent Korsakoff syndrome.

This sequence of the loss of symptoms is the counterpart of the patient's loss of memory. A common underlying mechanism, which Ziskind recognizes as physico-chemical for the memory defect, is therefore suggested for the therapeutic results and for the amnesia. Fortunately for therapy, in the early stages of mental disorder the abnormal functions of the nervous system (symptoms) are more vulnerable than the normal functions, such as memory.

It should be noted that Ziskind, in his reference to therapy, has limited himself to the manic-depressive depressions. It is in this disorder that one sees the response to metrazol most easily and constantly.

He suggests that the greater vulnerability of the symptoms is probably correlated with their pathologic unstable substratum and their more recent development, as contrasted with the normal anatomico-physiologic character and greater longevity of the memory process. Based on these considerations, he is constructing a test for prognosis.

Ziskind next discusses the relation of the memory defect to dosage. The degree of memory loss is in proportion, among other things, to the number of convulsions. This is in keeping with the greater incidence of the memory complication late in the course of therapy. In his experience, three convulsions per week tend to produce pronounced impairment of memory. He therefore

prefers not to administer more than two treatments, although this schedule can sometimes be violated for one or two weeks. He notes that some authors have advocated the induction of a period of confusion and amnesia, believing this to have definite therapeutic value. Because of the danger of irreversible residuals, he has preferred not to hazard this state.

Finally, Ziskind considers the significance of the memory impairment for the future of shock therapy. The question appears to be one of dosage. Severe damage to the nervous system has been produced with metrazol. Whether the usual course of treatment results in injury to the nervous system cannot as yet be stated. The reversibility of the memory defect in almost all cases removes this as a contraindication to the therapy.

Ziskind's theory seems reasonable. Simply because the memory defect produced by shock is not as long-lasting or as severe as the changes produced on the symptoms, we have no right to assume that the memory impairment is an epiphenomenon, or that it is not related to the mechanism of symptom relief. Obviously, the changes in memory and in symptoms are both behavioral representations of a fundamental disturbance in the brain, both produced by shock. A theory which attempts to account for both memory loss and symptom relief is more likely to be correct than a theory of the mechanism of shock which glosses over the memory impairment as a meaningless and not too serious occurrence. The latter type of theory is all too frequently stated or implied in the writings of most shock therapists. The following two papers present further evidence in support of Ziskind's theory.

Rodnick (35) also argues that the effect of shock therapy is to knock out recent as compared with remote habits. In the case of schizophrenics, these recent habits are the psychotic symptoms. The older habits are the more normal modes of adjustment, with which the recent habits are incompatible. Rodnick

presents the following evidence:

1. Best results are claimed with cases in which the onset of the psychosis is quite recent. Older cases are notoriously resistant to treatment.
2. The best prognosis occurs in the relatively intact cases, in which schizophrenic traits such as apathy, withdrawal, and autism are minimal.
3. In many cases the effects of shock are only temporary and are later followed by a return to the psychotic condition. The temporary changes in the psychotic patterns may account for the fact that schizophrenics tend to show much more partial improvement than complete remission. Experience at some hospitals indicates that shock therapy may be more effective if a program of psychotherapy is included between shocks. Thus, during the time that the newer schizophrenic patterns are in a weakened condition because of shock, the psychotherapy may serve to strengthen the more normal behavior patterns which are temporarily dominant. This is in line with the observation that one of the main effects of metrazol therapy is to make the patient more accessible to psychotherapy.
4. The fact that a number of shocks are essential to the therapy supports the hypothesis. Frequent repetitions of the shock may serve to weaken the newer patterns still more, with the result that the dominance of older patterns becomes more permanent.
5. The fact that metrazol shock has been tried on several other forms of psychosis, with perhaps even better success, indicates that it is by no means a specific for schizophrenia. The logical deduction is that the efficacy of metrazol lies primarily in its effect on the habit systems involved in the behavior of the psychotic.
6. Metrazol may be effective only in those cases where the older, more normal patterns are not effectively extinguished (in the conditioned response

sense), such as may be the case in older deteriorated psychotics. In the newer cases, where complete extinction of the normal responses has not yet taken place, the shock aids in return to dominance of the older habits. This conforms to various reports that shock therapies may not actually show a much higher remission rate than spontaneous remissions, but may merely serve to hasten the process of remission. But if the schizophrenic patterns are too well entrenched, so that the difference in strength between the older and newer patterns is too great, the shock is not sufficient to affect the dominance hierarchy. (Some experimental support for such an hypothesis will be found in the results of our own maze experiments to be reported later.)

Rodnick describes an experiment of his own. The experimental group consisted of 21 schizophrenic patients undergoing metrazol therapy. The control group of 21 schizophrenics was not given metrazol. Two conflicting habits were taught. Habit I consisted in training the subject to move his finger to the right for a tone of 500 cycles and to the left for a tone of 700 cycles. For Habit II the subject was trained to move his finger in the direction opposite to that in Habit I. On the first session the S's were given 100 training trials, 50 to each tone, the stimuli always being presented in a predetermined varied order. This constituted the training on Habit I. On the second session, 24 hours later, the S's were trained on Habit II by being instructed to reverse the direction of their responses to the tones. In this second session 75 training trials were given. The number of training trials to establish the habits was chosen after preliminary work on a separate group of controls. This had indicated that in most subjects 100 trials on Habit I and 75 on Habit II did not strengthen either habit to a point where it was strongly dominant at the time of retest.

One hour after the training on Habit II the subjects of the experimental

group were given a metrazol injection. They were retested 1½ hours after the injection to determine which habit was dominant. On this retest session the S's were instructed not to decide in advance in which direction to move the finger, but to respond as quickly as possible when the tone was presented. Ten retest trials were given, five to each tone. For both groups of S's the time relationships were identical between the learning and retesting sessions.

Since there were 10 test trials any S could vary from 0 to 10 reversals to the older habit. On this basis the following table of results was obtained.

No. of reversals	Experimentals	Controls
0	3	7
1	3	4
2	1	3
3	0	2
4	0	1
5	2	1
6	1	2
7	3	0
8	4	0
9	1	1
10	3	0
	N = 21	N = 21

It may be seen that 7 members of the metrazol group showed less than 50% reversals while 14 showed 50% or more reversals. For the control group 17 subjects showed less than 50% reversals while 4 showed 50% or more. The computed chi square for these numbers (with the Yates correction for small N) is 7.88. This corresponds to a P of less than 0.01 for this distribution. It is striking to notice that only one control case showed more reversals than did the median shock patient.

From these results it appears that one metrazol shock has a greater weakening effect on newer acquisitions than on older acquisitions which are incompatible. This is true even though there is only a comparatively small difference in the age of the habits at the time of shock.

Rodnick does not believe that his results are to be explained by confusion of the patients at the time of testing. Most of the subjects seemed quite well oriented and neither group showed a large number of responses entirely to one habit. Neither does he think that an amnesia for the events immediately preceding the shock explains the results. The training had preceded the shock by one hour and questioning of the subjects revealed no retrograde amnesia. On the other hand, he says that even if some general factor such as amnesia is an important variable in the effect obtained, it does not thereby disprove the hypothesis. It is indeed quite probable that the amnesia itself results from the same condition which produces the reversal of the habits. In any case, the empirical results are the same.

Rodnick believes that the importance of this study lies in indicating that a metrazol shock does have a differential effect on older as compared with more recently acquired habits. It suggests that a general psychological principle, quite analogous to the temporary inhibition of conditioned responses, may play a role in metrazol therapy. However, he cautions against the too general application of this principle to the complicated structure of symptoms seen in schizophrenia.

The experiment of Ziskind, Loken, and Gengerelli (55) is relevant here. These authors hypothesize that symptoms are of most recent origin and affect chiefly cortical structures; therefore they should be the habits most vulnerable to metrazol attack. For their experiment they wanted to establish new learning of a fairly high order, that at the same time would be subject to objective control. For this purpose a code transcription procedure was chosen.

The authors do not make quite clear the nature of the task. They say that after preliminary explanation and demonstration the patient was asked to

transcribe from memory a nonsense syllable code as rapidly as possible. He worked 4 minutes; his score was the number transcribed. After 2 minutes' rest he was given 6 minutes of practice at transcription. Then followed another 2-minute rest interval and a 4-minute retest. The number of items transcribed in this latter period was called Retest Score 1. During the week following Retest 1, two or three metrazol injections were administered. At the end of the week another 4-minute retest was given; this constituted Retest Score 2. By comparing the scores made on Retests 1 and 2, changes in performance induced by metrazol could be detected.

Two control experiments were used. In Control I the same patients were given an alternate and comparable form of the code test, but during the period of forgetting no metrazol was given. Control II consisted of administering the tests to another group of patients not undergoing metrazol treatment. There were 6 patients and 6 controls.

The results are presented in the following table.

Average difference in scores between Retest 1 and 2

Metrazol cases	-25.1%
Control I	+ 2.7%
Control II	+ 3.5%

The differences between the control groups and the metrazol group are statistically reliable. The authors conclude that the impairment in performance after metrazol appears to be due to impaired memory.

5. Summary

The evidence presented tends to indicate that insulin, metrazol, or electricity can have a deleterious effect on certain intellectual capacities. There is still disagreement as to which functions are most affected by shock. Some authors have emphasized a loss in ability to recall events which occurred

before the shock, but they vary in the reported extent and persistence of the loss. Other workers have shown that shock therapy changes the ability to learn; most found a loss, but some reported improvement in this function. Finally, those who studied intelligence, or an array of other psychological functions, do not agree on either the direction or the extent of the shock-induced changes. There is one rather obvious explanation for some of the divergent results. Lack of motivation to do well in the tests is all too frequent in psychotic patients. Under motivation we include interest, attention, rapport, etc. When one of the common definitions of a psychosis is "a disease which incapacitates the person for work," it is not hard to understand that the reliability of test scores on such persons is likely to be low. This criticism is most valid when the tests are administered in the acute stage of the symptoms, the time when shock therapy is most likely to be introduced.

Even if we allow for the difficulties in working with mental patients, there is still considerable evidence that shock therapy does produce some depression of function, particularly in recall memory. But it is interesting to note that the two most recent reviews of shock therapy, both appearing in 1946, differ in the emphasis placed on the importance of the memory loss. Kalinowsky and Hoch (14 , particularly pp. 133-136) tend to play down the memory loss and to stress the advantages gained from shock. On the other hand, Stainbrook says, "Whatever may be the reports concerning the complete disappearance of shock-induced memory impairment, no one who has talked to patients who have undergone electroshock treatment can doubt that there is a considerable amount of experience surrounding and during the course of treatment which remains permanently inaccessible to memory." (48 , pp. 45-46)

Perhaps the most interesting formulation to come out of this literature is the hypothesis that shock is more effective in disrupting recent learning

than older learning. This hypothesis forms the basis for those experiments to be reported in this paper in which the maze was used. Such a behavioral hypothesis will not serve as the final explanation of the mechanism of shock on the body. Eventually we must know the physiological effect of shock therapy. But at the present stage of knowledge, a behavioral hypothesis must serve.

B. Animal experiments utilizing shock

A number of experiments to determine the effects of insulin, metrazol, or electricity have been performed upon animals. These studies have been chiefly concerned with the effects of shock on the learning, the retention, or the recovery of habits. The following review will show that there is some divergence among the results of the various experiments. This disagreement is in part due to the different techniques used by different investigators. It may also be partly due to the fact that many of those experimenters who found no effect from shock used relatively simple habits. Results from the present experiments indicate, that it is difficult to produce changes in behavior with electroshock when the problem confronting the animal is quite simple. In reviewing this literature we have again attempted to classify the experiments. We distinguish between experiments in which (1) maze and problem situations, and (2) conditioning methods were used.

1. The maze and problem situations

Most of the experiments to be reported in this section are rather recent. Although there are several papers to report, the work has not been as extensive or as varied as that done with human subjects.

Bunch and Mueller (4) have shown that rats subjected to a series of metrazol convulsions manifest no differences from a control group in the subsequent learning of a 14-unit multiple T maze.

Stainbrook and Löwenbach (46) have found that in a simple water maze a long series of electroshock convulsions does not alter the maze behavior of the rat insofar as error scores are concerned. Time scores, however, are significantly increased.

Siegel (43) taught rats a modified Graham-Gagné runway. One group was

then subjected to ten electroshock convulsions; the other group had the electrodes clamped to the ears once each day but received no shock over the same period. In the first retention trial both groups showed a slight loss of the habit but the loss was equal for both. In several subsequent relearning trials there was very little difference between the two groups. Siegel concludes that electric shocks had no effect on the retention of a barely-learned response, and that the ability to learn a simple habit is not adversely affected by a series of shocks.

In contrast to the experiments cited above, several workers have found behavior changes after shock. In most cases the habits tested were more difficult than those used by experimenters who found no effect.

Loken (18) reported fairly large time and error differences in his animals' relearning of a 6-unit maze after metrazol convulsions.

Working from the hypothesis that the known effect of insulin on the brain, cerebral anoxia, tends to break up recently formed habits more than older ones, Riess and Berman (18) reported several experiments. Using a relatively complex and a simple maze, they found that: (1) Insulin had a greater disintegrative effect on the continued learning of a partially learned habit than on a habit of greater fixation. (2) Insulin had a greater detrimental effect on the longer and more difficult maze than on the shorter and easier maze. (3) Insulin had a disintegrating effect on the initial acquisition of a maze habit when compared to the learning of the same habit under normal conditions.

Stainbrook (47) found that the relearning time for a maze was greatly lengthened following a long series of electroshock convulsions, although the relearning errors were no greater than in a control group.

The present writer reported an experiment using the Lashley III maze (6).

This maze was chosen because it has been found to be a fairly difficult problem for the rat (16). Rats were trained on this maze until they had learned it to a criterion of three errorless trials. They were then divided into three groups, equated as nearly as possible in terms of learning scores: One group received 85 volts A.C. for .2 sec. once a day for 30 days, the shock passing through the head. Another group received the same series of shocks but the electrodes were attached to the hind legs. The third group received no shock. Shocking was begun 24 hours after the last criterion trial for each rat. The rats were retrained on the maze beginning 24 hours after the 30-day shock period and were retrained to the original criterion. The results were clearcut. The group that had been shocked through the head was significantly inferior to the other two groups in terms of: (a) trials, errors, and time scores during relearning, (b) percentages saved in trials, errors, and time in relearning over learning, and, (c) errors and time on the first retention trial. The group shocked through the legs was slightly, but not significantly, inferior to the group that had received no shock. The results thus indicated that shocks through the head caused an impairment that showed up either as a loss of retention or of relearning ability (or of both) for this habit. Most of the loss seemed to be in retention.

Taking their cue from this experiment, McGinnies and Schlosberg (21) studied the effect of electroshock on double alternation lever pressing in the rat. It is known from the work of Hunter and his students (see particularly the paper by Hunter and Hall (13)) that the double alternation maze is a particularly difficult one for rats. Schlosberg and Katz (38) extended this work with the double alternation lever pressing problem which, although more economical of time than the maze, is still a very difficult task for the rat to perform correctly. It therefore seemed to McGinnies and Schlosberg

that this habit would be particularly sensitive to the effect of electroshock. Their results amply support this hypothesis.

In the experiment McGinnies and Schlosberg attempted to train four rats to the correct double alternation sequence of lever pressing for food reward. Only two of the animals reached the criterion of 80% correct double alternations in the daily recordings of 20 runs. The other two rats which never mastered the problem appeared excited and over-active in the situation. The animals were then subjected to various electroshock procedures. The results showed that each of the two rats that had learned to criterion exhibited breakdown of performance following each of two pairs of cerebral shocks. In each rat one pair of shocks was followed by a rest period of 20 days; the other pair of shocks was succeeded by immediate retraining. The results suggested that electroshock is followed by a short period during which the rat cannot profit from retraining.

Following this experiment the authors subjected the same two animals to a series of convulsions induced twice weekly and accompanied by daily practice in the apparatus. The reaction to the initial shocks was sudden breakdown followed by rapid recovery. This was succeeded by a period of variable or generally poor performance. Finally, both animals again reached criterion and remained there despite continuance of bi-weekly convulsions.

In the final experiment one of the rats which had never performed at criterion was subjected to a schedule of daily shocks. This animal was larger than the others and had exhibited considerable over-activity in original training. He improved steadily during the period of convulsions and, following 19 consecutive shocks, performed better than he had for any comparable period prior to the shocks. The results of these experiments led the authors to conclude that the effect of the shock was to decrease activity

level, which in normally active rats resulted in impairment in the habit but which in an over-active rat led to improvement in performance.

Horowitz and Stone (12) tested the hypothesis that a previously-learned habit, disorganized by electro-convulsive shock, would give less interference with the learning of a new habit. Thirty animals were run on the Stone Multiple Discrimination Box to a criterion of four out of five trials. Of twenty-three successful animals, thirteen formed the shock group and ten the control. The new habit consisted in switching the correct path to the dark alleys. The shock had a measurable disorganizing effect on the original habit, but in switching to the new habit the control group tended to learn more readily. The original hypothesis was therefore discarded. It is interesting to note the similarity in procedure and results of this experiment with that of Zubin on the effect of electroshock on interference, (57). Zubin found that the interference introduced by changing only the stimulus words of paired associates was accentuated in human patients receiving electroshock therapy when compared with a control group. As in Horowitz and Stone's experiment, the control group learned more readily.

In a recent experiment Sharp, Winder, and Stone (40) studied the effect of electric shocks on 'reasoning' ability in rats. The apparatus used was a modification of one which Maier designed to study reasoning ability. The behavior of the animals was recorded on 22 post-shock trials with 22 hours of recovery from each shock, and on 10 post-shock trials with 1½ hours of recovery time. The impairment of performance was assessed in terms of (a) curtailment of distance traveled during the exploratory period before the test runs, (b) time, as measured from the moment of release for a test run until the animal found the reward, and (c) correct choices made. The results showed that the mean of exploration distance dropped appreciably in the 22

hour post-shock series and dropped even more in the 1½ hour series. The mean time of test runs following shock was greater than in the pre-shock period, and the time after short recovery periods greatly exceeded that after long recovery times. Finally, the accuracy scores showed a downward trend which resembled that of the other measures. The consistency of downward trends for all measures suggested that there was a real impairment in the function measured by the Maier test.

It has been suspected by a number of workers that one of the effects of electroshock is a depression of general activity. Time scores often shoot up considerably in the performance of skilled habits after shock and it is frequently observed that the activity of a shocked animal is less than that of his cage mates. Stone (49) made a quantitative study of the effect of electroshock on general activity in rats. The rats lived in revolving drums. As soon as a baseline was established the animals were put through a series of one convulsive shock per day for 5 days, followed by 5 days without shock, then 5 more shock days, and so on for 3 alternating periods of shock and no-shock, plus 2 additional post-shock periods. It was apparent from the activity graph that the rats were much less active during the shock periods than during the no-shock periods. Reduction in activity began in the first 24 hours after a convulsive shock and disappeared during the second day beyond the last shock. Although the major depression of activity occurred within the first 24 hours after a shock and disappeared within 48 hours after the last shock, it was not until 2 weeks after the final shock that the majority of the animals began to approach their pre-shock level of activity.

Page (27) has also found that general activity of rats is depressed during electric shock series and that the shocked animals lose weight.

In summary of this section it may be said that most experimenters have

found that shock impairs learning or performance of skilled habits. This is particularly true when the habits used are sufficiently sensitive; i.e., difficult for the animal.

2. Conditioning experiments

Except for the extensive research of Gellhorn and his associates, few experimenters have used the conditioning situation as a measure of the effects of shock. This is unfortunate since a conditioned discrimination, particularly if the animal is pushed to the limit of its ability, should be especially useful in determining the effect of shock on behavior. We do, however, have some evidence.

Page (27) tried to condition the electroshock convulsion by sounding a bell just before the electricity passed through the rat's head. No conditioning was obtained.

Rose, Tainton-Pottberg, and Anderson (35) administered a series of insulin shocks to a well-trained sheep in which a conditioned reflex had been standardized in tests extending over a period of seven years. They found that, following the hypoglycemic treatment, the conditioned reflex, which had been almost entirely absent for one year, reappeared with abnormal vigor. Movements of the reaction limb (left foreleg) were graphically recorded during the recovery period from each of 7 insulin shocks. For the first time in the history of the animal movements of the leg appeared in the rest interval between signals. These responses sometimes reached a frequency of 6 per minute. Such movements had been previously observed only in animals exhibiting experimental neurosis. On each occasion the number of such movements gradually decreased as the animal recovered from the coma and as this occurred the conditioned reflex (leg movements to the conditioned stimulus only) reappeared.

Rosen and Gantt (37) reported a brief experiment on the effect of

metrazol convulsions in dogs. They conditioned two dogs to make a salivary response, and two others to make a motor response. Discrimination of the conditioned stimulus to the motor habit was markedly impaired by the metrazol; the effect on the salivary discrimination was less pronounced.

Partly on the basis of his conditioned response experiments with shock, Gellhorn has formulated a theory of the mechanism of shock therapy. Later we shall briefly summarize his theory, but first we must review his experiments in some detail, since in several of them the procedure is rather involved.

Kessler and Gellhorn (15) investigated the effect of electrically and chemically induced convulsions on conditioning in rats. They first trained rats to a bell plus a grid shock in an avoidance situation. The response consisted of jumping over a partition in the center of the grid to avoid the grid shock. When this habit was established it was extinguished by omitting the grid shock. Then several cerebral electroshocks through the head or metrazol injections were administered. Either type of shock caused a temporary recovery from the extinction, although control experiments had shown that there was no spontaneous recovery.

It remained to be determined whether or not insulin could produce the same effect as had cerebral electroshock and metrazol. This was studied by Gellhorn and Minatoya (7). The procedure and apparatus were the same as in the experiment by Kessler and Gellhorn, except for the use of insulin. As before, when the CR had been inhibited by lack of reenforcement, no spontaneous recovery from the extinction was found in control rats. However, in rats subjected to insulin injections, the response appeared with full magnitude.

In view of these results the question arises whether the action of insulin coma on conditioned reflexes consists only in the removal of inhibitions, or whether excitatory reactions are also influenced. In the same

paper Gellhorn and Minatoya reported an experiment to decide this question. Animals were trained in the avoidance apparatus until they had just begun to learn the habit. Before complete learning had taken place, one group was given insulin shocks. It was found that the subsequent learning of the habit to criterion was facilitated in those animals that had received insulin. The authors concluded that the chronic effect of insulin hypoglycemia is not restricted to its action on inhibition, but affects likewise those excitatory processes which are the basis of the establishment of the CR. Although the authors did not make the point, it would seem that the insulin effect on excitation is opposite to its effect on inhibition, facilitating the former but impairing the latter.

In a further investigation Gellhorn (8) studied the effect of shock on several CR's. In one group of animals, 2 or 3 CR's were established in succession, but no new CR was formed until the preceding one had been inhibited by lack of reenforcement. The apparatus and procedure were the same as in Gellhorn's previous experiments, with the exception that the different CR's were established by using different conditioned stimuli. These were a doorbell, a sound of 250 vibrations, and a light. Results with this first group of animals showed that electroshock or insulin restored extinguished conditioned responses, as had been found previously. But they also showed that this effect may be exhibited simultaneously on several conditioned responses which had been established and extinguished in a definite sequence. In the original establishment and inhibition of the responses the sequence was: doorbell first, 250 sound next, and light last. However, in the recovery from inhibition after shock this sequence was exactly reversed in terms of magnitude of recovery, the response to the doorbell being recovered most and that to the light least. This result would seem to fit in with the hypothesis of

the effect of shock on recent and remote learning, except that we know that a sound is likely to be a more powerful stimulus for the rat than is a light.

In a second group of experiments reported in this paper Gellhorn studied the effect of shock on positive and negative CR's which were being maintained simultaneously in the same animal. The procedure was to establish one or two responses and then inhibit them by lack of reenforcement. Following this a third response was established and maintained in the rat. Animals trained in this fashion were used to decide the question as to whether the action of insulin coma or electrically induced convulsions is restricted to inhibited CR's, or exerts a depressant effect on non-inhibited CR's. When the animals were subjected to insulin comas and then retested it was found that the inhibited responses had been restored but that the positive CR remained uninfluenced and positive. The experiments thus showed that shock can act specifically on inhibited CR's without influencing positive CR's. Gellhorn contrasted this experiment with that of Rosen and Gantt, see above (37). The latter workers had found a loss of discriminative ability in their dogs after a series of metrazol convulsions. Gellhorn, however, found no effect of shock on positive CR's and thus no loss of differentiating ability, whereas the shock had produced a marked recovery in inhibited CR's. Gellhorn did not make the point, but it should be noted that a loss of inhibitory processes should break up sensory discrimination, if we accept Pavlov's view that inhibition is necessary to restrict generalized excitatory processes. This finding of Gellhorn's, that shock will remove inhibition but not excitation, is of particular interest to the present author, since he will later report results that are in some disagreement with those of Gellhorn.

Gellhorn's most recent experiment is a further study of the relative effect of shock on positive and negative conditioned reactions, (9). Before

making the generalization that shock acted only on the inhibited CR's, it seemed necessary to Gellhorn to investigate the effect of shock on a positive CR which, overtly at least, was similar to the negative CR. The author wanted to establish a positive CR resulting in suppression of a motor response, and in the same animal form a negative CR in which the rat likewise refrained from motor activity because of lack of reenforcement. If these two responses reacted differently to insulin coma, then new light would be thrown on the nature of the insulin effect.

In contrast to the 2-chamber apparatus used in all of the previous experiments, Gellhorn now employed a circular apparatus divided into 6 chambers. In this device the rat could escape from the shock, or avoid the shock, by running into either of the two adjacent compartments. After this conditioned response was established it was inhibited by lack of reenforcement as in the earlier work. Insulin coma was then produced. This constituted the first part of the experiment. Thereafter the CR was again fully established by reenforcing the conditioned stimulus. Then this CR was abolished, not by internal inhibition but by countershock; i.e., the charge was applied to the grid of the two adjacent chambers when the CS was presented. Upon presentation of the CS the rat jumped into one of the adjacent compartments but was driven back into the original compartment by the grid shock. Repetition of this procedure for several days abolished completely the response of running into an adjacent compartment when the CS was sounded. Then insulin coma was applied again, and the degree of recovery of the original running response was determined. Following this the rat was again conditioned, extinguished by omitting reenforcement, and given insulin as in the first part of the experiment. It was found that when the running response was abolished by lack of reenforcement, the administration of insulin induced recovery of the

CR, as in the earlier experiments. If, however, the CR had been inhibited by countershock, insulin produced no significant recovery of the response. Then when the rat was again retrained as in the first part of the experiment and again inhibited by omitting the unconditioned stimulus, insulin, as usual, led to recovery of the CR.

The failure of insulin coma to lead to recovery of the positive CR after countershock had been applied was obviously not due to a spontaneous change in the animal, since the repetition of the first part of the experiment again showed the striking recovery of an inhibited CR by insulin when the inhibition had been produced by lack of reenforcement. It was shown in later experiments that it was immaterial whether the suppression of the response by countershock preceded or followed the suppression of the response by lack of reenforcement. The recovery of the abolished CR through insulin coma was possible only when this reaction had been eliminated by internal inhibition, and not when it had been eliminated by countershock.

In discussing his results Gellhorn says that the clue to an understanding of the experiments seems to lie in the fact that the significance of the CS, previously the signal for an escape reaction, was altered by its combination with countershock. In these circumstances the escape reaction was suppressed and the behavior of the animals was overtly similar to that seen after internal inhibition. But important physiological differences existed between the two conditions. In the condition present in part A of the experiment the temporary association of the CR and CS still existed, though in an ineffectual form, so that, owing to the lowered excitability of the brain as a whole as a result of internal inhibition, the CS was unable to elicit the positive escape reaction. Under the influence of insulin coma these weak links between the CS and CR were apparently intensified and thus the original escape

reaction reappeared in response to the CS. However, in the case in which the CR was abolished by countershock the situation was quite different, since a new positive CR was substituted for the old one. Since this reaction was established under the influence of a strong unconditioned stimulus, countershock, it quickly replaced the former CR. The new behavior was easily acquired and apparently was a very stable response in which the animal refrained from running in response to sound. If the action of insulin coma and other related procedures had produced an increase in the general level of excitability, it might have caused the animal to react to the bell with a vigorous escape reaction in both parts A and B of the experiment, regardless of the fundamental difference in the physiological reactions which form the basis of the two responses. The experimental results showed clearly that this was not the case. The specificity with which shock procedures restore inhibited conditioned reactions, without affecting the avoidance reaction to countershock, indicates that cerebral shock acts only on those cortical processes which, although latent during internal inhibition, are the basis of the CR. This interpretation is in agreement with the results in previous studies in which the effectiveness of insulin coma and electroshock in the restoration of inhibited CR's was directly related to the stability of the CR. Reactions to a bell, which were established with ease but abolished with difficulty, were more effectively restored by shock procedures than were CR's to a light, which were established with difficulty but abolished with ease, indicating a lesser degree of stability. The experiments reported in this as well as in previous papers seem to warrant the statement that positive CR's, no matter whether the CR consists in a movement or in the suppression of a movement, are not altered by insulin coma.

These experiments of Gellhorn are part of an extensive series of investigations into the nature of shock therapy. From this work he has evolved a

theory of the effect of shock therapy which he believes accounts for the physiological changes which occur when a mental patient is relieved of symptoms by a course of shock. This theory is explained in detail in his book Autonomic Regulations (10) and new papers relating to the theory appear frequently. Briefly, Gellhorn's theory is that in mental disease there is an abnormal lack of balance between the vago-insulin branch and the sympathetico-adrenal branch of the autonomic nervous system. When the patient is subjected to insulin, metrazol, or electroshock, the sympathetico-adrenal system is stimulated to great activity, and this activity continues for a long time after the shock has been administered. Prolonged excitation of the sympathetic centers may restore the patient's disturbed autonomic balance and exert far-reaching effects on the cortex itself. This is shown by the restoration of inhibited conditioned reactions after shock procedures, and by other lines of evidence that we cannot review here.

In support of Gellhorn's theory it may be said that it is the only theory of the underlying mechanism of shock therapy which is supported by much experimental evidence. But almost all of this evidence has been contributed by Gellhorn and his collaborators, and the experiments are often so poorly described that they may be difficult to repeat. The only other theory worth mentioning is a descriptive one in which the shock is assumed to impair recent but not remote memories. It is only fair to mention, however, that in the recent book by Kalinowsky and Hoch (14), where a whole chapter is devoted to a discussion of theories of the mechanism of shock therapy, both Gellhorn's theory and the amnesia theory are given short shrift.

3. Summary

We have now reviewed the pertinent literature on the effect of shock procedures on learning and retention in human and animal subjects. The

conclusion seems justified that after one or more shocks, induced by any one of the three major methods of shock procedure, changes in psychological functions may occur. These changes are usually, but not always, depressions in the functions atudied. The changes are often greatest, particularly when adequately measured, in the learning or the retention of verbal or motor habits, although in human mental patients the emotional changes, in the sense of symptom relief, may well be more striking.

In view of the frequent report of amnesic difficulties from shock, and particularly because of the emphasis placed upon these amnesias by many writers, it is obvious that adequate experimentation on the problem is of greatest importance. The psychologist, limited in other respects in research on shock therapy, is especially able to investigate habit disintegration and memory impairment due to shock. We shall now report a series of experiments which are oriented about the hypothesis that shock impairs recently learned responses more than older responses. If this hypothesis is tenable, it may help considerably in the attempt to explain the underlying mechanism of shock therapy.

III. Maze experiments

A. Introduction

Our purpose in these experiments was to determine the effect of electroshock on a recently learned habit which matched in strength an older conflicting habit. We have already presented some evidence from the work of previous experimenters that shock procedures can weaken a recent habit, thereby permitting an older incompatible habit to regain dominance. Except for the work of Rodnick (35), however, the previous authors utilized rather complicated habits and a series of shocks. Their experiments are therefore subject to the criticism that uncontrolled variables may have been operating to bias the results. This criticism is particularly valid regarding the time of application of the shock, the spacing of the shocks, and the number of shocks administered. These variables are important; several writers have stressed the value of treating patients by shock therapy early in the history of the disorder, and Ziskind (54) has emphasized that, in view of the cumulative nature of shock, the number and spacing of the convulsions are crucial. It is difficult to assess the results of many papers which do not report these factors in exact detail.

In the present series of experiments we attempted to clarify these factors by keeping the learning situation simple. Hence we used a single unit T maze, which gave a choice of two simple, clear-cut responses; a turn to the right, or a turn to the left. One of these habits was strengthened by repeated reinforcement with a small portion of food, with practice distributed over many days. The animals were then coaxed down the other arm of the T, using a trail of food, and rewarded with a large portion of food, until the new habit was stronger than the old one. In a typical series, some of the

animals were then given a cerebral shock, and later retested on the maze. The critical question was whether the shock had weakened the new habit more than the old one. This is the basic scheme of all our maze experiments. The preliminary experiments were designed to determine the conditions of practice and reward necessary to yield two habits of fairly equal strength. The main experiments were concerned with the effect of varying the time intervals between the training and shock, and between the shock and testing.

B. General procedure and apparatus

1. Apparatus

The maze used in all of the experiments was an elevated one unit T maze. The stem of the T was 60 inches, and each arm 30 inches in length. Small wire mesh cages in the shape of Quonset huts served as a starting box and the two goal boxes. These cages were hinged on one side to allow insertion or removal of the rat. The interior of the starting box and the top of the maze were well lighted by an overhead bulb. The goal boxes were darkened by covering the wire mesh with black cloth and were placed at right angles to the ends of the maze arms. This prevented the animal from seeing the food reward until he had actually entered the goal box. The maze is shown in Figure 1.

As will be described below, it was necessary at times to have food particles on the arms and stem of the maze. At other times all traces of this food had to be removed to prevent the operation of olfactory cues. To accomplish this the top of the maze was covered with strips of Masonite, which formed the running surface for the rat. One strip covered the stem of the maze and another covered the two arms; all strips were 2 inches in width. Several of these covers were available, permitting a complete change of running surface in a few seconds. Tactual cues which might determine the rat's choice of turn were also controlled by changing the maze covers. Visual and auditory cues were controlled by illuminating all sections of the maze equally by the overhead light and by running the animals in the evening when the building was quiet.

The electroshock apparatus permitted varying the voltage and the duration of current flow. These two factors are reciprocal in their effects; an

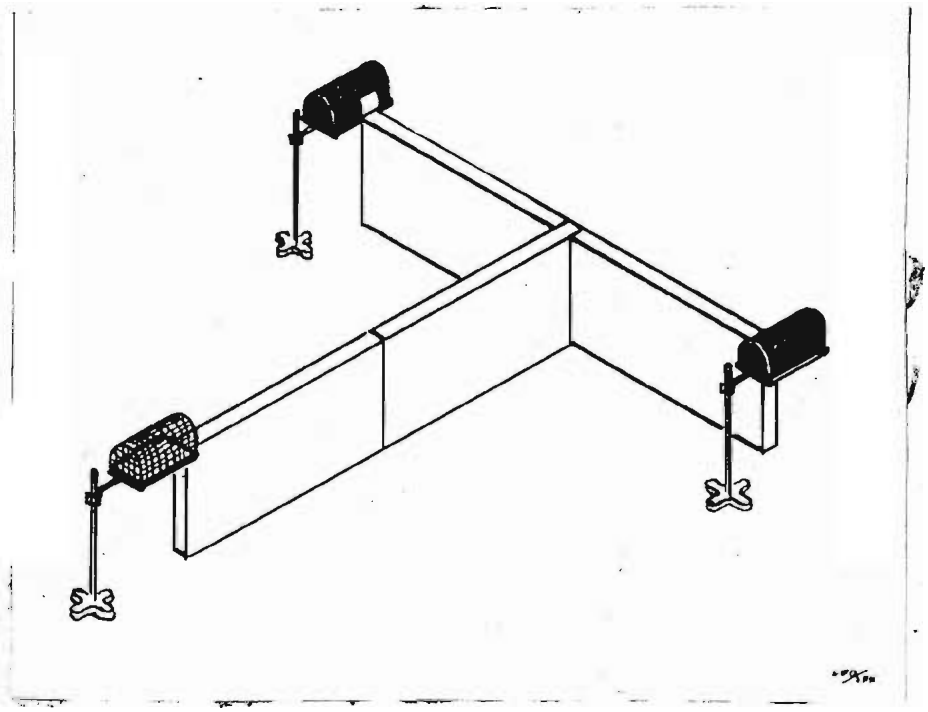


Figure 1

The maze used in the present experiments

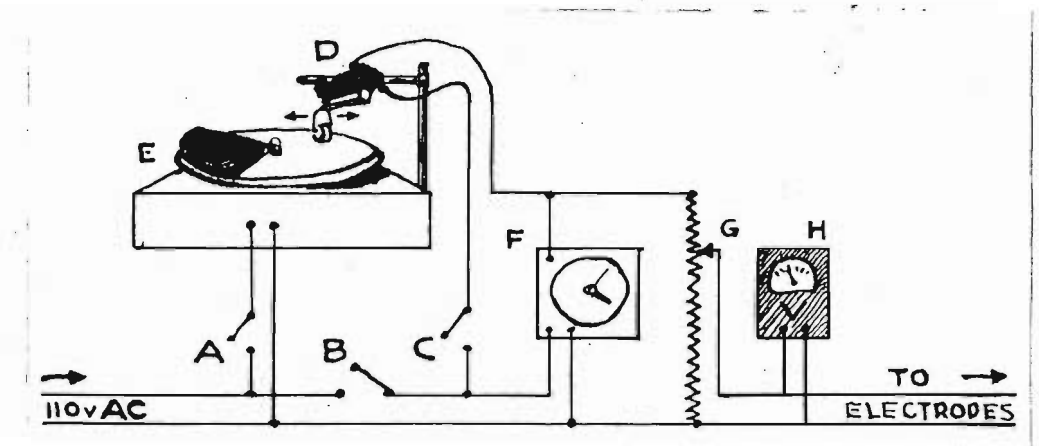
increase in either increases the chances of inducing a major convulsion.

Leads from the ordinary A.C. circuit in the building were attached to the poles of a 27 ohm potentiometer. Wires from the potentiometer led to alligator-clip electrodes. The jaws of the electrodes were wrapped in gauze and were soaked in physiological salt solution just before an animal was shocked. Throughout all the experiments the potentiometer was set at 85 volts, as determined by a voltmeter in the circuit.

The following device was built to control the duration of the current flow. A phonograph motor was mounted in a wooden box with the shaft protruding through the cover of the box. To this shaft was fixed a Masonite turntable 9 inches in diameter. A wooden lug 4 inches long and 4 inches wide was bolted to the top of the turntable. On top of the wooden box and directly beside the turntable a ringstand and clamp were mounted. The clamp held a wheel microswitch which rested on the turntable. As the turntable revolved, the wheel of the microswitch passed over the lug, tripping the current on and off. By moving the ringstand clamp the microswitch could be made to pass over the lug at any point between the perimeter and the center of the turntable. The total range of durations thus obtained varied between .05 and .35 of a second. A .01 second stop-clock was used to determine the duration of the shock. In all of the experiments the timer was set for .20 second. Thus, the cerebral electroshock consisted of 85 volts A.C. lasting for .20 second. The electroshock apparatus is shown, somewhat schematically, in Figure 2.

2. General procedure

The two habits employed were the running to the right or to the left on the T maze for food reward. The assumption was made that these habits were of equal difficulty for the rat when the operation of position habits had



WIRING DIAGRAM
ELECTRO-SHOCK APPARATUS

A. MOTOR SWITCH
B. LINE SWITCH
C. OPERATING PUSH-BUTTON
D. WHEEL MICROSWITCH
E. TURNTABLE and LUG
F. TIMER CLOCK
G. POTENTIOMETER
H. VOLTMETER

Figure 2

The electroshock apparatus, partly schematic

been controlled. The habits were, of course, conflicting in that the performance of one precluded the simultaneous performance of the other.

Since we wanted to balance the relative strengths of the two habits, several preliminary experiments were run in which the amount of practice and reward were varied. After considerable work the following general procedure was chosen, and was then used throughout the main series of experiments.

Rats were trained to run to the left[1] arm of the maze for small food reward, approximately .1 gram of wet mash being given for each trial. The animals were run 3 trials per night. To permit greater distributed practice the 3 trials were run in alternation, i.e., all animals ran their first trial, then all their second trial, etc. Since the animals were always run in groups of 6, the inter-trial interval was about 3 minutes. Training was continued in this way for 15 to 20 days. During this time no food was present in the right goal box and the rats quickly learned to run only to the left. At the end of this training, running to the left constituted an old, well-established habit.

Training on the recent habit was begun 24 hours after the last night of practice on the old habit. This newer habit consisted of running to the right for a large food reward, about 2 grams of mash being given for each trial. On the night following the last night of training on the old left habit the animals were first allowed to run 2 trials to the left as usual. The right food box was then baited with the large (2 gram) reward and training was begun on the right habit. Since the rats could not know that the right habit was now rewarded, it was necessary to lure them down the right arm of the maze.

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1. To avoid confusion in considering the results we shall always write as if the left habit were the older habit. Actually, to prevent biasing the results by position habits, half of the rats received their initial training on the right, and thus turning to the left became their recent habit.

This was accomplished by placing small food particles on the stem of the maze near the choice point and along the right arm. The particles were placed about 1 inch apart and led directly into the right goal box. As the rat ran down the stem of the T he encountered the food particles near the choice point, and in eating the successive particles was lured down the right arm and into the right goal box. He was allowed to eat the 2 gram reward and was then placed back in the starting box for the next lured trial. Each rat ran all of his trials to the right in succession, since it was found to be very difficult to change the habit preference if the method of running in alternation were used. After each rat had run 3 or 4 lured trials, fresh covers were placed on the maze and the rat was run without benefit of the olfactory cues from the food stains on the maze covers used during luring. In practically every case the animals continued to run to the right for the large reward. If not, one more lured trial was always sufficient to change the animal's preference to the right habit. After the luring trials another 1 to 3 trials to the right were run without luring. The entire right training never exceeded 7 trials, including the luring trials. The right habit was now dominant, at least temporarily, over the left habit. Preliminary experimentation had indicated that the animals would continue to run to the right until satiated. The rats had been trained, then, 3 trials per night for at least 15 nights to the left for small reward, and 4 to 7 trials, all given in one night, to the right for large reward. The right habit, although practiced much less than the left habit, was slightly dominant over the older habit because of the much greater reward associated with it. However, it must be emphasized that <u>the old habit had not been extinguished</u>, in the classical sense, for the small reward was always present in the left food box.

At varying times after the last right trial some of the rats were given

a single electroshock. Alligator-clip electrodes, wrapped in gauze and soaked in physiological salt solution, were attached to the rat's ears and 85 volts A.C. passed through the head for .2 second. In some of the experiments the experimental animals were shocked immediately (within 20 seconds) after their last right trial. In other experiments the rats waited in their cages up to 2 hours after finishing right training before being shocked. Control animals were treated in the same way as the experimental rats throughout, except that they received no shock.

We wanted to test both the immediate and the long-run effects of the shock. Therefore, in some experiments the animals were tested on the maze a half hour after the shock, while in others they were tested 24 hours after the shock. No animals were tested earlier than a half hour after shocking. It was found that the shocked animals needed at least this much time for recovery from the convulsion; otherwise they could not be induced to run. Since the control animals were tested on the maze at the same time as the shocked rats, we could determine if there had been any spontaneous recovery of the left habit. Our chief interest was in the rats' choice of turn (habit) on the first test trial. Enough test trials were run, however, to allow the animals to exhibit a definite preference, since some animals would occasionally change to the other habit after a few trials.

The animals used were male and female albino rats between 60 and 100 days of age at the time of the experiment. It is sometimes difficult to produce the full electroconvulsive attack in animals much older than this, presumably because of the increased thickness of the skull. In each experiment the animals used had had no previous experience in experimental work.

C. Preliminary experiments

Since the procedures used in the preliminary experiments were rather involved, the experiments will not be described in detail. Only the results will be summarized, since from them we derived the methods used in the main experiments.

The first problem considered was the amount of reward to be given for each of the two habits. We found that there were actually two questions to be answered: how much more food would induce the animals to change their preference from the left to the right, and how large should be the differential between the rewards to prevent frequent reversals to the left. After trying several variations in the relative amounts of food reward, we finally chose .1 gram of wet mash for the left and 2 grams for the right. With so large a differential the rats not only learned to go right in very few trials, but reversals to the left rarely occurred.

Holding the rewards constant at the values given above, the problem of balancing the strengths of the two habits was solved by varying the relative degrees of practice on each. The results of several experiments indicated that 4 to 7 trials of practice would make the right habit dominant. The right preference built up so rapidly, however, that electroshock would never induce reversals to the left habit unless the latter had had several days of practice. Therefore, in the main experiments at least 15 days of left training were given. With this amount of practice the old habit was strong enough so that reversals to it could be induced even if the new habit were dominant at the time of shocking.

The final problem concerned the time of application of the electroshock. In experiments to answer this question it was found necessary to give all of the right training in one session, and to introduce the shock no more than 2

or 3 hours after the last right trial. If more than one day of practice on the right were allowed, a long series of shocks would not cause reversals to the left. Furthermore, there seemed to be a 'consolidation' effect of the right training, since an electroshock would not cause reversals if it were administered several hours after the last right trial. Therefore, to show the effect of a single electroshock most clearly, the shock was administered within 3 hours after termination of a single session of practice on the recent habit.

In summary, then, the following results were obtained in the preliminary experiments. To produce quickly a stable change of preference from an old habit to a new habit, the former should be poorly rewarded while the latter is highly rewarded. With a highly rewarded new habit the old habit should be well practiced, and only one session consisting of few trials allowed on the recent habit; otherwise, the new habit will become so dominant that electroshock will not disrupt it. Finally, when the habits have been carefully balanced, a single electroshock will cause reversals to the older habit only if the shock is administered within a few hours after termination of training on the recent habit.

D. Main acquisition experiments

The purpose of this group of five experiments was to determine the effects of a single electroshock on a recent habit which closely matched in strength an older habit. Two factors in the situation were varied: the time elapsing between termination of training on the recent habit and the administration of the shock, and the time allowed for recovery from the shock before the test trials were run.

In the case of the first variable, how soon the shock was applied after training on the new (right hand)habit, three intervals were studied; in different experiments the shock was given immediately (within 20 seconds), one half hour, and two hours after the last trial on the recent habit. Preliminary attempts to demonstrate the effects of a single shock applied much later than two hours after right training were unsuccessful. It will be noted that at the time of administering the shock the right habit must necessarily be slightly dominant over the left. If a shock is applied within 2 or 3 hours after the right training this dominance can be disrupted, as the results of the preliminary experiments suggested. But if many hours elapse between the last right trial and the shock, test trials show that the shock has had little effect. Thus, there seems to be a 'consolidation' or 'perseveration' effect of the right training, such that the slight dominance of the right habit becomes greater without additional practice. In the main experiments, therefore, the shock occured no later than 2 hours after right training.

For the other variable, time allowed for recovery from the shock, two intervals were used; in different experiments, the test trials came one half hour or 24 hours after the shock. In this way we hoped to differentiate between immediate and long-run effects of the shock. We had hoped to run both a half hour test and a 24 hour test for each of the three shock administration

intervals. However, in the case of the shock given one half hour after right training, we were able to run test trials only one half hour after shock. Other work necessitated interruption of the series of studies before data were obtained on test trials run 24 hours after a shock which followed right training by one half hour. Thus, the experiments may be described as follows: Experiment I, shock given immediately after right training, tested one half hour later; Experiment II, shock given immediately after right training, tested 24 hours later; Experiment III, shock given one half hour after right training, tested one half hour later; Experiment IV, shock given 2 hours after right training, tested one half hour later; and Experiment V, shock given 2 hours after right training, tested 24 hours later.

The training procedure on the left habit[2] was identical in all experiments. The animals were run 3 trials per day for 15 days. The reward was .1 gram of wet mash for each trial and the trials were run in alternation.

On the 16th day the animals were trained on the right habit. The right goal box was baited with 2 grams of mash, while the left goal box still contained the small reward. The rats were lured 3 trials to the right and then run 1 to 4 trials without luring. The five experiments thus differed slightly in the total number of right trials allowed; the minimum being 4 and the maximum, 7. The reason for this variation in the number of right trials was that in some experiments a few more right trials were obviously necessary to assure dominance of the new habit.

As soon as it was decided that the right habit was dominant, training was terminated. Certain of the animals were selected to receive the shock, while the others served as controls. The experimental animals then received

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[2] It might be pointed out again that "left" and "right" serve as convenient designations for old and new habits, respectively. In practice, the old habit was to the right for half of the animals.

a single electroshock consisting of 85 volts A.C. passed through the head for .2 second. As noted above, in different experiments the shock was administered immediately, one half hour, or 2 hours after right training. All animals were allowed either one half hour or 24 hours rest and were then tested on the maze.

In the test trials an arbitrary standard of habit preference was adopted. Test trials were continued until each animal had run 4 consecutive trials to the left or 2 consecutive trials to the right. Preliminary work had indicated that only rarely did a reversal of habit preference occur after either criterion had been satisfied. Furthermore, we did not want either habit to become greatly dominant as a result of the test trials, since some of the rats were to be used in later extinction experiments in which the question of habit dominance would again be of primary importance.

Each of the five experiments employed 6 like-sexed albino rats. All animals were naive to experimental work. In each experiment 4 rats served as the Shock Group, and the other 2 as the Control Group. The small number of animals in each experiment was made necessary by the laborious training procedure on the right habit.

The results are presented in Tables 1 through 5. In the tables an S following the animal's number indicates Shock Group, a C indicates Control Group.

Experiment I

After 15 days of left training the animals were given 4 trials on the right habit. The shock was administered immediately (within 20 seconds) after the last right trial and testing occurred one half hour later. The results are presented in Table 1.

Table 1

Shocked immediately after right training, tested ½ hour later.

Rat	1S	2S	3C	4S	5C	6S
Trial 1	L	L	R	R	R	L
2	L	L	R	R	R	L
3	L	L				L
4	L	L				L

Table 1 shows that 3 of the 4 shocked animals returned to the old left habit when tested one half hour after they had been shocked. All 3 of these animals were consistent in their behavior. The other shocked rat, 4S, continued to run right. The table also shows that the one half hour rest did not affect the 2 control rats, since they continued to choose the right habit.

The results of the experiment show that a single cerebral electroshock, administered immediately after training on a recent habit, can disrupt the temporary dominance of the new habit, thereby permitting an old habit to be reasserted.

Experiment II

The training procedure was the same as in the first experiment except that 7 trials were necessary to obtain dominance of the right habit. Again the shock was administered immediately after the last right trial, but the test trials were not run until 24 hours after the shock. The results are presented in Table 2.

Table 2

Shocked immediately after right training, tested 24 hours later.

Rat	1S	2S	3S	4C	5C	6S
Trial 1	R	L	R	L	R	R
2	L	R	R	L	R	R
3	R	R		L		
4	R			L		

Table 2 shows that only one member of the Shock Group, rat 2, returned to the old habit on the first trial. The animal then reversed to the recent habit. The other 3 shocked rats chose the recent habit on their first test trial, and except for the single reversal by rat 1 on the second trial, continued to run right. The behavior of the control animals further confuses the results. Rat 5 chose the right habit consistently, but rat 4 was equally consistent in choosing the old left habit.

The results of the experiment are inconclusive. Even if it could be said that the shock did not affect the experimental animals, the behavior of the control rats remains unexplained.

Experiment III

Again there was no variation in training procedure except that 5 trials were run on the right habit. In this experiment the animals were shocked one half hour after the last right trial and tested one half hour later. The results are presented in Table 3.

Table 3

Shocked ½ hour after right training, tested ½ hour later.

Rat	1S	2C	3S	4C	5S	6S
Trial 1	L	R	L	R	L	L
2	L	R	R	L	L	R
3	L		R	R	L	R
4	L			R	L	

Table 3 shows that the shock caused a return to the old left habit in all 4 of the shocked rats on the first test trial. Although rats 1 and 5 were consistent, the other shocked rats, 3 and 6, changed back to the recent habit on the second trial. Except for one reversal on the second trial by rat 4, both control animals continued to choose the new habit after the one hour rest.

The results of this experiment show that even if one half hour of rest is permitted before the shock is applied, the new habit is disorganized when testing occurs one half hour after the shock.

Experiment IV

The training procedure was again the same with 5 trials being given on the right habit. The animals were shocked 2 hours after the last right trial and tested one half hour later. The results are presented in Table 4.

Table 4

Shocked 2 hours after right training, tested ½ hour later.

Rat	1S	2C	3S	4C	5S	6S
Trial 1	L	L	R	R	L	L
2	L	R	R	L	L	L
3	R	L		R	L	L
4	R	R		L	L	L
5		L		R		
6		R		L		
7		R		R		
8				L		
9				R		
10				R		

Table 4 shows that 3 of the 4 shocked rats returned to the old habit after the shock. Rat 3S, however, showed no loss of the new habit. Of the 3 Shock Group rats showing loss of the recent habit, number 5 and 6 were consistent, while rat 1 changed back to the right habit on the third trial. The two control animals did not react in the same way. The table shows that rat 2 went left on the first test trial, while rat 4 went right. Furthermore, for the first time we encounter animals needing more than 4 test trials to show a definite habit preference by our standard, 4 consecutive L or 2 consecutive R. Before the right habit was finally chosen, rat 2C ran 7 trials and rat 4C ran 10 trials. The behavior of the controls is particularly

difficult to explain in view of the fairly consistent behavior of the experimental rats.

Although the behavior of the control rats reduces the conclusiveness of the experiment, the results with the shocked rats indicates that a new habit may be disorganized when the shock is administered 2 hours after termination of training on the recent habit.

Experiment V

Training was again the same as in the previous experiments with 7 trials being allowed on the recent habit. As in Experiment IV the rats were shocked 2 hours after the last right trial, but test trials were not run until 24 hours after shocking. The results are presented in Table 5.

Table 5

Shocked 2 hours after right training, tested 24 hours later.

Rat	1S	2S	3S	4C	5C	6S
Trial 1	L	L	L	R	R	R
2	R	R	R	R	L	R
3	R	R	R		L	
4					R	
5					R	

Table 5 shows that 3 of the 4 shocked rats returned to the old left habit on the first test trial. All 3 of these animals, however, changed back to the recent habit on the second trial. The other member of the Shock Group, rat 6, was not affected by the shock. Both of the control animals showed a right preference on the first trial; in rat 4 this preference was stable, but rat 5 showed some alternation of response.

The results of this final acquisition experiment show that the effects of a single electroshock, administered 2 hours after training on the recent habit, can be demonstrated as long as 24 hours after the shock.

Discussion

Although the data are occasionally inconclusive, the results suggest, on the whole, that a single cerebral electroshock can weaken a recent habit, thereby permitting an old habit to regain dominance. We would agree with Rodnick (35) that the loss of the recent habit may be only temporary, and would emphasize that this is particularly true when only one electroshock has been given. In shock therapy with mental patients the tendency has been to give many shocks in a course of treatment before the therapist concludes that the patient has not benefited. Ziskind (54) has pointed out that termination of the treatment after only a few shocks has probably colored statistics on remissions unfavorably. It may well be that in the present maze experiments a series of shocks would have abolished the recent habit completely, provided that the series of shocks began soon after the training on the recent habit. However, we would not support such a prediction without experimental evidence. Furthermore, the use of a series of daily shocks would introduce complications, especially when one is dealing with habits of different ages. A 'recent' habit is obviously not very recent if its acquisition and testing are separated by a week of shocks. On the other hand, testing the rat after each shock would reinforce one or the other habit. Hence, it seemed best to limit the experiment to one shock.

The fact that many of the animals in our experiments returned to the recent habit after showing an initial reversal to the old habit indicates that the effect of a shock is to disorganize rather than to destroy the recent memory traces. This conclusion has also been reached by Zubin (57) in his experiment showing that in human patients electroshock accentuated rather than minimized 'interference' effects.

In running test trials one half hour after a shock it was noted that the

shocked rats often appeared confused. At times a shocked animal would not leave the starting box. However, if the animal was pushed out onto the maze, he would then run to one of the goal boxes without further urging. In trials preceding the shock the entire run from starting box to goal box occupied only a few seconds, and almost never did hesitation at the choice point occur. But rats tested one half hour after a shock proceeded very slowly along the maze and often paused several minutes at the choice point before turning left or right. Although no quantitative record was kept, considerable VTE behavior at the choice point was observed in animals run one half hour after a shock. Much less VTE was exhibited in animals run 24 hours after the shock, and never was any hesitation or slowness in running observed. If generalizations can be made to shock therapy in human patients, we would emphasize that a course of psychotherapy should run concurrently with a course of shock therapy. We have previously cited Rodnick (35) to the effect that results with shock therapy appear to be better when the shock treatments are supplemented with psychotherapy. If we accept the evidence, presented in these and in previous experiments, that shock weakens but does not destroy recent habits which conflict with older habits, then any method (psychotherapy, retraining, guidance, etc.) which aids in the return to dominance of the older habit should be valuable. In our animal experiments no inducement to choose one or the other habit was employed. Such inducement could not have been introduced without biasing the basic method of the experiments. Therefore, in both human and animal subjects that have been subjected to shock the situation appears to be about as follows. At the time the shock or series of shocks is introduced the recent habit is more rewarding. Only in this way can we explain the persistence of the psychotic symptoms or the preference for the right habit. When now the rat or patient is shocked the recent habit is disorganized but by no

means destroyed. The subject is now allowed a choice of habit. Whether through confusion, amnesia, or punishment there is no strong preference for the recent habit. At the same time, however, nothing has been done, at least directly, to induce the organism to choose the old habit. Any choice of the old habit rests entirely on the state of disorganization produced in the new habit by the shock. If the organism is allowed to choose freely which habit it prefers, it may frequently prefer the older habit with no added inducement. However, as has been reported with mental patients and as was observed with our rats, choice of the old habit is frequently only temporary, and the organism soon returns to the more recently learned response. But if during the period of recovery from the shock some encouragement is given to relinquish the recent habit in favor of the old, choice of the old response may become much more stable than if the organism is allowed to choose freely.

We emphasized that at no time in the experiments here reported was the old habit entirely unrewarded. Extinction of the old response, at least in the classical sense of Pavlov, did not occur. Rather the new habit was so much more rewarding that the goal of the old response was overshadowed. If we accept the view that psychotic behavior is a response to frustration of normal modes of behavior, then the present maze procedure probably parallels the situation with humans. It seems likely that when a human relinquishes normal behavior patterns he does so not because these patterns are extinguished by lack of reinforcement, but because abnormal patterns are more highly rewarding relative to the reward value of normal behavior. We would not want to press this distinction too far, but it may be noted that some organisms will continue to work for poor rewards, while none will persist indefinitely in the face of complete lack of reward. It sometimes happened in our experiments that a rat would not learn the right habit in spite of our best efforts. This may be analogous to

those situations where some humans will continue to react with normal though poorly rewarded responses, while others subjected to the same conditions will choose abnormal behavior patterns which to them are more rewarding.[3] This point is illustrated very clearly in Grinker and Spiegel's discussion of war neuroses (11). It should be emphasized, however, that we do not consider the right habit on the T maze any more 'normal' or 'abnormal' than the left.

The effects of a single electroshock are probably very slight, except for the immediate effects of unconsciousness and confusion. In a way it is surprising that we were able to demonstrate any behavior changes resulting from a single shock, especially in those experiments where 24 hours were allowed for recovery. As far as the author knows, no case has been reported where a single shock treatment produced recovery from a psychosis. Furthermore, the habits used in our experiments were relatively simple for the rat. In reviewing the work done on animals with shock we pointed out that the majority of writers who used simple habits found no effects from shock, whereas in almost all the experiments in which a more difficult habit was used, some loss of the habit resulted from the shock. What success we did achieve is probably to be explained in terms of a delicate balance between the two habits. The shock could disturb this balance by a slight disruptive effect on the newer habit. It may be well to point out here that it was difficult to equate the two habits in strength. The fact that in some animals a single shock did not cause return to the old habit probably indicates that the new habit was strong enough to resist disorganization.

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3. Our interpretation of abnormality as a 'progression' from normal to maladjusted patterns of behavior would seem to conflict with the Freudian theory of mental disorder as a 'regression' to earlier modes of adjustment. However, the regression theory is confused, and is not subscribed to by all psychopathologists.

Any conclusions drawn from these five acquisition experiments are to be considered as tentative only. To obtain results that are reliable in a statistical sense large numbers of animals would be required. The time involved in training an adequate sample of animals would be prohibitive. However, a summary of the results suggests the following conclusions.

A single cerebral electroshock can often weaken a recent habit which conflicts with an older habit, so that the old habit again becomes dominant. This effect is difficult to demonstrate if the shock occurs more than a few hours after training on the recent habit. The effect is usually only temporary, the recent habit again becoming dominant. It is possible that a shocked animal would return to the old habit more frequently and more persistently if added inducement were given over and above the disorganizing effect of the shock. We can perhaps generalize from this conclusion and suggest the value of a course of psychotherapy along with shock therapy in human patients. In continuing to reward the old habit while attempting training on the new habit we have probably paralleled the situation with humans where the normal response patterns are continually, though poorly, rewarded, while at the same time abnormal patterns are highly rewarded. Finally, the permanent effects of a single electroshock are probably slight, particularly on rather simple habits.

E. Main extinction experiments

The series of main acquisition experiments demonstrated that a single cerebral electroshock could weaken a newly acquired habit to the extent that an older conflicting habit often regained dominance. In view of these results on acquisition we may ask if electroshock has the same disruptive effect on extinction. Gellhorn has attempted to answer this question in a series of experiments utilizing the avoidance conditioning situation (7, 8, 9, 15). Although we have already reviewed Gellhorn's work briefly, we shall consider his experiments in more detail later, since his results do not completely agree with those to be reported here.

The purpose of the extinction experiments was to determine if electroshock would weaken the internal inhibition of the right habit, when this habit had been extinguished by omitting reinforcement. It may be well to point out again that this is the first time we induce habit reversal by the classical technique of extinction. In the acquisition experiments the animals were lured to the new habit while the old habit remained rewarded. In preliminary training for the extinction experiments this is again the case, but when we wish to induce a second habit reversal, from the new habit back to the old, this is accomplished by discontinuing reinforcement of the new habit. We therefore produce internal inhibition of the new habit by the classical method and study the effect of electroshock on this inhibition.

The training procedure in the series of extinction studies was similar to that used in the acquisition experiments. Animals were trained to the left on the T maze for several days, receiving .1 gram of food for each trial. The animals were then trained on the right habit for 2 grams reward, using the luring procedure described above. After the right habit was well established, the food reward was removed from the right goal box. Extinction trials were

run until all animals had returned to the old left habit. Throughout the entire experiment the left habit was always reinforced with the small reward. After the right habit had been inhibited, some of the rats were given one cerebral electroshock. All animals were tested on the maze one half hour after the shock. Since details of the procedure varied from one experiment to another, the exact method will be reported for each experiment separately.

Experiment I

Six rats were trained to the left for 20 days, running 3 trials per day. The reward was .1 gram of wet mash for each trial. On the 21st day the rats were lured to the right by placing small particles of food along the right arm. The reward on the right was 2 grams of mash. On this first retraining day the animals were run 3 lured and 1 unlured trials. In order to strengthen the new right habit, 3 more days of training were given. On each of these days 3 trials were run to the right. Thus, the animals had received 60 trials in 20 days to the left and 13 trials in 4 days to the right. All animals were then given a 10 day rest. They were tested on the maze after the 10 day rest, at which time it was found that the right habit was still dominant in all 6 rats. On the following night the right habit was extinguished. Food was removed from the right goal box and the animals were run 20 extinction trials each. For all rats the last 6 of the 20 extinction trials were consecutively to the left. Thus, it appeared that the right habit was completely extinguished, at least for the moment. Immediately following the 20th extinction trial rats 2, 4, and 6 were given an electroshock. Again the shock consisted of 85 volts A.C. passed through the head for .2 second. Rats 1, 3, and 5 served as controls. All animals were tested on the maze one half hour after the shock. During the test trials, both habits were rewarded with their usual amounts of

food.

The results are presented in Table 6 below. In the table, an S indicates Shock Group, a C indicates Control Group.

Table 6

Shocked immediately after 20th extinction trial on the right, tested ½ hour later.

Rat	1C	2S	3C	4S	5C	6S
Trial 1	L	L	L	L	L	L
2	L	L	L	L	L	L
3	L	L	L	L	L	L
4	L	L	L	L	L	L

Table 6 shows that the one half hour rest did not cause spontaneous recovery from the extinction in the control rats. Furthermore, the single shock did not weaken the inhibition of the right habit in the shocked rats. All 6 rats continued to go to the left.

On the following night the rats were again tested on the maze to determine if the right habit was still extinguished. The test showed that the right habit was still inhibited; all animals ran to the left. All 6 animals were then given one shock and tested one half hour later. Again all rats continued to run left after the shock.

The results of this experiment show that a single electroshock could not weaken the internal inhibition produced by extinction of the right habit. However, the results also show that the right habit was rather thoroughly extinguished, since no spontaneous recovery occurred in the control rats. Perhaps the shock could show an effect if the extinction were not so complete. Evidence on this point is presented in the experiments below.

Experiment II

The training procedure on both the left and right habits was the same

in this experiment as in Experiment I. Again a test after the 10 day rest showed the right habit still dominant. On the following night the right habit was extinguished. In this experiment only 16 extinction trials were run, the last 2 of which were consecutively to the left for all rats. Immediately after the 16th extinction trial rats 1, 2, and 4 were given one shock. Rats 3, 5, and 6 were controls. All animals were tested one half hour later. The results are presented in Table 7.

Table 7

Shocked immediately after 16th extinction trial on the right, tested ½ hour later.

Rat	1S	2S	3C	4S	5C	6C
Trial 1	L	L	L	R	L	L
2	L	L	L	L	L	L
3	L	L	L	L	L	L
4	L	L	L	L	L	L

Table 7 shows that the shock had no effect in weakening the inhibition of the right habit except in rat 4 on the first trial. The table also shows that the one half hour rest did not produce spontaneous recovery in the control rats.

On the following night the rats were tested to determine if the effects of extinction on the right habit had worn off. Again all animals continued to run to the left. It thus appeared that the right habit had been completely inhibited and it was not considered worthwhile to test the effect of another shock.

The results of the experiment show that even with only 16 extinction trials on the right habit, neither shock nor spontaneous recovery weakens the inhibition.

Experiment III

The procedure in this experiment varied slightly from that of the previous experiments. Only 15 days of left training were given, followed by 3 days of right training. On the last (3rd) day of right training the animals were given 5 rewarded right trials. Food was then removed from the right goal box and extinction on the right begun. The method of giving an equal number of extinction trials to each rat, as in the previous 2 experiments, was abandoned. In this experiment each animal was run until he had made 3 consecutive left choices, at which time the right habit was considered to be inhibited. Immediately after the 3rd left trial for each rat he was either given a shock or put back in the living cage. All animals were tested on the maze one half hour after the shock or the last extinction trial.

Since in this experiment we are using 3 consecutive trials to the left as a criterion of extinction of the right habit, the number of extinction trials is not the same for all rats. The number of extinction trials for each rat, including the last 3 criterion trials, is presented with the test trials in Table 8.

The Shock Group consisted of rats 1, 3, 5, and 6. The Control Group was rats 2 and 4.

Table 8

Shocked immediately after 3 consecutive left trials, tested ½ hour later.

Rat		1S	2C	3S	4C	5S	6S
No. of trials to extinction		7	13	10	11	14	6
Test trial	1	L	R	L	R	R	L
	2	L	R	L	R	R	L
	3	L		L			L
	4	L		L			L

Table 8 shows that in 3 of the 4 shocked rats the shock did not remove the extinction of the right habit. Only rat 5S returned to the right after the shock. Surprisingly enough, however, both control animals showed spontaneous recovery from the extinction after the half hour rest; when tested they ran to the right.

It may also be seen from the table that the 3 animals that showed no loss of the extinction, rats 1S, 3S, and 6S, ran fewer extinction trials than the 3 animals that returned to the right. The interpretation of this fact is not particularly clear; perhaps those animals needing more extinction trials had a stronger right preference. In any case, as will be seen below, repetition of the experiment gives exactly the same results on the test trials, even though the number of extinction trials differs.

On the following night the animals were retrained on the right. If necessary, the animals were lured; all were run until each had completed 6 right trials, at which time the right habit was again dominant. Immediately after the last right trial all rats were again extinguished on the right. Again the criterion of extinction was 3 consecutive left trials.

Immediately after the last extinction trial for each rat he was either shocked or put back in the living cage. The Shock Group and the Control Group were the same as the previous night. All animals were tested one half hour after the shock or the last extinction trial. The results on the test trials, as well as the number of trials to extinction, are presented in Table 9.

Table 9

Retrained on right, extinguished to 3 consecutive left trials, shocked immediately, tested ½ hour later.

Rat	1S	2C	3S	4C	5S	6S
No. of trials to extinction	19	14	16	11	14	8
Test trial 1	L	R	L	R	R	L
2	L	R	L	R	R	L
3	L		L			L
4	L		L			L

It is striking to notice that the results of Table 9 are identical with those of Table 8. Although a Mean of 13.7 extinction trials was necessary as compared to a Mean of 10.1 on the previous night, the behavior of each rat was the same when tested one half hour after the shock or the last extinction trial. The same 3 animals, rats 1, 3, and 6, showed no effect of the shock, while rat 5 returned to the right. Again the one half hour rest produced spontaneous recovery in the control rats, since both returned to the right.

The results of this last extinction experiment would seem to indicate that when the extinction of the new right habit is not too thorough the inhibition may be weakened by spontaneous recovery, thus allowing the new habit to regain dominance. On the other hand, the shock seems to accentuate rather than diminish internal inhibition, since 3 out of 4 of the shocked animals continued to go left while the control animals were showing spontaneous recovery. An alternate explanation might be that the shock interferes with spontaneous recovery. We see no clear way of differentiating between these two alternatives.

Discussion

Taken as a whole the results of this series of experiments on the effect

of electroshock on acquisition and extinction do not corroborate the conclusions of Gellhorn (7, 8, 9, 15). In one paper (8) this author takes issue with the results of Rosen and Gantt. The latter workers had found that ten metrazol convulsions led to an impairment of differentiating ability in dogs, as shown by the conditioned response technique (37). Gellhorn contradicts Rosen and Gantt on the basis of his own experiments. In one of Gellhorn's studies (8) two or three conditioned responses were successively established. Using the avoidance situation with rats, the animals were first conditioned to one conditioned stimulus. This response was then inhibited by omitting reinforcement, following which the animals were trained to a different conditioned stimulus. Thus two or three responses were successively conditioned in the same animal, one or two being inhibited while the other was maintained at one hundred percent. When this had been accomplished, electroshocks or insulin comas were administered to the animals. It was found that the positive CR remained unaffected, whereas the inhibited responses returned to a high positive level. On the basis of these results Gellhorn argues that a diminished ability to discriminate, as Rosen and Gantt observed, would lead one to expect not only an increase in the response of previously inhibited reactions but also a diminution in the response of positively established reactions[4] The fact that such a diminution was never seen by Gellhorn leads him to conclude that shock procedures diminish inhibitory processes and enhance excitatory processes.

Although in our experiments we used a different type of learning situation, it should be pointed out that our results contradict Gellhorn's conclusions. In the acquisition experiments we found that an electroshock weakened the excitatory processes associated with the new right habit, thereby allowing the old habit to regain dominance. Furthermore, it would not be strictly

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4. The present author does not necessarily agree with Gellhorn's deductions.

accurate to say that the shock had merely removed the inhibition associated with the old habit, since inhibition in the sense that Gellhorn has used it was not present. Instead, the old habit had been actively blocked by a newer and stronger one.

Our results with the extinction experiments also fail to corroborate Gellhorn. In the extinction studies the classical type of internal inhibition was produced by omitting reinforcement of the right habit. In this situation Gellhorn might predict that shock would remove the inhibitory effects, thus allowing the right habit to become dominant once more. This prediction was not borne out. It might be argued that in Experiments I and II of the extinction series the internal inhibition was too strong to be affected by a single electroshock, in view of the fact that neither one half hour or 24 hours rest produced any spontaneous recovery in the control animals. But this is not true of Experiment III. Here the mean number of extinction trials was less than in the two previous experiments. Furthermore, the control animals showed spontaneous recovery of the right habit after the one half hour rest. But in 3 out of 4 of the shocked rats the shock seemed to accentuate rather than to weaken the inhibition, since they continued to show extinction of the right habit when tested after shock.

Although we must emphasize again the differences in method between Gellhorn's experiments and ours, it appears that we cannot accept his conclusions without some qualification. If suggestions concerning the theory of shock therapy are to be drawn from animal experimentation, we must make such suggestions only on the basis of consistent results. This is particularly important in view of the fact that Gellhorn has evolved a rather elaborate theory of the mechanism of shock therapy which draws much of its support from his work on the effect of shock on conditioned responses. Furthermore, the

dangers of generalizing from results on "normal" rats to "abnormal" human beings are great.

We may conclude from our T maze experiments that electroshock apparently weakens excitatory processes. With less assurance the suggestion is made that shock seems to have little effect on inhibitory processes, except perhaps to accentuate them slightly. It is to be noted that these conclusions are not in agreement with those of some other investigators. This disagreement may be due to the small numbers of animals used and the methodological difficulties involved in our experiments.

IV. Conditioned Avoidance Experiment

A. Introduction

The results of the maze studies indicated that a single electroshock could disrupt a recently acquired habit. During the preliminary work for those experiments it was observed that the time between the termination of training on a habit and the application of the electroshock was important in determining the effect of the shock. Thus, in the maze acquisition experiments, if the shock was administered more than 3 or 4 hours after training on the recent right habit, later test trials showed no effect of the shock. No very careful determinations were made of the effect of varying the time of interpolation of the shock in the maze work. It is the purpose of the present experiment to investigate this variable more thoroughly.

The study to be reported measures the result of varying the time between the completion of each trial in an avoidance situation and the administration of the electroshock. This method should enable us to determine the retroactive effect of electroshock on learning. Patients undergoing electroshock treatment frequently report a retrograde amnesia as a result of the shock. By varying the time between completion of a response and application of the shock we had hoped to measure the backward temporal spread of retrograde amnesia. The results of the experiment reported below show a definite retroactive effect of the electroshock. In the discussion we shall consider the question as to whether our results are related to retrograde amnesia in the clinical sense.

Since we wanted to vary the time interval studied over a range of a few seconds to several hours, it was necessary to employ a situation in which the behavior of the animals was fairly well controlled by the experimenter. With

the maze the behavior in any one trial may show considerable variability both in time consumed and in responses made. Therefore, for the present experiment the method of conditioned avoidance was employed. In the typical avoidance apparatus the animal escapes from an electrically charged grid into a safe compartment. After a few trials the animal responds before the grid is charged; i.e., he avoids the charge. For our purposes the avoidance method has the advantages that the behavior is relatively simple and that the animal is forced to respond quickly to escape or avoid the grid. Thus, we are able to measure with fair accuracy the time elapsing between the termination of a single trial and the application of an electroshock, and can vary this interval over a wide range.

B. Apparatus and Procedure

The apparatus consisted of a box, 14" long, 10" wide, and 9" high. The box was divided into two compartments by a partition extending from side to side and reaching from the floor to the top. One side of the apparatus consisted of a plastic window through which the animals could be observed. The floor of the grid compartment was a series of quarter-inch steel rods, spaced half an inch apart. The floor of the safe compartment was wire mesh. A small opening in the partition allowed the animals to run from the grid compartment to the safe compartment.

The sides and top of the grid compartment and the floor below the grid were painted flat black. All surfaces of the safe compartment were white. A 100-watt bulb enclosed in a light-proof housing shone directly through the plastic window into the safe compartment. Thus, with low room illumination, the grid compartment was quite dark while the safe compartment was well lighted. Since the grid was not charged until 10 seconds after an animal had been placed in the grid compartment, lighting the safe compartment prevented the animals (albino rats) from wandering into the safe side, particularly in the early trials.

The grid was charged from the output of a 220 volt stepup transformer and a variable potentiometer. A 350,000 ohm resistance in the output of the potentiometer reduced to a minimum effects of changes in the rat's resistance. The apparatus is shown, somewhat schematically, in Figure 3.

The animals used were albino rats between 60 and 100 days of age at the beginning of the experiment. As noted above, if rats much over 100 days of age are employed, the standard electroshock is not always successful in producing a convulsion.

The electroshock apparatus has already been described and is shown in

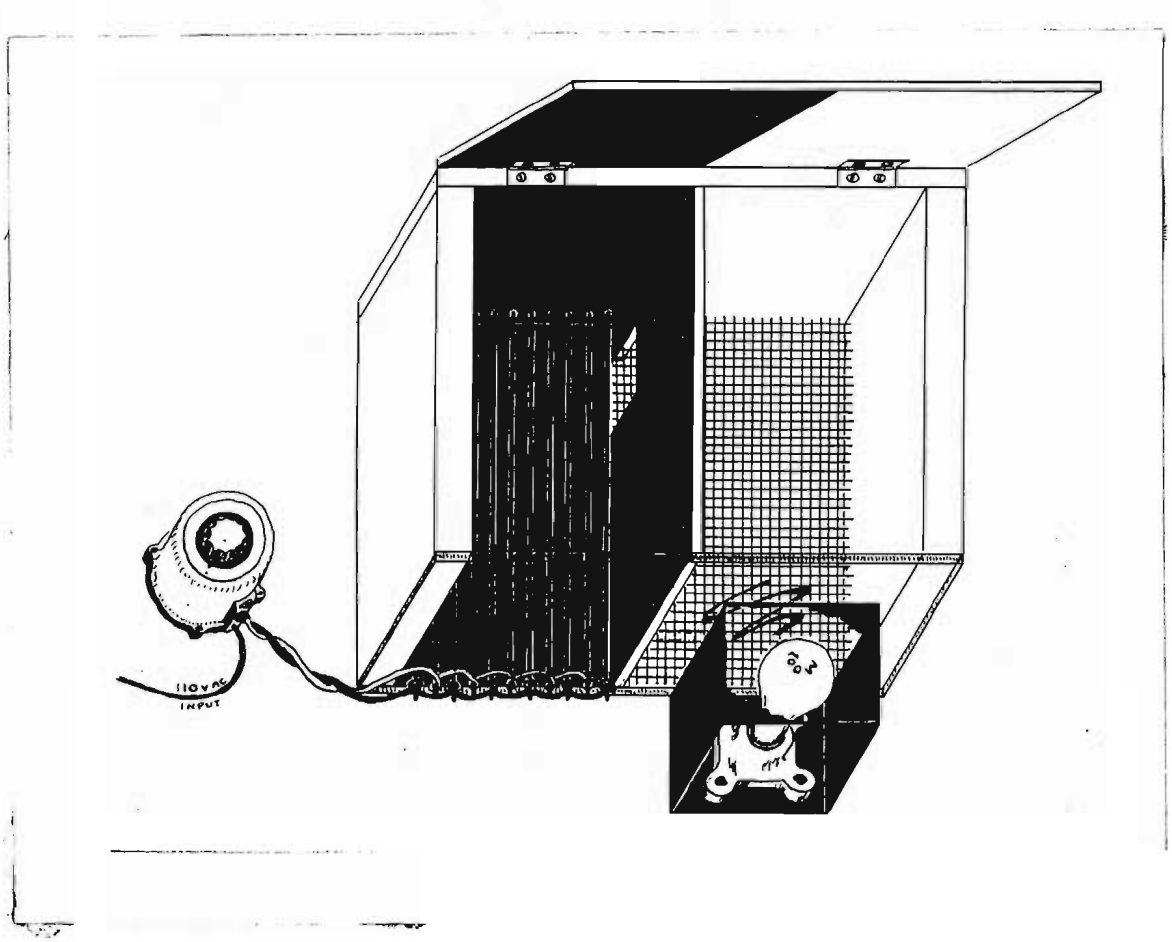

Figure 3

The avoidance apparatus

Figure 2. Throughout the avoidance experiment the electroshock consisted of 85 volts A.C. passed through the head for .2 second.

The procedure in the avoidance apparatus was the same for all Experimental Groups and the Control Group. On the first day of training an animal was placed on the grid and the cover of the avoidance box was put in place. The animal was allowed to explore the apparatus for at least 2 minutes and until he had made one or more trips into the safe compartment. At some convenient time when the animal was standing on the grid, the charge was applied. By means of the transformer the voltage on the grid was varied in an attempt to get a stimulus strong enough to drive the animal into the safe compartment but not strong enough to evoke violent jumping. The time from the application of the grid charge until the animal's body had passed through the door of the partition into the safe compartment was recorded as a latency.

On the following day the animal was again placed on the grid and at the same time a watch was started. When the watch had reached 10 seconds the grid was charged and the watch was stopped when the animal had run into the safe compartment. Thus, the animal had 10 seconds in which to run and avoid the shock. Cerebral electroshock may produce a depression of general activity level and a lowered running speed in some animals. By using a CS-US interval of 10 seconds these effects were minimized, as the results will show. No matter whether the animal avoided or escaped the grid; i.e., whether the animal ran anticipatorily or waited until the 10 seconds were up, the grid was charged at all times when the animal was in the safe compartment, thus preventing the rat from returning to the grid. This procedure, allowing the rat 10 seconds in which to run before the grid was charged, remained constant throughout the rest of the experiment. The animals were given one trial per day and the experiment continued for 18 days.

There were 8 Experimental Groups. At a certain time after the animal had run into the safe compartment, the time differing for each group, a cerebral electroshock was administered. The electroshock was administered at the same time after each of the first 17 trials; the experiment was then terminated after the 18th trial. The Experimental Groups were as follows: For Group I the electroshock followed each trial immediately (within 20 seconds); for Group II the electroshock followed each trial by 40 seconds; for Group III, 60 seconds; for Group IV, 4 minutes; for Group V, 15 minutes; for Group VI, 1 hour; for Group VII, 4 hours; and for Group VIII, 14 hours. A Control Group was run in the same way as the Experimental Groups, but received no cerebral shock. Hereafter, each experimental group will be referred to by the time which elapsed between the daily trial and the application of the electroshock in that group.

The measure of learning was the number of anticipatory runs; i.e., the runs in which the animal avoided the grid shock by running before the 10 second CS-US interval was up. Our hypothesis was that if the cerebral electroshock had a retrograde amnesic effect this would be shown by a failure of certain of the Experimental Groups to anticipate the grid charge, since they would not "remember" from one trial to the next that the grid was punishing. Thus, by varying the time between the completion of each trial and the interpolation of the electroshock in different groups, we should obtain a measure of the duration of the retroactive effect. Presumably, with a long enough interval between completion of the daily trial and application of the electroshock, the retroactive effect of shock would not extend back to the time of day when the trial was run, and the animals in that group would learn to anticipate as well as the controls. We should not expect a definite break between groups learning to anticipate and those showing no evidence of learning. More probably a gradient of anticipation will occur.

C. Results

Using the number of anticipatory runs as the measure of learning, i.e., the number of runs in which the animal ran with a latency of less than 10 seconds and thus avoided the grid shock, the data in Table 10 were obtained. In the table the 8 groups that received cerebral electroshock are designated by the time which elapsed between each trial and the application of the shock. The group called "Immediate Group" indicates animals that were shocked through the head as quickly as the experimenter could remove the rat from the safe compartment of the avoidance apparatus after the daily trial and apply the electrodes. In no case did this require more than 20 seconds. The table also includes the data for the Control Group, which received no cerebral shocks.

Table 10

Data for anticipatory responses for all 9 groups. Each animal ran 18 trials at the rate of 1 trial per day.

Group	No. of animals*	Mean anticipatory responses	SD	SE_M
Immediate	11	2.54	2.71	.85
40 second	7	5.85	2.52	1.02
60 second	9	8.00	2.20	.77
4 minute	9	9.11	4.70	1.66
15 minute	10	10.20	2.36	.79
1 hour	6	12.33	1.52	.68
4 hour	6	12.16	3.05	1.36
14 hour	15	12.66	2.55	.68
Control	18	12.00	2.21	.53

*The number of animals which began the experiment for each group (in the same order as the groups appear in the table) is as follows: 12, 12, 12, 12, 12, 6, 6, 18, and 18. However, deaths due to the cerebral electroshock reduced the number of animals in every group, except, of course, in the Control Group.

Table 10 clearly shows the gradual increase in the mean number of anticipatory responses as the interval between the daily trial and the application of the electroshock increases. We may represent these results graphically by plotting the mean anticipatory runs as a function of the logarithm of the time between the completion of each trial and the electroshock. When this is done the curve shown in Figure 4 is obtained. The marked retroactive effect of electroshock on learning, particularly in the early groups, shows distinctly in this graph. At the same time the steep slope of the line connecting the means indicates that this retroactive effect drops off rapidly as more time is allowed between each day's run and the administration of the shock. Actually, within the limits of the number of groups used, no retroactive effect of the shock is found when the shock is administered more than 15 minutes after the day's trial; no depression of learning ability was found in the one hour, four hour, or 14 hour groups.

The significance of the differences between the means of each of the experimental groups and the Control Group was determined by the t test. It was found that the Immediate, 40 second, 60 second, 4 minute, and 15 minute Groups were significantly different from the Control Group at, at least the five per cent level of probability. The one hour, four hour, and 14 hour Groups were not significantly different from the controls.

Since Figure 4 presents only the mean anticipatory runs for all 18 trials, it does not show any possible progressive changes in behavior over the experimental period. To show the progress of learning, therefore, Figure 5 was constructed. In this graph the period of learning is broken up into units of three trials each. Thus, any point on the graph represents the mean anticipatory runs during the three-day interval indicated on the abcissa.

The curves in Figure 5 show that three different types of learning

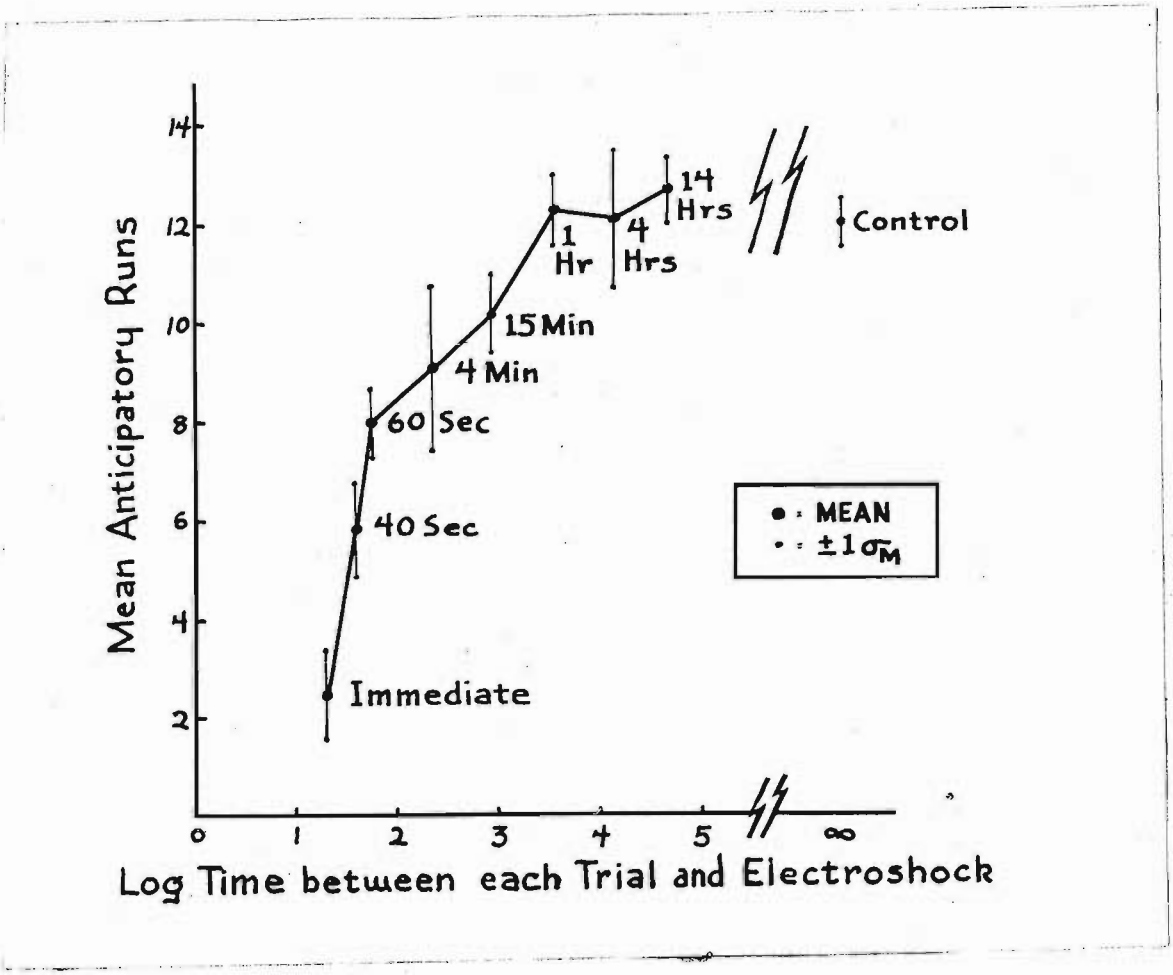

Figure 4

Mean anticipatory runs for all 18 trials as a function of the logarithm of the time between each trial and the application of cerebral electroshock.

function were produced by the electroshock. The first type is shown by the one hour, four hour, and 14 hour groups. Electroshock had no detrimental effect on these three groups; they learned as well as the controls. We feel justified, therefore, in plotting on the graph only the range of means of these four groups, with a dotted line indicating the size of the range at each abcissa point. The solid line connecting the midpoints of these ranges is above all other curves except at days 1-3, where all groups showed little evidence of learning, and at days 16-18, where the range overlaps the mean of the 15 minute Group..

The second type of response to the electroshock consisted of a depression in the rate of learning over the entire experimental period. This effect of the shock is characteristic of the 15 minute, the 4 minute, and the 60 second groups. None of these 3 groups learned as well as the controls except, as noted above, the 15 minute animals on days 16-18. The 4 minute animals progressed more slowly than the 15 minute animals, with one inversion on days 10-12. The 60 second group learned even more slowly than the 4 minute group except on days 4-6, where a plateau begins. This plateau in the learning of the 60 second group continues until days 10-12, after which there is again improvement.

The Immediate and 40 second groups show a third type of response to the shock. After some initial improvement the curves of these two groups show a definite drop late in learning. The 40 second animals learn more rapidly at first, but they eventually show the same fall in the learning curve as is shown by the Immediate animals.

If the curves in Figure 5 are representative of real differences in response to electroshock, the mechanism underlying the behavior should be of interest. Later in the discussion we shall present a tentative explanation

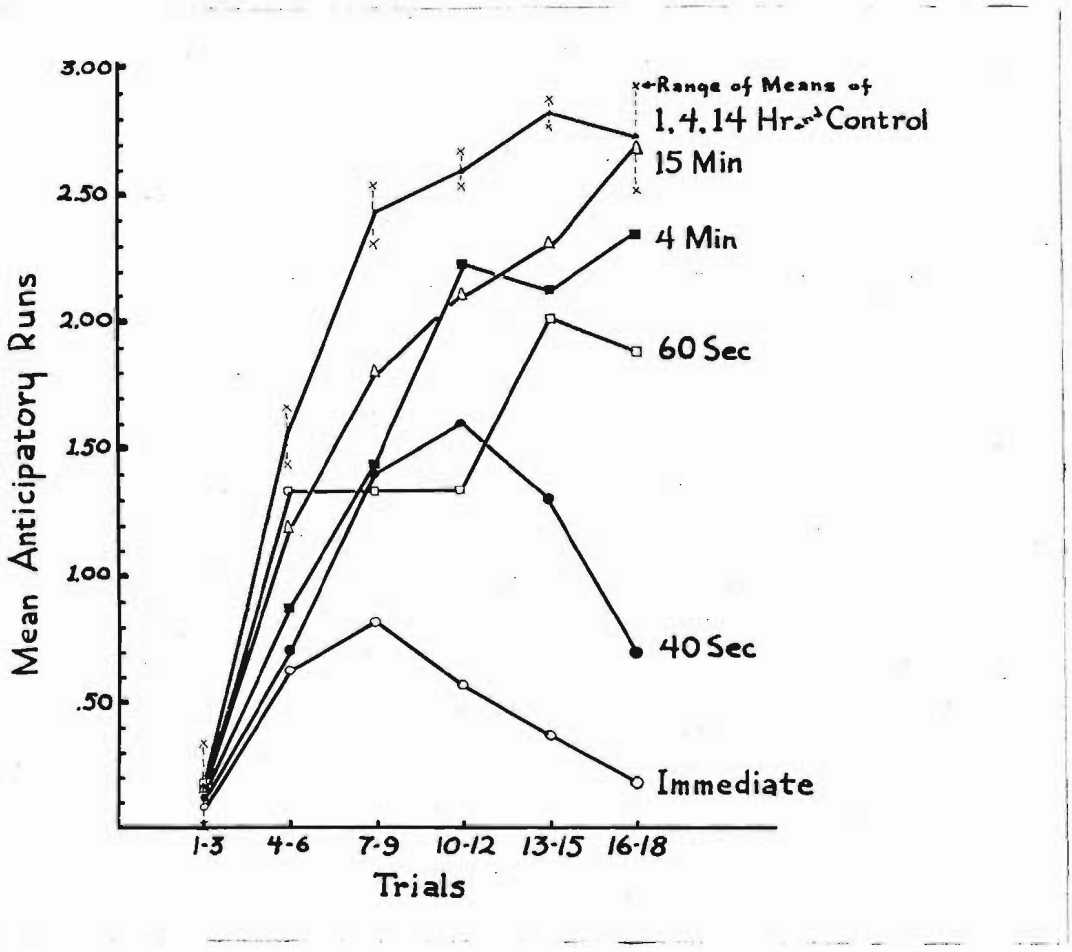

Figure 5

Mean anticipatory runs as a function of trials. The 18 trials are divided into 6 blocks of 3 trials each.

for the differences among the groups.

Little information of value could be derived from the data on latency of running. In attempting to treat latencies to obtain a useful measure of response, difficulty was encountered because of the cut-off score at 10 seconds. Since the grid was charged 10 seconds after the animal had been placed in the apparatus, latencies of runs occurring before 10 seconds are not strictly comparable to latencies longer than 10 seconds. However, purely for purposes of illustration, we have presented in Figure 6 the percent frequency of occurrence of each latency plotted as a function of latency in seconds. Only latencies up to 19 seconds are shown, since any longer latencies occurred very rarely. If all 9 groups of animals are represented, the graph becomes almost impossible to read; we have therefore presented only the data for the Immediate, 40 second, and 14 hour groups, and the Control Group. Tables in an appendix show the latencies of all trials for all animals.

The conditioned latencies of the four groups in Figure 6 show some overlap. The maximum percent frequency for the Control Group occurs at 2 seconds, the maximum for the 14 hour Group is at 1 second, and the maximum for the 40 second Group is at 3 seconds. The Immediate Group shows no particular maximum in the avoidant latencies.

Sharp differences among the groups appear at the 11 second escape latency. Since latency was recorded from the instant the animal was put into the apparatus, 11 seconds indicates that the animal arrived in the safe compartment 1 second after the grid had been charged. Although the 14 hour and the Control animals show a relatively high frequency at 11 seconds, the pile-up is not nearly as great as it is for the Immediate and 40 second animals; for these latter two groups the percent frequency at 11 seconds falls far off the graph with the ordinate scale used. Beyond 11 seconds the frequency drops off rapidly for all four groups.

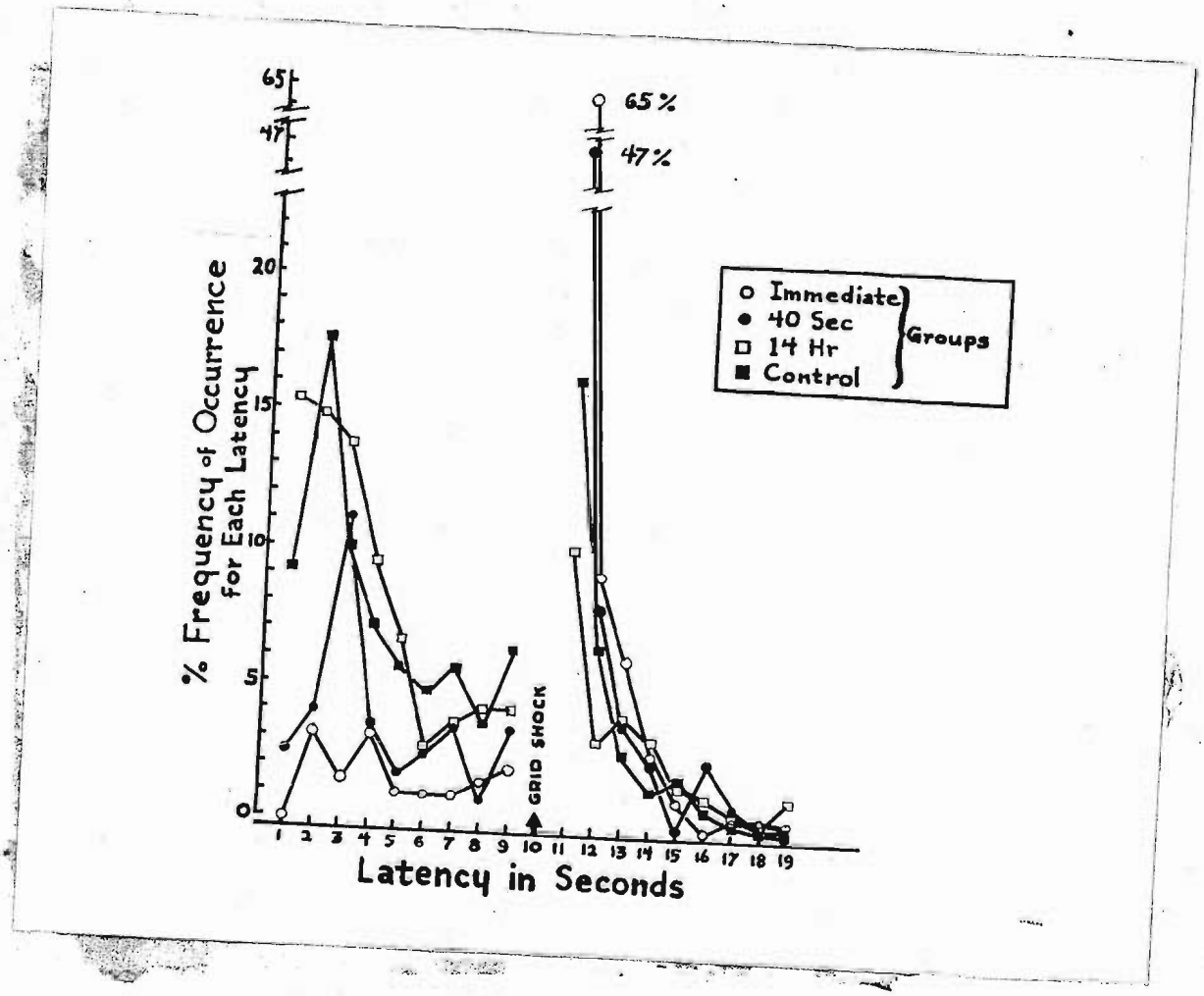

Figure 6

The percent frequency of occurrence of each latency as a function of latency in seconds. The graph shows only the data of 4 of the 9 groups of animals Latencies beyond 19 seconds are not represented.

D. Discussion

If our assumption is correct, that the learning curves in Figure 5 can be classified into 3 different types of response to electroshock, an adequate explanation is difficult to find. There are two chief obstacles to a simple explanation: First, the 1 hour, 4 hour, and 14 hour groups learn as well as the control animals, and second, the Immediate and 40 second groups show a distinctive drop in the learning curves after some initial improvement. Presumably an explanation which could account for these two extreme phenomena would also encompass the compromise behavior of the 60 second, 4 minute, and 15 minute groups. No attempt will be made to present a quantitative explanation of the empirical curves. Except for the familiar increase in habit strength with repeated reinforcement, little or nothing of an exact nature is known about the variables operating in this experiment, and we do not feel justified in attempting to derive functions describing these variables from the empirical data. However, if we assume that in addition to the expected improvement in performance with practice two other variables are operating, we can offer a tentative explanation of the empirical curves.

The first of these variables is the time allowed after each trial before the electroshock is administered. Both Figures 4 and 5 show that when the shock is applied within 15 minutes after the daily trial, learning is hindered. The next group beyond 15 minutes is the 1 hour Group, which learned as well as the controls. The results indicate that a period somewhat longer than 15 minutes, but less than an hour, must be allowed after each trial for "consolidation" or "perseveration" to run its course. Attention has already been called to a similar finding in the previously reported maze experiments. As a result of that work, 3 to 4 hours was suggested as the interval of rest following the response, after which electroshock had no effect. The much

shorter consolidation time found in the avoidance experiment is probably due to the fact that there was no conflicting habit as in the maze work, and that the reinforcement, grid shock instead of food, was different. Thus, the first variable is the trial-shock interval, the consolidation time.

The second variable which we assume to be operating is the general physiological effect of cerebral electroshock. It is known that electroshock has a debilitating effect on the organism; in addition to the numerous studies showing that shock produces memory impairment we have the evidence of Stone (49) that the general activity level of the rat is reduced after as little as one cerebral shock. Furthermore, there is reason to believe that with frequent shocks this general debilitating effect cumulates. We have already reviewed Ziskind's paper (54) in which he warned against the administration of too frequent shocks in treating mental patients. He argued that the memory impairment from shock treatment is cumulative, and that the severity of the memory loss depends to a considerable extent on the frequency with which shocks are administered. In other words, after the immediate effects of the convulsion have worn off, there remains a general depression of function which lasts many hours. If a long recovery period, perhaps of the order of several days, intervenes before the next shock, this depression will be dissipated. But with frequent shocks, as, for example, one per day in the present experiment, there remains a residual impairment which is not regained. This impairment gradually cumulates, and finally may become overt in the behavior of the animal.

These two variables, the time between the daily trial and the shock, and the debilitating effect of the shock cumulating in measurable impairment, may interact in the following way. In the first place, the grid avoidance situation is a simple one for the rat, and the only measurable "error" is a delay

of response long enough to receive the grid charge. We have previously emphasized the fact that considerable care is needed to demonstrate any effect of electroshock when the response is simple. Thus, although the 1 hour, 4 hour, and 14 hour groups are receiving electroshock daily, the shock comes too late after each trial to have any effect on learning; the consolidation period after each trial is long enough so that increments to habit strength are not affected. However, when the shock is applied within 1 to 15 minutes after each trial, the consolidation interval is slightly shortened, but not entirely eliminated. Learning is partly interfered with, and shows up as a slower rate of improvement. Finally, when the trial-shock interval is less than 1 minute, there seems to be an actual disturbance of retention. The consolidation time after each trial is so shortened that the increments to habit strength are small. The gradually increasing increments of shock-induced general impairment (including memory impairment) "catch up with" and become greater than the gradually decreasing increments to habit strength, resulting in an actual drop in the learning curves. Such a drop was not observed in animals receiving a long delayed cerebral shock because, with the longer consolidation time, they had developed a highly stable habit which was resistant to the cumulated impairment.

An alternative to the "general decrement" theory might be phrased in terms of inhibition of reinforcement. We may assume that each reinforcement sets up both excitatory and inhibitory response tendencies and that actual performance is a resultant of the two. If the excitatory tendencies are more sensitive to the effects of the shock than are inhibitory ones, there might be an excess accumulation of inhibitory effects toward the end of learning in the Immediate and 40 second groups. Thus, the learning curves of these two groups would fall. The further elaboration of this interpretation is similar to that for the general decrement theory. Although there

is some evidence in our maze experiments that excitation is more seriously affected by shock than is inhibition, the question is still controversial. Hence, we prefer to use the more firmly established theory of general impairment as an explanation for the shape of the learning curves.

Any explanation of the shape of the obtained learning curves is offered as one possibility, not as the final answer. Perhaps disagreement is most likely to arise in connection with our use of the variable called "consolidation time." We shall, therefore, consider the results from the point of view of the retroactive effect of the electroshock, and determine what evidence there may be for a theory of consolidation.

In considering the retroactive effect of one activity upon another, we may distinguish two types of study, which represent extremes. The first is the classical retroactive inhibition situation in which the interpolated task is similar to the original task. For example, most of the studies of retroactive inhibition reviewed by Britt (2) and by McGeoch (20) involve an original task, which may be verbal or motor, and an interpolated task which is similar in nature to the original task.

In the second type of study, the interpolated activity is quite different from the original activity. This type is exemplified by the clinical studies of retrograde amnesia, anoxia, brain lesions, etc. The initial task may be almost any sort of learned activity; that which disrupts the retention of the learned behavior may be a blow on the head, exposure to severe oxygen lack, or cerebral electroshock, to name but a few of this class of disorganizing agents. Ordinarily, the term "retroactive inhibition" has not been applied to situations of this type.

The mechanism that has been used to explain the impaired retention of the initial task also differs for the two types. Retroactive inhibition has

usually been explained by habit interference, by which is meant competition of responses at the time of measuring the retention of the original task, McGeoch (20). Competition of responses is probably to be expected in the classical retroactive inhibition design where the initial and the interpolated tasks are similar in content.

Habit interference, however, is not appropriate in those situations where the interpolated activity is very different from the initial activity, as, for example, where a blow on the head has caused a retrograde amnesia for the events occurring a short time before the injury. Attempts at explanation of the retroactive effect in these cases have been less systematic than in the case of retroactive inhibition. Usually the impairment is referred to some general disorganizing or disrupting effect on brain function, Cobb (5), Shock (42).

In view of the type of interpolated material used, it is understandable that the retroactive inhibition studies have not tended to confirm a perseveration theory. McGeoch's (20) conclusion that the perseveration theory does not suffice to explain the facts of retroactive inhibition is based almost entirely on studies where the interpolated activity was very similar to the original activity. The transfer, or competition of response, theory advanced by McGeoch to explain retroactive inhibition rests heavily upon the variable of similarity between initial and intervening tasks.

It is significant, however, that since the work of Melton and his collaborators (23, 24) investigators have tended to add another variable, along with habit interference, to explain retroactive inhibition, McGeoch (20), Postman and Alper (30), Postman (31), Minami and Dallenbach (25). Melton proposed that an unlearning factor, occurring during the interpolated activity, be added to competition of responses. According to this view the impairment

in retention of the original activity cannot be entirely accounted for by habit interference at the time of measuring the retention of the original task. Melton therefore proposed that the residual decrement was due to an unlearning of the initial task induced by the interpolated task. With the proposal of a two factor theory of retroactive inhibition we believe the gap has been bridged between the clinical type of study emphasizing disruption of traces and the typical experiment on retroactive inhibition emphasizing habit interference due to similarity.

It seems reasonable to assume that those studies emphasizing unlearning or disruption best illustrate the perseveration theory. The purest case would be one in which the interpolated activity is very different from the original activity. Furthermore, the interpolated response should be presented soon enough after the initial response to fall within the perseveration period. We would agree with McGeoch when he says that no one has assumed that perseveration continues for more than a few minutes. If that is true then the ideal experimental design for testing the perseveration theory would be one where the intervening response is presented within a few seconds or minutes after the original response, and where habit interference plays no role.

The experiment of Minami and Dallenbach (25) is an intermediate case. The original task consisted of learning to avoid a darkened box. The interpolated task consisted of running on a treadmill. Of their several experiments only one concerns us here. In this part of the work the interpolated activity was presented at various points in the three hour interval between learning and relearning. It was found that when the forced running on the treadmill occurred immediately after learning, 2 hours after, and 3 hours after (immediately before relearning), a significant decrement in relearning ability appeared. No decrement was found when the forced activity occurred 1 hour

after original learning.

Minami and Dallenbach conclude that their results favor the perseveration theory. Arguing on the basis of a two factor interpretation they say that the impaired retention which appeared when the interpolated activity followed original learning immediately is due to Factor X, an anti-consolidation factor. When the forced activity occurred as long as 1 hour after learning, no decrement in relearning appeared because perseveration had ceased. Finally, Factor Y, which the authors call excitement or irritability, must be postulated to explain the decrement when the interpolated activity occurred immediately or 1 hour before relearning.

Our results support the position taken by Minami and Dallenbach. These investigators believe that the detrimental effect of interpolated activity when close to the original learning may depend on the following conditions: (1) the interpolated activity must be strong enough to involve a general excitement, and (2) original learning must not be too well established. Both of these conditions are met in the present experiment.

In terms of the present experiment, then, there occurs after each trial a period of perseveration or consolidation. This period is less than 1 hour and very probably is not significantly longer than 15 minutes. If some disrupting factor, in particular the violent convulsion induced by electroshock, occurs during the interval of perseveration, retention is impaired. The loss will show up as impaired reproduction or relearning when the familiar A-B-A order is used. But when learning trials are alternated with the disrupting activity, as in the present experiment, the loss shows up as a slower rate of learning. The closer the intervening activity is in time to the preceding behavior, the greater is the decrement when the original response is retested. Finally, when the interpolated activity is introduced after perseveration has

ceased, but before a rest interval which precedes relearning, no decrement is found.

V. Summary and Conclusions

A. Maze experiments

The experiments were designed to determine the effect of a single cerebral electroshock on the acquisition and extinction of a recently learned habit which was balanced in strength with an older incompatible habit. In the five acquisition experiments rats were trained to go to the left on a T maze for a small reward, consisting of .1 gram of wet mash. In the typical experiment training on the left continued at the rate of 3 trials per day for 15 days. On the day following termination of left training the animals were trained to go to the right. The right goal box had not previously been rewarded; it was now rewarded with 2 grams of mash. Food particles on the right arm of the maze lured the animals to the right goal box. All animals chose the right habit after from 4 to 7 trials with the large 2 gram reward; all right trials were given in one session. Some of the rats were then selected to receive the electroshock, while the others served as controls. The Shock Group animals received 85 volts A.C. passed through the head for .2 second. In different experiments the shock was administered immediately after, one half hour after, or 2 hours after the last right trial. Both the shocked animals and the controls were tested for habit dominance on the maze either one half hour or 24 hours after administration of the shock.

In most cases it was found that when the animals were tested for habit preference, the shocked animals returned to the old left habit, while the control animals continued to choose the right. Thus, even though the recent habit was dominant at the time of shocking, a single shock was sufficient to allow the old habit to regain dominance.

The return to the old habit was only temporary in most of the shocked

animals; after a few trials they again chose the recent, more highly rewarded response. The shock therefore disorganizes rather than destroys the recent memory traces.

In test trials run one half hour after a shock the shocked rats were slow, appeared confused, and showed considerable VTE at the choice point. This suggests the value of psychotherapy concurrently with shock treatment in human patients.

In order to demonstrate any effects of a single electroshock it was necessary to balance the conflicting habits rather carefully. This suggests that a single shock produces no permanent effects, particularly on simple habits.

The training procedure in the three extinction experiments was the same as in the acquisition experiments for both habits. Following the last trial on the recent right habit, the reward was removed from the right goal box and extinction of the right habit begun. Extinction was continued until all animals had returned to the old left habit. The three experiments differed in the number of extinction trials allowed. Immediately after the last extinction trial some of the rats were given a single electroshock. Both shocked and control animals were tested on the maze one half hour after the shock. A second test for habit preference was given 24 hours after the first test.

In the first two experiments the effects of the extinction were not dissipated by either the shock in the experimental groups or spontaneous recovery in the control groups when the animals were tested one half hour after the shock. This result was confirmed at the second test 24 hours later.

In the third experiment fewer extinction trials were allowed. The control animals showed spontaneous recovery after one half hour of rest, indicated by their return to the right habit. However, except for one animal, the shocked rats continued to run left; extinction of the right habit was not

affected by the electroshock. Both groups of animals showed the same behavior on the second test, 24 hours later.

It is concluded that the results of the acquisition and extinction experiments somewhat contradict the theory that shock procedures diminish excitation and enhance inhibition. In the present experiments a single cerebral electroshock disrupted excitation but not inhibition.

B. Conditioned avoidance experiment

Nine groups of animals were trained to avoid a charged grid. One trial per day was given on each of 18 days. Eight of the groups received an electroshock of 85 volts A.C. passed through the head for .2 second at various times after each trial. The groups were as follows, designated by the time which elapsed between the daily trial and the administration of the electroshock: Immediate (shocked within 20 seconds after each trial), 40 second, 60 second; 4 minute, 15 minute, 1 hour, 4 hour, and 14 hour. The remaining group consisted of control animals that received no electroshock. Anticipatory runs and latencies of running were recorded.

1. When learning was measured by the mean anticipatory runs over all 18 trials, the groups were ranked in the following order from poorest to best learning: Immediate, 40 second, 60 second, 4 minute, 15 minute, and finally all other groups. The differences between the means of each of the first five groups and the Control Group were statistically significant. No significant differences were found between the means of the 1 hour, 4 hour, and 14 hour groups, and the Control Group.
2. When learning curves were plotted for the groups it was shown that the Immediate and 40 second groups exhibited an actual loss in the latter half of learning, i.e., after an initial rise the learning curves of these two groups dropped. The 60 second, 4 minute, and 15 minute groups showed a slower rate of learning than the controls over all 18 trials. The 1 hour, 4 hour, and 14 hour groups showed as rapid a rate of learning as the controls.
3. No particularly useful measure of the effect of shock on learning could be obtained from the data on latency of running.
4. A tentative explanation was offered for the three types of response to

the shock as shown by the learning curves. This explanation involved the variables of perseveration time and shock-induced general impairment.

5. The results as a whole were interpreted as evidence for a perseveration theory. Learning was impaired because the shock disrupted the normal process of perseveration; the shorter the trial-shock interval, the greater was the impairment.

VI. Bibliography

1. Birois, R. Les troubles de la mémoire après l'électro-choc. Ann. méd.-psychol., 1942, 100, Part 2, 338-342.

2. Britt, S. H. Retroactive inhibition: A review of the literature. Psychol. Bull., 1935, 32, 381-440.

3. Brody, M. B. Prolonged memory defects following electro-therapy. J. Ment. Sci., 1944, 90, 777-779.

4. Bunch, M. E., and Mueller, C. G. The influence of metrazol upon maze learning ability. J. comp. Psychol., 1941, 32, 569-574.

5. Cobb, S. Personality as affected by lesions of the brain. In Hunt, J. McV. Personality and the Behavior Disorders. New York, The Ronald Press, 1944. Vol. I., pp. 550-581.

6. Duncan, C. P. The effect of electroshock convulsions on the maze habit in the white rat. J. exp. Psychol., 1945, 35, 267-278.

7. Gellhorn, E., and Minatoya, H. The effect of insulin hypoglycemia on conditioned reflexes. J. Neurophysiol., 1943, 6, 161-172.

8. Gellhorn, E. Further investigations on the recovery of inhibited conditioned reactions. Proc. Soc. exp. Biol., N. Y., 1945, 59, 155-161.

9. Gellhorn, E. Is restoration of inhibited conditioned reactions by insulin coma specific for Pavlovian inhibitions? Contribution to the theory of shock treatment. Arch. Neurol. Psychiat., 1946, 56, 216-221.

10. Gellhorn, E. Autonomic Regulations. New York, Interscience Publishers, Inc., 1943.

11. Grinker, R. R., and Spiegel, J. P. Men Under Stress., Philadelphia, Blakiston, 1945.

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

13. Hunter, W. S., and Hall, B. E. Double alternation behavior of the white rat in a spatial maze. J. comp. Psychol., 1941, 32, 253-266.

14. Kalinowsky, L., and Hoch, P. Shock Treatments and other Somatic Procedures in Psychiatry., New York, Grune & Stratton, 1946.

15. Kessler, M., and Gellhorn, E. The effect of electrically and chemically induced convulsions on conditioned reflexes. Amer. J. Psychiat., 1943, 99, 687-691.

16. Lashley, K. S. Brain Mechanisms and Intelligence., Chicago, Univ. Chicago Press, 1929.

17. Levy, N. A., Serota, H. M., and Grinker, R. R. Disturbances in brain function following convulsive shock therapy. Arch. Neurol. Psychiat., 1942, 47, 1009-1029.

18. Loken, R. D. Metrazol and maze behavior. J. comp. Psychol., 1941, 32, 11-16.

19. Luborsky, L. B. A psychometric study of electroshock treatment. Master's thesis, Duke Univ., 1943.

20. McGeoch, J. A. The Psychology of Human Learning., New York, Longmans, Green and Co., 1942.

21. McGinnies, E., and Schlosberg, H. The effects of electroshock convulsions on double alternation lever-pressing in the white rat. J. exp. Psychol., 1945, 35, 361-373.

22. McNeel, B. H., Dewan, J. G., Myers, C. R., Proctor, L. D., and Goodwin, J. E. Parallel psychological, psychiatric and physiological findings in schizophrenic patients under insulin shock treatment. Amer. J. Psychiat., 1941, 98, 422-429.

23. Melton, A. W, and Irwin, J. McQ. The influence of degree of interpolated learning on retroactive inhibition and the overt transfer of specific responses. Amer. J. Psychol., 1940, 53, 173-203.

24. Melton, A. W., and Von Lackum, W. J. Retroactive and proactive inhibition in retention: evidence for a two-factor theory of retroactive inhibition. Amer. J. Psychol., 1941, 54, 157-173.

25. Minami, H., and Dallenbach, K. M. The effect of activity upon learning and retention in the cockroach. Amer. J. Psychol., 1946, 59, 1-58.

26. Myerson, A. Borderline cases treated by electric shock. Amer. J. Psychiat., 1943, 100, 355-357.

27. Page, J. D. Studies in electrically induced convulsions in animals. J. comp. Psychol., 1941, 31, 181-194.

28. Perlson, J. Psychologic studies on a patient who received two hundred and forty-eight shock treatments. Arch. Neurol. Psychiat., 1945, 54, 409-411.

29. Plattner, P. Amnestisches Syndrom nach Insulincardiazolbehandlung. Z. ges. Neurol. Psychiat., 1938, 162, 728-740.

30. Postman, L., and Alper, T. G. Retroactive inhibition as a function of the time of interpolation of the inhibitor between learning and recall. Amer. J. Psychol., 1946, 59, 439-449.

31. Postman, L. Reaction time as a measure of retroactive inhibition. Amer. Psychologist, 1946, 1, 258-259. Abstract.

32. Purcell, P. Psychometric experiments following electrical convulsive therapy. Master's thesis, Columbia Univ., 1945.

33. Rennie, T. A. C. Present status of shock therapy. Psychiatry, 1943, 6, 127-137.

34. Riess, B. F., and Berman, L. The mechanism of the insulin effect on abnormal behavior. Amer. J. Psychiat., 1944, 100, 674-680.

35. Rodnick, E. H. The effect of metrazol shock upon habit systems. J. abnorm. soc. Psychol., 1942, 37, 560-565.

36. Rose, J. A., Tainton-Pottberg, A., and Anderson, D. D. Effects of insulin shock on behavior and conditioned reflex action in the well-trained sheep. Proc. Soc. exp. Biol., N. Y., 1938, 38, 653-655.

37. Rosen, V. H., and Gantt, W. H. Effect of metrazol convulsions on conditioned reflexes in dogs. Arch. Neurol. Psychiat., 1943, 50, 8-17.

38. Schlosberg, H., and Katz, A. Double alternation lever-pressing in the white rat. Amer. J. Psychol., 1943, 56, 274-282.

39. Schnack, G. F., Shakow, D., and Lively, M. L. Studies in insulin and metrazol therapy. J. Personality, 1945, 14, 106-149.

40. Sharp, H. C., Winder, C. L. and Stone, C. P. Effects of electro-convulsive shocks on "reasoning" ability in albino rats. J. Psychol., 1946, 22, 193-197.

41. Sherman, I., Mergener, J., and Levitin, D. The effect of convulsive treatment on memory. Amer. J. Psychiat., 1941, 98, 401-403.

42. Shock, N. W. Physiological factors in behavior. In Hunt, J. McV. Personality and the Behavior Disorders. New York, The Ronald Press, 1944. Vol. I, pp. 582-618.

43. Siegel, P. S. The effect of electroshock convulsions on the acquisition of a simple running response in the rat. J. comp. Psychol., 1943, 36, 61-65.

44. Smith, L. H., Hughes, J., Hastings, D. W., and Alpers, J. Electroshock treatment in the psychoses. Amer. J. Psychiat., 1942, 98, 558-561.

45. Smith, L. H., Hastings, D. W. and Hughes, J. Immediate and follow-up results of electroshock therapy. Amer. J. Psychiat., 1943, 100, 351-354.

46. Stainbrook, E. J., and Löwenbach, H. The reorientation and maze behavior of the rat after noise-fright and electroshock convulsions. J. comp. Psychol., 1942, 35, 293-299.

47. Stainbrook, E. J. Maze behavior of the rat after electroshock convulsions. J. exp. Psychol., 1943, 33, 247-252.

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

49. Stone, C. P. Effects of electro-convulsive shocks on daily activity of albino rats in revolving drums. Proc. Soc. exp. Biol., N. Y., 1946, 61, 150-151.

50. Stone, C. P. The course of change in intellectual functions associated with electro-convulsive shock. Amer. Psychologist, 1946, 1, 449-450. Abstract.

51. Stone, C. P. Characteristic losses and gains in scores on the Wechsler Memory Scales as applied on psychotic patients before, during, and after a series of electro-convulsive shocks. Amer. Psychologist, 1946, 1, 245. Abstract.

52. Wechsler, D., Halpern, F., and Jaros, E. Psychometric study of insulin-treated schizophrenia. Psychiat. Quart., 1940, 14, 466-476.

53. Wortis, J. Physiological treatment of the psychoses. In Review of Psychiatric Progress 1942. Amer. J. Psychiat., 1943, 99 602-604.

54. Ziskind, E. Memory defects during metrazol therapy. Arch. Neurol. Psychiat., 1941, 45, 223-234.

55. Ziskind, E., Loken, R., and Gengerelli, J. A. Effect of metrazol on recent learning. Proc. Soc. exp. Biol., N. Y., 1940, 43, 64-65.

56. Zubin, J., and Barrera, S. E. Effect of electric convulsive therapy on memory. Proc. Soc. exp. Biol., N. Y., 1941, 48, 596-597.

57. Zubin, J. The effect of electroshock therapy on "interference" in memory. Psychol. Bull., 1942, 39, 511.

58. Zubin, J. Psychological changes in patients receiving electric shock therapy. Amer. Psychologist, 1946, 1, 461. Abstract.

VII. Appendix

The following tables show the latency of running (in seconds) for each animal for every trial in the conditioned avoidance experiment. Latency was measured from the moment the animal was placed on the grid until the animal's body exclusive of tail arrived in the safe compartment of the apparatus. A separate table is presented for each group of animals; the title of the table indicates the time which elapsed between each trial and the application of the cerebral electroshock in that group. All runs less than 10 seconds permitted the animal to avoid the grid charge and were counted as anticipatory runs.

Table A

Immediate Group

Trial		1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18
Rat	1	23	17	21	11	11	11	11	11	9	11	11	11	11	11	11	11	11	11
	2	15	11	11	14	11	11	11	11	11	11	11	11	11	11	11	11	8	11
	3	18	12	11	11	12	11	13	11	12	11	11	11	11	11	11	11	11	11
	4	31	11	11	11	12	11	11	11	11	11	11	11	11	13	11	11	11	11
	5	12	11	14	12	5	11	7	12	11	6	7	9	11	9	11	14	11	8
	6	12	13	11	11	12	11	11	11	11	11	11	13	11	11	11	11	11	11
	7	25	9	11	35	11	12	13	12	11	11	11	11	13	11	11	13	12	11
	8	13	14	14	13	died													
	9	11	12	12	12	11	4	2	3	2	11	11	11	12	11	2	11	11	11
	10	11	14	13	13	11	11	11	11	2	11	11	11	11	11	11	11	11	13
	11	11	12	11	11	4	5	15	12	11	4	4	11	11	11	6	11	11	11
	12	11	11	12	3	4	3	11	2	4	2	11	11	11	11	8	11	11	11

Table B

40 Second Group

Trial	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18
Rat 1	12	19	died															
2	13	15	12	23	11	11	12	12	15	11	11	11	11	11	12	died		
3	17	27	27	13	12	12	14	12	died									
4	12	13	13	11	died													
5	14	21	12	died														
6	13	11	12	14	12	11	12	3	2	6	9	11	3	11	11	11	11	7
7	13	11	23	22	27	12	3	3	2	11	3	3	3	11	2	2	11	11
8	12	40	12	12	11	9	2	11	11	11	2	3	3	3	3	5	11	11
9	11	9	22	40	17	11	9	8	11	11	11	11	7	11	11	4	11	11
10	17	14	11	11	16	11	11	11	11	11	12	11	11	14	13	11	11	11
11	16	20	12	5	4	2	11	11	11	7	6	7	3	11	4	4	11	11
12	16	21	12	13	3	11	1	1	11	1	11	6	11	11	11	11	11	11

Table C

60 Second Group

Trial	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18
Rat 1	12	22	11	11	11	11	19	4	11	2	5	11	6	12	2	3	11	11
2	11	12	15	14	13	2	3	2	2	11	11	11	11	12	2	3	4	3
3	12	20	15	13	9	3	5	3	6	11	20	11	2	4	5	6	3	4
4	12	15	13	11	25	3	12	1	11	2	4	3	4	15	3	11	1	1
5	11	11	13	12	12	11	13	12	12	11	14	3	11	3	4	17	11	3
6	12	13	16	3	3	2	4	2	11	12	3	2	2	3	2	2	11	11
7	12	13	12	12	9	4	3	13	4	4	2	11	11	17	5	11	11	5
8	15	11	14	22	11	5	15	3	3	3	11	died						
9	31	8	13	died														
10	12	11	25	11	died													
11	12	9	11	15	11	17	40	11	14	11	3	3	8	3	2	4	4	6
12	12	38	15	9	6	9	11	11	11	11	11	11	11	5	12	2	11	7

Table D

4 Minute Group

Trial		1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18
Rat	1	12	11	11	11	11	11	11	11	11	11	11	11	13	11	11	11	11	11
	2	12	26	13	9	9	4	3	5	3	died								
	3	12	19	12	5	died													
	4	15	26	9	9	6	11	9	11	4	3	3	2	3	3	3	2	2	3
	5	17	14	12	11	died													
	6	16	16	13	22	16	11	13	11	9	9	11	11	11	11	11	11	9	11
	7	12	12	12	11	11	11	11	11	13	11	7	6	11	12	9	4	9	8
	8	12	12	12	13	11	11	11	3	11	2	2	3	1	3	2	3	4	3
	9	14	11	12	8	11	11	12	11	5	3	2	3	3	5	3	3	2	4
	10	13	19	11	12	3	11	11	9	2	8	2	2	5	4	5	3	7	3
	11	12	11	11	12	11	3	8	2	2	4	9	11	9	2	3	2	3	2
	12	12	12	12	9	6	3	3	2	3	3	2	3	3	7	2	9	5	11

Table E

15 Minute Group

Trial		1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18
Rat	1	12	11	15	11	16	3	11	2	2	2	9	7	11	1	2	8	3	4
	2	12	12	13	12	3	17	5	5	4	1	11	11	3	3	9	3	2	1
	3	13	15	11	12	12	11	11	16	11	3	3	1	1	4	3	3	3	2
	4	12	13	12	12	8	11	4	3	2	4	3	11	11	6	3	8	14	11
	5	11	11	12	9	11	11	11	11	11	13	4	11	11	7	11	7	7	1
	6	12	11	8	11	11	9	11	11	3	4	3	3	3	7	15	14	6	7
	7	12	15	20	11	11	11	11	11	11	9	11	4	2	2	3	3	1	1
	8	12	13	11	died														
	9	18	13	11	9	5	5	7	3	died									
	10	12	34	12	11	3	2	2	3	1	7	3	1	1	2	5	3	3	2
	11	12	13	14	11	9	6	6	3	8	11	11	11	11	4	5	4	3	6
	12	12	23	16	9	6	3	6	7	2	2	3	3	2	2	18	4	6	2

Table F

1 Hour Group

Trial		1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18
Rat	1	12	18	16	11	11	5	11	11	5	3	3	8	1	7	5	9	3	3
	2	12	13	11	11	4	3	2	2	1	1	1	1	1	1	1	2	11	1
	3	15	12	19	11	1	8	2	1	1	1	8	5	1	7	1	1	2	1
	4	18	18	11	4	11	11	8	2	2	1	1	11	1	5	4	3	3	1
	5	19	27	11	12	4	11	11	1	5	1	4	11	5	11	7	3	1	1
	6	12	14	12	3	4	7	6	4	5	7	5	8	1	8	8	6	11	7

Table G

4 Hour Group

Trial		1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18
Rat	1	12	12	12	11	11	11	11	11	3	4	4	11	4	4	11	11	11	6
	2	13	24	11	2	9	2	1	1	2	2	4	1	1	2	2	2	1	4
	3	13	8	11	11	9	4	2	5	2	2	7	3	2	2	5	1	11	4
	4	11	14	11	12	2	3	1	1	1	5	1	3	1	6	2	2	1	1
	5	13	14	14	11	12	11	3	6	11	1	1	1	2	1	2	1	1	1
	6	12	13	6	11	2	2	1	2	2	1	15	1	6	5	5	3	1	1

Table H

14 Hour Group

Trial		1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18
Rat	1	17	11	18	11	11	11	2	11	9	5	4	4	1	4	8	3	3	2
	2	14	13	13	13	11	9	8	5	1	1	2	3	3	7	5	5	9	9
	3	15	11	13	3	7	3	1	2	2	3	2	4	2	2	1	8	1	1
	4	12	16	5	4	4	7	2	4	4	3	1	1	2	3	2	5	5	1
	5	14	13	18	11	8	3	6	5	3	4	13	5	2	2	2	2	5	1
	6	14	12	18	11	11	12	died											
	7	17	30	32	11	11	2	11	18	2	2	1	11	died					
	8	12	13	12	13	11	11	9	11	5	4	7	11	11	11	2	4	4	3
	9	22	15	4	7	4	8	4	2	3	2	2	3	1	4	2	6	2	2
	10	11	18	11	5	2	15	14	2	1	2	1	1	1	1	died			
	11	19	27	20	13	13	11	4	4	3	3	1	1	1	1	1	1	1	1
	12	22	16	11	5	3	9	6	1	1	2	1	1	1	1	2	1	1	1
	13	14	14	11	40	20	14	9	9	8	11	9	4	2	3	1	1	5	8
	14	15	14	12	12	25	11	11	9	3	11	11	7	1	8	8	3	1	3
	15	17	19	15	12	8	3	3	3	3	2	2	7	4	2	3	11	2	5
	16	12	14	12	7	11	6	2	2	2	3	6	1	4	3	1	1	2	1
	17	16	14	14	11	12	11	20	27	11	6	3	3	2	2	2	1	1	2
	18	12	26	19	9	5	6	8	4	4	4	5	3	7	2	3	3	3	7

Table I

Control Group

Trial		1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18
Rat	1	31	12	11	5	3	2	2	3	11	13	2	2	1	2	4	11	2	2
	2	36	13	11	6	3	3	2	2	3	3	2	9	4	7	7	4	11	11
	3	13	12	12	9	5	2	2	2	3	4	2	1	2	2	3	9	2	5
	4	32	12	11	5	2	11	12	12	12	11	11	9	4	2	2	5	4	2
	5	12	11	15	11	4	6	7	2	2	2	1	1	4	1	1	1	1	3
	6	11	12	12	13	4	6	5	4	11	3	2	3	3	3	11	6	2	11
	7	12	12	15	11	7	7	11	2	2	5	11	11	3	1	1	1	1	3
	8	13	20	20	15	6	4	3	4	4	4	5	3	6	2	7	7	7	11
	9	12	37	18	11	8	11	6	11	6	9	7	9	8	2	2	2	1	2
	10	14	15	16	9	7	2	11	9	8	9	11	11	11	8	9	8	5	2
	11	12	16	16	11	11	13	3	11	2	1	1	3	1	2	1	12	1	1
	12	12	20	11	9	4	11	2	8	6	8	8	9	2	2	2	1	1	1
	13	12	17	11	14	12	12	12	5	1	2	7	9	7	3	2	7	8	5
	14	11	15	11	11	11	11	9	11	11	9	6	7	11	8	4	7	6	5
	15	11	20	11	11	9	3	3	5	3	6	5	11	9	1	3	3	2	1
	16	11	15	18	11	9	4	9	4	2	9	8	14	4	7	6	3	3	3
	17	13	40	14	20	40	9	6	3	3	6	2	2	2	2	3	7	5	4
	18	13	20	12	11	11	11	2	2	2	2	5	1	5	1	4	2	1	4