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THE EFFECTS OF ELECTROCONVULSIVE TREATMENTS ON MEMORY EFFICIENCY

BY IRVING L. JANIS AND MYRTLE ASTRACHAN
Department of Psychology, Yale University

PARALLELING the widespread clinical use of shock therapy, there has been a considerable amount of research on the psychological effects of electroconvulsive treatments (ECT). Nevertheless, only the gross effects of ECT have been widely observed and verified. As yet very few controlled investigations have been carried out to determine the more subtle psychological changes which might be produced.

Many investigations have concentrated upon the temporary "organic" reaction which develops during the course of treatment, especially the decline in intellectual abilities and the extensive memory impairment. It has been generally observed that these deficits tend to disappear within approximately two weeks after the last convulsive treatment (6, 7). Scores on standard psychometric tests of intelligence return to, or even exceed the pretreatment level. The diffuse amnesias characteristic of the treatment period tend to clear up to such an extent that most clinical observers have claimed that within two or three weeks following termination of the treatment, memory functions show complete recovery.

Although it is well established that the gross deficits in mental efficiency are temporary and reversible, the possibility remains that after the usual recovery period there are some residual defects. One of the present authors has recently reported the results of a controlled experiment which provide definite and consistent evidence that there are circumscribed amnesias persisting long after the period when temporary "organic" reactions clear up (2, 3). From the observed characteristics of the posttreatment amnesias, it appears that they tend to blot out memories which are likely to evoke guilt, lowered self-esteem or other painful affective reactions. Consequently, it is likely that motivational factors account for the *selectivity* of the forgotten material. But this hypothesis by no means precludes the possibility that there are underlying changes in basic memory processes. There are, in fact, some indications that the circumscribed amnesias arise as a result of a general deficit in memory functioning, probably involving actual organic impairment of a rather subtle kind. For example, a separate experiment on changes in word-association reactions, carried out by the same author (4), revealed the presence of a residual disturbance: four weeks after the termination of electroshock treatments, the patients displayed an increase in certain types of association disturbances and in defective reproductions of the word-association responses. On the assumption that spontaneous word associations normally tend to be recalled personal responses based on prior learning, these findings suggest that there may be an underlying disturbance in basic recall processes—a disturbance which might markedly reduce the patient's efficiency on any task requiring the production of verbal or symbolic associations. Qualitative observations of the difficulties exhibited by electroshock-treated patients in their efforts to produce personal memories (3) also imply that there is a residual memory impairment which is not restricted to the posttreatment amnesias but extends to other personal memories as well.

The purpose of the present experiment is to test systematically certain of the implications of the earlier observations. The specific hypothesis with which we are primarily concerned is the following: After the gross organic effects of electroshock treatments have cleared up, the patients are left with a residual memory impairment which is manifested by a reduction in mental efficiency on tasks requiring the production of previously acquired symbolic associations. The present research report deals with the memory efficiency of electroshock-treated patients when they are required to give routine personal information. A later report will present the results of a concomitant experiment on changes in performance on intellectual tasks

requiring the production of *impersonal* symbolic associations.

If there is increased difficulty in producing personal memories following electroshock treatments, we should expect to find a quantitative increase both in *errors* of recall and in the *latency* of memory responses. Moreover, if there is a generalized memory defect after electroshock therapy, some form of impairment should be found which affects routine personal memories as well as dynamically important memories. Accordingly, the present experiment has been designed to test the prediction that electroshock-treated patients will exhibit more recall errors and will be generally slower in responding to routine questions about their past.

PROCEDURE

In order to determine the availability of routine personal memories, a personal information questionnaire was used. The beginning of the questionnaire contained six items from Test 1 of Babcock's test of mental efficiency (1): name, birthplace, year of birth, family composition, etc. To these initial items we added 34 questions requesting simple, descriptive information about the patient's life history: schools attended, names of grade school teachers, jobs held, names of employers, out-of-town trips, personal activities on certain outstanding historical dates during World War II, etc.

The personal information questionnaire was administered to each patient individually, in a face-to-face interview. Verbatim responses, reaction time in responding to each question, and the total time required to answer each question were systematically recorded. Each patient was interviewed twice, in identical fashion, with the same standardized set of questions. For the ECT patients, the first interview took place a few days before the treatments began. The retest was administered at least four weeks after the last convulsive treatment.

In order to identify the series of electroshock treatments as the critical variable responsible for any significant changes in the test performance of the ECT patients, an equated control group was used. The control patients were given the same questionnaire under similar interview conditions, with approximately the same time interval between the two administrations of the test. The mean time interval between the test and retest was 13 weeks for both the control group and the ECT group. During the interval, the control patients did not receive any form of shock treatment but they were in the same hospital wards and were exposed to the same general environmental conditions as the ECT patients.

Subjects

The two groups were drawn in an unbiased way from among the "cooperative" patients in two psychiatric hospitals: The Psychiatric In-Patient Clinic of Yale University and the Middletown State Hospital.[1] There were nine patients in the experimental (ECT) group and eight patients in the control group.

The two groups were equated as closely as possible on the relevant background characteristics. The mean age for the two groups was 38.2 and 38.9 years, respectively. Both groups completed an average of 11 years of schooling; within both groups there was a comparable spread in educational status, ranging from only a few years of grammar school to a college degree. The two groups were also fairly well equated with respect to sex, occupation, duration of current hospitalization, and date of onset of the mental disorder. With respect to formal psychiatric diagnosis, the ECT group contained one neurotic depression, one borderline schizophrenia, one undifferentiated schizophrenia, four paranoid schizophrenias, one paranoid condition, and one involutional depression; the control group contained three neurotic depressions, one early schizophrenia, two paranoid schizophrenias, and two involutional depressions. Although there was some difference in the diagnostic labels assigned to the patients in the two groups, a detailed examination of the case records showed that there were only slight differences between the two groups with respect to severity of illness and type of mental symptoms.

The ECT patients received from 10 to 30 electroshock treatments spaced three times a week, as administered in standard hospital practice. Six of the nine ECT patients were given close to 20 treatments; the mean for the entire group was 18.7 convulsive treatments. According to the clinical ratings of the psychiatric staff, all the electroshock-treated patients were "improved" or "asymptomatic" at the time of the posttreatment test.

RESULTS

Posttreatment Recall Failures

The present experiment focuses primarily upon relatively subtle changes in memory efficiency—minor errors in recall (such as omission of circumstantial detail) and slower speed in producing personal information. Nevertheless, certain of the results provide direct evidence of total recall failures and are indicative of a rather gross type of memory defect. These gross failures will be described first inasmuch as they provide a general context for discerning the implications of the more subtle forms of decline in memory efficiency to be described later on.

[1] The authors wish to express their appreciation to the staffs of these two hospitals, particularly to Drs. Frederick C. Redlich and Stanley Leavy, at the Yale Clinic, and to Drs. Benjamin Simon and Jules Holzberg, at Middletown State Hospital, for their helpful cooperation on this research project.

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Recall Failures

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Gross amnesias. One of the most prominent and distinctive features of the performance of the ECT patients on the posttreatment test was the occurrence of pronounced recall failures. In most extreme form, these failures reveal the presence of amnesic gaps of the sort described earlier (3).

In the earlier study it was observed that although the retroactive amnesias tended to blot out emotionally charged experiences which had been deeply disturbing to the patient, the residual memory gaps occasionally affected relatively neutral material. The results we are about to present on gross memory failures confirm the occurrence of posttreatment amnesias and establish the fact that the residual memory loss extends to the simple type of life history information covered by our present recall test.

As in the former study, the patients in the present experiment had been given ample time to recover from the gross deficit in intellectual functioning which occurs during the treatment period and which generally clears up within two or three weeks after the last treatment. At the time of the posttreatment test there was no evidence that any of the patients had failed to show the usual recovery from the temporary organic syndrome. In general, their scores reached or exceeded the posttreatment level when we tested them on a series of standard intelligence test items. But in their attempts to answer routine questions about their past, these patients displayed a significant deficit.

Initially there was no difference between the control group and the ECT group in ability to answer the questions. The 40 items in the test elicited a wide range of detailed information; the initial (pretreatment) performance served to indicate the specific memories which each patient could readily produce. The mean number of questions answered by the electroshock patients on the pretreatment test was 33.22; the corresponding mean for the control patients was 32.85. The very slight difference between these initial means was not significant ($t=0.14$, $p=.45$). After treatment, however, the electroshock group displayed a significant decline in the number of questions they were able to answer. Four weeks or more after the last electroshock convulsion, the patients were totally unable to answer some of the questions that had been readily answered before treatment. On the average, the ECT patients failed on 3.22 questions which they had successfully answered on the initial test. The corresponding mean number of failures for the control group on retest was 1.12. The two groups differ significantly with respect to the number of questions failed ($t=2.30$, $p=.02$).[2]

We regard the results for the control group as a base line for estimating the extent of recall failure to be expected upon retest when mental patients are not given any form of shock therapy. The small number of failures exhibited by the control patients may be attributed to (*a*) the slight amount of forgetting which might spontaneously occur among mental patients during a period of several months of hospitalization and (*b*) momentary factors which introduce some degree of variability into memory-functioning when tested at different times. The fact that the electroshock-treated patients displayed a significantly larger number of gross recall failures implies that their memory functioning had become impaired, to some degree, as a result of the electroshock treatments they had received.

Incomplete answers and subtle amnesias. Further evidence of a residual memory impairment following electroshock treatments comes from a detailed examination of the amount of information given by ECT patients in response to those questions which they were able to answer. Included in the test were the following ten questions which required more than one piece of information for a complete answer.

12. Now I would like you to tell me the names of all of the other schools you have gone to since that one (the first school). Just give me the names of all the schools you have gone to.

14. What were the names of some of the teachers you had during the first years of grade school—when you were in first, second, or third grade?

15. What were the names of some of the teachers you had when you were in sixth, seventh, or eighth grade?

16. What were the names of some of your high school teachers?

24. Now I would like you to tell me all of the other places where you worked. Just give me the name and address of each one.

[2] All *p*-values reported in this paper were based on one tail of the theoretical distribution of *t*, since specific hypotheses were being tested.

13. Tell me everything you can remember about what happened on the day you graduated from grade school—what the graduation ceremony was like and what other things you did that day.

30. What things did you do that day (when you learned of the news of the atom bomb)?

33. What did you do that day (when you learned the news that Pearl Harbor had been bombed by the Japanese)?

37. What things did you do and see on that trip (your first trip out of town)?

39. What things did you do and see on that trip (the last time you left your home town)?

While testing the electroshock-treated patients, we observed that even when they were able to give an answer to these questions, their answers tended to be incomplete. Frequently they left out important details that had been described before treatment. Although prompted by specific probing questions (containing memory cues to elicit the missing information), they were nevertheless unable to remember some portions of their pretreatment account.

Quantitative data in support of this observation are presented in Table 1. The scores represent the mean number of details produced per (answered) question.[3]

With the exception of one case, all the control patients displayed a slight increase in the number of details produced on retest. This increase may be due to the facilitating (practice) effect of prior rehearsal; having had the experience of producing the same information several months earlier on the initial test, they were able to produce almost all the details again and to add a few more. The electroshock-treated patients, however, were not able to produce as many details as they had given before treatment. The decline shown by the ECT patients differs significantly from the change displayed by the control group. Hence, we find that as a result of electroshock treatments, there is a decline in ability to produce complete answers in response to those routine questions which require a series of details about the individual's life history.

More precise information about the nature of the memory defect is provided by Table 2.

TABLE 1

MEAN NUMBER OF DETAILS PRODUCED PER ANSWER FOR TEN LIFE-HISTORY QUESTIONS

Control Patients (N=8)			Electroshock-Treated Patients (N=9)		
Before	After	Change	Before	After	Change
2.83	3.66	+0.83	5.25	4.00	−1.25
6.66	7.33	+0.67	3.66	1.33	−2.33
4.00	4.38	+0.38	7.75	5.75	−2.00
2.66	3.33	+0.67	4.38	3.50	−0.88
6.83	4.83	−2.00	8.00	3.56	−4.44
3.10	4.30	+1.20	3.71	4.29	+0.58
2.25	2.75	+0.50	2.63	2.75	+0.12
4.50	5.50	+1.00	5.11	4.22	−0.89
			3.75	3.37	−0.38
Mean 4.10	4.51	+0.41	4.92	3.64	−1.28

Difference between mean changes: t=3.0, p=<.01.

Here the results are limited to the last five of the ten questions listed above. The first five questions differ from the latter in that they require a series of facts about separate events in the life history—such as the patient's job history over a period of many years. Each of the last five questions, however, deals with a fairly discrete episode which occurred at one particular time in the patient's life history. The loss of details in responding to both types of questions is revealed by Table 1. A failure of the first type—for example, inability to recall a particular job—generally reflects the occurrence of a gross amnesic gap. A failure of the second type, on the other hand, indicates a much more subtle form of amnesia: the event itself is remembered but some of the specific details that had been readily given before treatment can no longer be recalled. Minor amnesias of this sort are much more circumscribed than the more obvious retroactive amnesias in which an

[3] Technical details concerning the standardized procedures used in scoring the patients' records are presented in a more extensive research report, transcript copies of which are available upon request from the Institute of Human Relations, Yale University.

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> produce as many details as they
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those routine questions which
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:tory.

:ise information about the nature
ıry defect is provided by Table 2.

N LIFE-HISTORY QUESTIONS

ECTROSHOCK-TREATED PATIENTS (N=9)	
AFTER	CHANGE
4.00	−1.25
1.33	−2.33
5.75	−2.00
3.50	−0.88
3.56	−4.44
4.29	+0.58
2.75	+0.12
4.22	−0.89
3.37	−0.38
3.64	−1.28

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entire past experience is persistently unavailable to consciousness. That the latter, more obvious kind of amnesia is a residual effect of electroshock treatments has been documented in the earlier research report; but only incidental, qualitative observations were mentioned with respect to "partial" amnesias (3). Table 2 provides definite evidence that the more subtle form of amnesia is, in fact, one of the residual sequelae of electroshock treatments. The scores are based only on those questions to which a relevant answer was given. The significant decline shown by the ECT group provides a further indication of the residual memory defect due to electroshock treatments. More specifically, these results reveal that even when an electroshock-treated patient is able to remember a particular past experience, he is likely to fail to recall certain of the circumstantial details which he had been able to produce before treatment.

TABLE 2

MEAN NUMBER OF DETAILS PRODUCED PER RECALLED EVENT FOR FIVE LIFE-HISTORY QUESTIONS

CONTROL PATIENTS (N=8)			ELECTROSHOCK-TREATED PATIENTS (N=9)		
BEFORE	AFTER	CHANGE	BEFORE	AFTER	CHANGE
3.60	4.40	+0.80	9.75	2.25	−7.50
3.00	4.00	+1.00	4.00	3.40	−0.60
7.67	9.34	+1.67	7.50	6.00	−1.50
4.00	3.80	−0.20	6.67	3.34	−3.33
2.50	3.00	+0.50	2.80	2.00	−0.80
7.50	5.25	−2.25	9.50	5.00	−4.50
5.50	7.00	+1.50	3.67	1.34	−2.33
1.00	1.00	0.00	7.20	5.00	−2.20
			3.75	5.00	+1.25
Mean 4.34	4.72	+0.38	6.09	3.70	−2.39

Difference between mean changes: $t=2.80$, $p=<.01$.

Latency of Memory Responses

From the results presented so far, we have seen that the electroshock-treated patients were unable to produce a substantial portion of the routine information about their own life histories which they had been able to recall before the treatments were begun. We turn now to another aspect of their memory efficiency, namely, the *speed* of their performance in producing the personal information which they actually were able to recall. It will be seen that the residual memory defect following electroshock treatments shows up in the form of increased latency of response. First we shall examine the evidence on reaction time, and then we shall describe other indicators of latency which imply a general decline of memory efficiency.

Reaction time. In the present experiment, "reaction time" refers to the length of time which elapses between the final word of the examiner's question and the first word of the patient's answer. An over-all reaction time score was computed for each patient which represents his mean reaction time for all questions to which an answer was given. Table 3 presents the results on the changes in reaction time scores produced by electroshock treatments.

Before treatment, the mean score of the ECT group did not differ significantly from that of the control group (7.28 seconds vs. 7.14 seconds). On retest, the controls showed a mean *decrease* of 1.77 seconds, probably due to the prior practice on the initial test. The electroshock-treated patients, however, showed a mean *increase* of 3.99 seconds. The change displayed by the latter group, as compared with the former, is highly significant, i.e., below the 1 per cent confidence limit.

These results show that the treatments have the effect of slowing up verbalized recall. This may be regarded as another feature of the impairment in memory efficiency which occurs in addition to recall failures described in the preceding section. Since the reaction time scores are based only on those questions to which an answer was given, the results in Table 3 reveal a separate kind of disturbance, viz., an initial inhibition—or delay in getting started—when pro-

TABLE 3

MEAN REACTION TIME SCORES FOR ALL LIFE-HISTORY QUESTIONS (IN SECONDS)

CONTROL PATIENTS (N=8)			ELECTROSHOCK-TREATED PATIENTS (N=9)		
BEFORE	AFTER	CHANGE	BEFORE	AFTER	CHANGE
6.41	9.41	+3.00	12.75	23.71	+10.96
4.97	4.27	−0.70	8.24	7.50	− 0.74
7.73	4.85	−2.88	3.26	12.95	+ 9.69
7.35	6.23	−1.12	9.07	12.56	+ 3.49
6.03	3.90	−2.13	6.22	14.14	+ 7.92
5.80	4.48	−1.32	8.20	9.90	+ 1.70
12.69	5.46	−7.23	7.11	8.95	+ 1.84
6.14	4.35	−1.79	4.52	6.32	+ 1.80
			6.12	5.42	− 0.70
Mean 7.14	5.37	−1.77	7.28	11.27	+ 3.99

Difference between mean changes: t=3.20, p=<.01.

ducing those memories which are successfully recalled.

Average response time. From the results on prolonged reaction times we are justified in concluding only that the ECT patients are slower in getting their answers started, i.e., the very first word of their answer is delayed. But to give an adequate answer to many of the questions, more than one verbal association was necessary. Even a simple question such as "What was the address of that school?" requires a chain of several symbol associations for a complete answer: street number, street name, city, state. A fair proportion of the questions were designed to elicit a much larger number of separate items of information. How rapidly were the ECT patients able to produce an entire series of successive associations?

In assessing the effect of electroshock treatments on memory efficiency, it is essential to examine the speed with which the entire answer is given in order to check on a possible source of error in interpreting prolonged reaction time as an indicator of impairment in memory efficiency. Although slower in getting started, the ECT patients might compensate for the initial delay by completing their answers more rapidly.

The results in Table 4 show that following electroshock treatments there is a significant increase in the time required to answer the questions. Each patient's total response time had been recorded by measuring the time interval between the last word of the question and the last word of his *spontaneous* response (i.e., before any probing questions were asked by the examiner to obtain a more complete answer). In order to obtain the response time per item of information, the total response time for each question was divided by the number of items of information contained in the answer. This value was obtained for each question answered by the

TABLE 4

MEAN RESPONSE TIME PER ITEM OF INFORMATION FOR ALL LIFE-HISTORY QUESTIONS (IN SECONDS)

CONTROL PATIENTS (N=8)			ELECTROSHOCK-TREATED PATIENTS (N=9)		
BEFORE	AFTER	CHANGE	BEFORE	AFTER	CHANGE
11.32	17.23	+5.91	22.13	39.70	+17.57
8.57	9.59	+1.02	10.02	11.03	+ 1.01
12.78	10.72	−2.06	5.67	22.50	+16.83
12.96	7.70	−5.26	11.94	37.03	+25.09
8.40	5.78	−2.62	8.65	11.94	+ 3.29
10.22	6.11	−4.11	8.79	10.07	+ 1.28
8.76	8.88	+0.12	11.66	11.38	− 0.28
15.89	7.39	−8.50	6.37	15.69	+ 9.32
			10.70	9.52	− 1.18
Mean 11.12	9.18	−1.94	10.66	18.76	+ 8.10

Difference between mean changes: t=2.72, p=<.01.

UESTIONS (IN SECONDS)

CTROSHOCK-TREATED PATIENTS (N=9)

AFTER	CHANGE
23.71	+10.96
7.50	− 0.74
12.95	+ 9.69
12.56	+ 3.49
14.14	+ 7.92
9.90	+ 1.70
8.95	+ 1.84
6.32	+ 1.80
5.42	− 0.70
11.27	+ 3.99

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Questions (in Seconds)

OCK-TREATED PATIENTS (N=9)

AFTER	CHANGE
39.70	+17.57
11.03	+ 1.01
22.50	+16.83
37.03	+25.09
11.94	+ 3.29
10.07	+ 1.28
11.38	− 0.28
15.69	+ 9.32
9.52	− 1.18
18.76	+ 8.10

patient, and then his mean value for all answered questions—shown in Table 4—was computed. The mean response time per item of information is a direct measure of the speed with which the patient produced whatever recalled information he was able to give, irrespective of whether or not his answers were complete.

The findings indicate that electroshock had the effect of slowing up the production of personal memory information. The results in Table 4 show the same pattern as the results on reaction time in Table 3. On the initial test there was no significant difference between the two groups, but on retest the electroshock-treated patients showed a marked and statistically significant increase (at the 1 per cent level of confidence). We conclude, therefore, that as a result of electroshock treatments, the patients expended *more* time, in general, when producing whatever memories they were spontaneously able to give.

Rate of recall. The fact that there was an increase in the average response time following electroshock treatments raises the possibility that in addition to the observed inhibition in producing the first relevant association (prolonged reaction time) there may also be a slower performance in producing the subsequent associations contained in the answer. Since the time scores in Table 4 include reaction time, we cannot discern from these results alone whether (*a*) the increase is due entirely to the prolonged reaction time or (*b*) in addition to the initial delay, the patients are also slower in producing the remainder of the information in their answers. The latter alternative refers to the speed of relevant memory production *after* the patient has started to give the answer. We shall refer to this factor as the "rate of recall."

Since we are concerned with the rate of production of successive memory details, we have confined our analysis to the same ten questions used in Table 1, each of which required an answer containing a series of items of information. From the patient's time record, a score on the rate of recall for each of his answers was computed by the following formula: $\frac{TT-RT}{N}$, where TT is the the total response time (interval between the last word of the question and the last word of the spontaneous answer), RT is the reaction time, and N is the number of items of specific information (details) contained in the answer. This gives a precise time score which is the reciprocal of the rate of recall for associated memory details in the spontaneous portions of the answers. When computed in this way, a decline in the rate of recall cannot be attributed to an increase in initial reaction time nor to the occurrence of recall failures inasmuch as both factors are systematically excluded. The rate of recall as measured by the above formula is a new factor which, independently of reaction time and recall failures, can be used as a separate indicator of memory efficiency.

The mean rate of recall score (reciprocal) for each patient is shown in Table 5. On the initial test the ECT group responded at a slightly faster rate than the control group, but the difference is not statistically significant (t=0.80). After electroshock treatments, the ECT patients responded at a slower rate than before treatment. The change in their rate of recall, as compared with change in the control group, approaches the magnitude necessary for statistical significance (p=.07). This finding provides tentative evidence in support of the hypothesis that electroshock treatments have the effect of slowing down the rate of recall.

Qualitatively, we observed that some of the ECT patients displayed a marked increase in hesitations, repetitions, self-corrections, and irrelevant remarks. Sometimes these occurred in the spontaneous answer given to the question, but even more often the patient would give a spontaneous answer which was incomplete and then, when the examiner introduced follow-up probe questions to elicit the omitted details which had been given before treatment, the patient would display a very slow rate of recall in giving the remainder of his answer. Because systematic time records were kept only for the spontaneous portions of the answers, hesitations and irrelevant comments in the nonspontaneous portions of the ECT patient's answers are not at all represented by the results in Table 5. From our inspection of the protocols, we believe that if it had been possible to include the latter instances in our quantitative analy-

sis, the observable decline in the rate of recall following electroshock treatments would have been much more pronounced.

DISCUSSION

A major purpose of the present experiment on memory efficiency was to test certain of the theoretical implications derived from an earlier series of investigations on the psychological effects of electroshock treatments (2, 3, 4, 5). From these studies a set of hypotheses was formulated which provide an initial theoretical framework for explaining various behavioral changes produced by the treatments. The core of the tentative theory is contained in three general hypotheses:

1. Hypothesis I specifies that electroshock treatments produce a subtle impairment in the recall process which persists after the usual recovery period, i.e., after the obvious organic effects of the treatments clear up: "... there is some generalized difficulty or inhibition in recalling past experiences (perhaps as a residual 'organic' effect of the treatments)" (3). In its most general form, this hypothesis predicates a general disturbance in recall functions which is not limited to the posttreatment amnesias or to any particular type of personal memory but extends to all varieties of previously learned symbolic associations.

2. Individual case study observations imply that electroshock-treated patients are able to overcome the residual difficulties in recalling past experiences provided that they have sufficient motivation for exerting the intensified effort necessary. Motivational factors appear to play an important role in the *selectivity* of the unremembered material: the posttreatment amnesias appear to affect disproportionately those memories which would tend to arouse anxiety, guilt, or a lowering of self-esteem. Such observations led to Hypothesis II: The residual memory defect produced by electroshock treatments facilitates the selective forgetting of emotionally disturbing material.

TABLE 5

MEAN RESPONSE TIME (IN SECONDS) PER ITEM OF INFORMATION WITH INITIAL REACTION TIME EXCLUDED: $\frac{TT-RT}{N}$

CONTROL PATIENTS ($N=8$)			ELECTROSHOCK-TREATED PATIENTS ($N=9$)		
BEFORE	AFTER	CHANGE	BEFORE	AFTER	CHANGE
12.82	19.77	+ 6.95	18.49	13.26	− 5.23
16.89	5.33	−11.56	31.79	41.11	+ 9.32
18.28	19.14	+ 0.86	6.65	9.33	+ 2.68
16.03	8.47	− 7.56	9.36	11.43	+ 2.07
9.18	12.13	+ 2.95	14.86	47.36	+32.50
18.55	19.07	+ 0.52	11.01	17.36	+ 6.35
28.95	14.02	−14.93	12.44	7.42	− 5.02
9.87	13.98	+ 4.11	9.02	6.50	− 2.52
			8.39	16.70	+ 8.31
Mean 16.32	13.99	− 2.33	13.56	18.94	+ 5.38

Difference between mean changes: $t=1.58$, $p=.07$.

During the weeks that follow termination of ECT the patients may be able to recover considerably from the extensive, diffuse amnesias which occur during the period of treatment by exerting the necessary effort to regain personal memories which are not readily available to recall; they may remain amnesic, however, for certain memories which elicit anxiety, guilt, or other unpleasant affects when they are motivated, consciously or unconsciously, to avoid expending the extra effort on recalling those particular past experiences (3, p. 380).

3. In general, it appears improbable that the posttreatment amnesias play a *primary* causal role in producing the therapeutic improvement of affective symptoms achieved by electroshock therapy. Nevertheless, various observations obtained in an investigation of changes in affective disturbances (5) provide an empirical basis for Hypothesis III, which specifies that the amnesias contribute—at least as a secondary mechanism—to the

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those memories which would tend
anxiety, guilt, or a lowering of self-
Such observations led to Hypothe-
he residual memory defect produced
roshock treatments facilitates the
forgetting of emotionally disturbing

WITH INITIAL REACTION TIME EXCLUDED:

ELECTROSHOCK-TREATED PATIENTS (N=9)		
RE	AFTER	CHANGE
9	13.26	— 5.23
9	41.11	+ 9.32
5	9.33	+ 2.68
5	11.43	+ 2.07
	47.36	+32.50
	17.36	+ 6.35
	7.42	— 5.02
	6.50	— 2.52
	16.70	+ 8.31
	18.94	+ 5.38

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reduction of affective disturbances. The following theoretical formulation of this hypothesis appears to be consistent with the available findings and observations:

A. By partially eliminating from the patient's consciousness a substantial block of memories which tend to arouse intense affect, the posttreatment amnesias may have the effect of reducing certain areas of affective disturbance. In other words, the posttreatment amnesias may be equivalent to a new mode of defense which has an effect similar to "repression" in facilitating the avoidance of disturbing affect.

B. By providing a new defense mechanism for warding off intolerable subjective states and thereby reducing the frequency and intensity of disturbing affective reactions, the posttreatment amnesias may contribute to the abandonment of some of the pathological symptoms which had previously functioned as a defense against intense affective reactions (5, p. 488).

The above set of hypotheses forms a tentative theory which ties together, to some extent, outstanding psychological changes produced by electroshock treatments. Hypothesis I, which specifies a residual memory defect, occupies a key position since the gist of the theory is that this defect facilitates the development of selective amnesias which, in turn, contribute to the decrease in affective disturbances. The present experiment was oriented most directly toward testing Hypothesis I, but the findings also have some indirect bearing on Hypotheses II and III, since the latter are linked to the first.

The earlier findings in support of Hypothesis I are now supplemented by those from the present experiment. With a new, independent group of subjects, certain of the original observations have been replicated. Even more important, precise quantitative evidence has been obtained on various features of memory performance which previously had not been investigated systematically. All the various indicators of memory efficiency which were investigated consistently point to a residual memory defect which persists after the patients have recovered from the usual cognitive impairment characteristic of the treatment period. Our results show that the performance of the electroshock-treated patients, as compared with the control group, was characterized by the following features which are symptomatic of memory impairment:

1. More questions were entirely unanswered (gross amnesias).

2. Fewer specific details were produced in response to those questions which elicited at least some personal information (partial amnesias).

3. There was a much longer reaction time in responding to those questions to which an answer was given (initial inhibition of recall).

4. In giving whatever information was contained in the spontaneous answers to the questions, the mean response time per item of information was much greater (slower over-all performance).

5. In responding to questions requiring more than one item of information, there was a decline in the rate of recall even after the initial delay in getting started (slower rate of producing successive memory details).

The above findings provide clear-cut evidence in support of the general hypothesis that following electroshock treatments there is a residual memory deficit that is sufficiently generalized as to affect the recall of routine life-history information.

The results not only tend to confirm Hypothesis I but they also contribute some additional weight to Hypothesis II. One of the most elementary predictions from the second hypothesis is that whenever a patient displays posttreatment amnesias, he should also be found to display signs of a more general memory defect. This is a necessary, although not a sufficient, condition for assuming that the latter factor plays some causal role in producing the former. Our results tend to confirm this elementary prediction since we have found in our group of electroshock-treated patients the joint occurrence of: (*a*) gross recall failures (posttreatment amnesias) and (*b*) the subtler forms of memory difficulty which imply a more general deficit in recall functioning.

The hypothesis under consideration also postulates that the memory disturbance is of such a character that it *requires the patient to exert additional effort* in order to recall his past experiences. This is the critical factor which is assumed to bring about a shift in the dynamic balance of competing motivations involved in normal recall functioning, thereby creating circumscribed amnesias simi-

lar to those occurring in hysterical memory disorders. Loosely speaking, the hypothesis asserts that electroshock treatments give rise to "artificially induced" repressions because of the heightened effort required for bringing memories into consciousness, enabling anxiety-avoidance motives to become predominant over "reality-testing" motives. Our present findings tend to bear out the assumption that the memory defect produced by electroshock treatments is of the type which necessitates additional effort to recall past events. As we have seen, the memory disturbance is not an all-or-none affair; our results contradict the assumption that in electroshock-treated patients, personal memories are either totally unavailable to recall or else immediately available to consciousness. The fact that a longer *time* is necessary for recalling routine personal information implies that more effort is required. Unless added time is invested in concentrating on the memory task, the appropriate memory apparently fails to emerge into consciousness.

Often the patients did, in fact, stop far short of complete recall, and it appeared that it was only as a result of prodding by the examiner that they continued to "work" on the task until a more complete answer was attained. The large number of probing questions required to elicit details about past events probably functioned to keep the patients motivated to overcome the memory defect. Presumably, if the patients had been kept at the task for a longer time, many more of the omitted details would have been forthcoming as was noted in the earlier investigation (2, 3).

Although the present evidence tends to confirm the assumption that recall is more effortful following electroshock treatment, it is not sufficient to establish Hypothesis II, since we have not demonstrated that there is a causal relationship between the memory defect and motivated forgetting. Further research on the selectivity of the posttreatment amnesias and on their relationship to the motivational structure of individual patients is obviously required.

Further case studies and other types of research are also needed for testing Hypothesis III, which assigns a (secondary) causal role to the posttreatment amnesias in bringing about emotional improvement. The present experiment adds only a slight increment to the empirical plausibility of this hypothesis. The results on gross recall failures tend to confirm one of the elementary assumptions on which the hypothesis is based, namely, that posttreatment amnesias regularly occur following electroshock. The fact that our electroshock patients displayed evidence of amnesias and also had responded to electroshock therapy with some degree of clinical improvement (especially with respect to the clearing-up of affective symptoms) confirms the earlier observations (5) on the joint occurrence of emotional improvement and posttreatment amnesias.

It is worth noting that the absolute number of gross recall failures observed in the earlier study (3) was far greater than in the present study. Roughly, the same number of questions was asked in the two studies. The former study, however, included many questions designed to elicit memories of an *emotionally disturbing* character (e.g., circumstances involved in the onset and development of the mental disorder, family quarrels, personal failures, etc.), whereas, the present study was deliberately restricted to routine information of a comparatively *neutral* character.

Our results show that the average number of questions completely failed by the present group of electroshock patients was slightly more than 3 out of 33 (approximately 10 per cent). In the earlier study, the proportion of failed items was far higher: the majority of patients had been asked some 30 to 40 questions about specific past events and were totally unable to recall from 10 to 20 experiences (over 30 per cent) that had been recalled in the pretreatment session. Since the same types of hospitalized mental patients were used in both studies, it seems fairly likely that the higher proportion of gross recall failures in the earlier study is attributable to the difference in the type of personal material covered by the two sets of questions. This incidental observation is consistent with the hypothesis that the posttreatment amnesias are selective in character, affecting emotionally disturbing memories more often than emotionally neutral memories.

SUMMARY AND CONCLUSIONS

1. An experimental study of the effects of electroshock treatments on memory efficiency was carried out in order to test various hypotheses derived from an earlier series of investigations. The experimental design consisted of testing each of nine patients in the electroshock group before the series of treatments began and again after the series was terminated. The same observations were made on an equated control group of eight patients who received no form of shock therapy. The recall test was administered in a face-to-face interview and consisted of a standardized set of questions covering routine personal information: school and job history, activities during the war, and other simple facts about the life history. Various measures of recall failure and of the latency of response were systematically investigated. The posttreatment test was administered four weeks or more after the last electroshock treatment, at a time when the "organic" impairment syndrome which occurs during the treatment period had already cleared up. Consequently, all of the findings refer to the *residual* effects of the treatments, after the usual recovery period.

2. The quantitative findings on recall failures show that the electroshock-treated patients, as compared with the control patients, were unable to answer a significantly larger number of questions about their life history and, when they were able to give an answer, their responses contained significantly fewer details. These findings tend to confirm an earlier study in which gross retroactive amnesias were consistently found as a residual effect of the treatments. In addition, the findings on incomplete answers indicate the presence of subtle, sharply circumscribed amnesias which consist of a loss of circumstantial details about past experiences that are partially remembered.

3. A statistically significant increase in reaction time was found for the electroshock-treated group, indicating that they exhibit an initial inhibition or delay in getting started when they are producing those memories which are successfully recalled. That the prolonged reaction times reflect a genuine decline in the speed of memory functioning is indicated by additional findings which show that these patients exhibit a statistically significant increase in the average amount of time per item of information. In part, this slower over-all performance is attributable to the initial delay in getting started. In addition, there is evidence that even after the initial delay, they produce successive details at a slower rate of speed. Qualitatively, the decline in the rate of recall was observed in the form of more expressions of doubt, frequent self-corrections, and an increase in irrelevant verbalizations.

4. The indicators of memory efficiency investigated in this experiment had been selected so as to provide evidence relevant for testing a set of interrelated theoretical propositions derived from an earlier series of investigations. The hypothesis of primary interest was the following: After the usual recovery period following electroshock treatments, there is a generalized, residual impairment in recall processes. All the findings and observations from the present experiment consistently tend to confirm this hypothesis and, therefore, contribute to the empirical basis for a tentative theory which postulates this type of impairment. According to the theory, the generalized memory impairment plays a causal role in the development of newly formed repressions (selective amnesias) which, in turn, contribute to the reduction of affective disturbances. In particular, certain of the findings were shown to support the hypothesis that the residual memory impairment increases the effortfulness of recall which would facilitate the selective forgetting or repression of emotionally disturbing material.

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