THE EFFECT OF RECALL CUING ON THE POST-ECT ANTEROGRADE MEMORY DYSFUNCTION

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by

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An experiment was conducted to determine if the memory loss following electroconvulsive therapy (ECT) was due to acceleration of loss of material from storage or to impairment in the ability to retrieve that still in storage. The method used to distinguish between these two possibilities was recall cuing, which should serve to provide access to any material retained. Twenty psychiatric inpatients heard equivalent lists of words, grouped into five taxonomic categories of three words each, prior to and after a series of bilateral ECT treatments. After each list, two recall periods were given, with three hours between them. Ten subjects were tested by means of free recall, and ten were given category names as cues to aid recall. Recall of words, categories, and words per category dropped significantly after ECT and over the time lapse, but though the administration of ECT resulted in anterograde amnesia, there was insufficient evidence to conclude that the amnesia was due to acceleration of forgetting. Thus, a retrieval-failure hypothesis of the post-ECT amnesia was also not supported, and though cuing resulted in higher category recall, it had no effect on word recall, and did not modify the recall loss over time and after ECT. It was concluded that an impairment in retention or retrieval was not a significant part of the post-ECT memory disorder for the present subjects; rather, the disorder was likely a result of difficulty in getting the material to be learned into storage. Large between-subject variability and the possibility that the cuing procedure was not maximally effective for this group of subjects were also discussed in relation to the findings.

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Memory Dysfunction and Electroconvulsive Therapy

Since Cerletti and Bini's introduction of electroconvulsive therapy (ECT) in 1938, many psychiatric patients have been plagued with the annoying, and sometimes frightening side effect of a memory loss or memory disorder. The cause of this disruption is thought to be the local effects of ECT upon the critical temporal lobe areas that underlie the usual sites of the electrode placement (Inglis, 1969). The extent of the influence is quite variable. According to Squire and Miller (1974), memory functions may improve markedly or even return to pre-ECT levels within hours of a series of ECT treatments, or the amnesic effect may still be detected weeks after treatment. On the other hand, Cronholm and Molander (1964) found no difference in immediate reproduction or in forgetting from pre-ECT to one month after the last ECT in the series. Massing of treatments and increasing the intensity of the shock have been noted to enhance the amnesia (Abrams & Fink, 1972; Valentine, Keddie, & Dunne, 1968).

The disruption of the memory function may be classified into three categories, according to Cronholm (1969):

> "First, there is a total amnesia for the period of unconsciousness during treatment and for the period of profound confusion immediately following it. Second, there is a retrograde amnesia, that is, difficulties in remembering events before treatment, especially what happened just before it, but also in recalling certain things that used to be well-known.

> Third, there is an anterograde amnesia, that is, the patient may complain of difficulties in remembering recent occurrences, in learning new names, in recalling what has been read in the papers, and so on."

An important conclusion may be drawn from numerous studies: The memory impairment produced by ECT can be separated from its therapeutic action. It has been reported that the generalized convulsion produced by the shock is therapeutically effective, whereas effects other than the seizure are of greater importance for the retrograde and anterograde amnesia after ECT (Cronholm, 1969). Further evidence for this conclusion is contained in the comparison of the effects of bilateral and unilateral ECT, where administration of the electrical stimulus to the non-dominant hemisphere only has often resulted in less verbal memory loss than bilateral administration, but has shown similar therapeutic effects (Hurwitz, 1974). For example, Costello, Belton, Abra, and Dunn (1970) tested recall, recognition, and relearning of paired associates in patients treated with unilateral stimulation to the non-dominant hemisphere as compared to patients treated bilaterally. Patients receiving unilateral treatment performed better on recall and relearning than those treated bilaterally. Patients' responses on self-rating scales of depression showed no evidence of difference in therapeutic benefit between unilateral and bilateral treatments.

The study of memory and memory dysfunction has provoked vast amounts of research. Generally, though, there are two accounts which recur in the explanation of disturbances in both normal and

abnormal memory functioning. One of these is the disruption of storage or retention, involving either a failure to adequately encode and store the material or a loss or deterioration of adequately stored information. Such a disruption would lead to unavailability of the material. The other account postulates a disruption of retrieval of stored material, through a failure to find information that is stored. A disturbance in the retrieval process would result in the inaccessibility of potentially available material (Tulving & Madigan, 1970; Weiskrantz, 1966). The basis for the distinction between these two accounts will be more fully discussed later in terms of the measures used to differentiate them.

The unavailability - inaccessibility distinction appears to be a suitable framework in which to attempt an analysis of the post-ECT memory deficit; however, due to the difficulty of assessing what was initially stored independently of what has been lost from storage to the time of measurement, the issue of unavailability can perhaps be examined more productively in terms of the loss of retention of that information which was once clearly recalled. In other words, a post-ECT decline in recall may be investigated with a view to determining if the impairment is due to a failure to retain material previously recalled or if the impairment really reflects a failure to retrieve stored material.

The focus of the literature review is, first of all, the memory changes that have been linked with electroconvulsive therapy,

and second, a discussion of forgetting from long-term memory in terms of a failure of retention or retrieval as a particular aspect of the human anterograde memory change. An evaluation of the factors that influence forgetting will lead to specific predictions regarding the deficit after ECT. Though the emphasis of the review will be placed on anterograde amnesia, a discussion of retrograde amnesia is also included for completeness of the background information.

The Nature of the Memory Dysfunction

Retrograde Disorder

Some post-ECT loss of memory for events that occurred prior to the convulsive treatment, or retrograde amnesia (RA), has been demonstrated in many studies using animal subjects. However, there appears to be a dearth of such research with human subjects. Early studies have given evidence of forgetfulness for remote personal events following electroshock (Brody, 1944; Janis, 1950). Ebtinger (1958) reported that such 'gaps' of memory may remain permanently. Zubin and Barrera (1941) demonstrated diminished recall of paired associates after shock treatment, and Mayer-Gross (1944) demonstrated the presence of a retrograde effect of post-shock amnesia in the recall and recognition of visual stimuli. More recently, Costello et al. (1970) had patients learn a list of paired associates prior to bilateral, unilateral dominant, or unilateral non-dominant ECT. After four

treatments, results showed impaired retention in those patients receiving bilateral or unilateral dominant ECT, but not if the unilateral treatment was administered to the non-dominant hemisphere. Miller (1970) also found evidence of RA associated with ECT. Jarvik (1972) observed that RA is more difficult to demonstrate in humans than in animals, but that brief RA has been induced in man by ECT.

The faulty post-treatment retention of pre-treatment experience may be due to either of two factors: i) erasure of the trace, failure of consolidation of the trace, or any other failure of storage; or, ii) interference with an adequately stored trace or some other failure of retrieval (Weiskrantz, 1966). The first factor is one of unavailability of the material, and the second factor is one of inaccessibility of available material. RA has been traditionally explained in terms of a neural consolidation theory: For any memory to become established it is necessary for the original memory traces, which are only temporary in form, to be transferred to a more permanent form. ECT during the consolidation period will disrupt the process, resulting in the unavailability of material and consequent retrograde amnesia (Miller, 1967).

Several models of possible relationships between long-term memory (LTM), short-term memory (STM), and the RA gradient have been reviewed by McGaugh and Dawson (1971). They reached the conclusion that the model which accounts for the bulk of the data is one which suggests that electroconvulsive shock (ECS) impairs consolidation by speeding the decline of STM. LTM is hypothetically related to STM in that the asymptote of LTM is determined by the duration of STM.

This model can therefore account for variation in the RA gradient with variations in treatment strength or delay.

The results of many animal studies in which ECS was the amnesic agent have provided support for the consolidation hypotheses. McGaugh and Landfield (1970), who trained mice to avoid footshock in a one-trial inhibitory avoidance task, showed that retention over one hour occurred if the interval between training and ECS was 20 seconds, but not if the ECS was given five seconds after the training. These findings were consistent with the consolidation interpretation that memory storage processes become decreasingly susceptible to disruption with increases in time following training. Similarly, Luttges and McGaugh (1967), using the same sort of task in which mice were given footshock as punishment when they stepped off a platform, found that animals who received the footshock and no ECS had high retention as demonstrated by high latencies in stepping behavior, and this retention performance was stable over a one month period. However, mice given both footshock and ECS had latencies as short as the un-punished groups, and the amnesia was stable over at least a one month period. As memory did not return, it was assumed that the ECS blocked the storage processes by impairing the neurobiological processes involved in the formation of long-term memory. The conclusion that memory storage processes are active for fairly long intervals following an experience has also been supported by studies on the facilitation of learning by drug administration (McGaugh, 1969; McGaugh & Krivanek, 1970).

Findings from these and other studies have given rise to a formulation of what are thought to be three common properties of consolidation. The first of these is the property of fixation, which requires that a memory trace must be transferred to a permanent form and be fixed. The second property is the time-bound effect, since the fixation of the traces must require time. The third property is the permanence of the amnesic disruption, which means that the memories are irretrievably lost if the consolidation process is disrupted (Lewis, 1969). However, results of some recent studies have questioned the validity of these properties.

Lewis, Miller, and Misanin (1969) have demonstrated that in animals familiarized with the apparatus beforehand, even with only .5 second intervening between learning and ECS, no RA was present. The existence of a process of fixation was not supported by these findings, since any 'fixation' occurring would have to be practically instantaneous. According to consolidation theory, any sort of disruption outside the limits of the fixation time could not give rise to RA.

Researchers have looked closely at the generally accepted premise that consolidation occurs over a significant period of time and only during this time can traces be disrupted (the time-bound effect). Misanin, Miller, and Lewis (1968) found that RA could be produced 24 hours following learning if a conditioned stimulus that had been paired with the footshock during training immediately preceded the ECS. With amnesia occurring after such a long interval, it seems that the interpretation of a time-bound effect is certainly open to question.

Finally, memory functions have been found to recover under certain conditions, for example, when reminder trials are given (Miller & Springer, 1974; Zinkin & Miller, 1967), and after administration of drugs (Mah & Albert, 1975). Obviously, the amnesia is not always permanent, and the assumption of permanence of RA is discredited to an appreciable extent.

The failure to support the predictions derived from the theory of consolidation has led to a decline in its popularity as a 'complete' explanation for retrograde amnesia, although some theorists still subscribe to it (Dawson & McGaugh, 1969; Gold & King, 1974). Other hypotheses based on failures of storage have been put forward. For instance, Williams (1969) regarded RA as being much like normal forgetting. Her patients were given paired associate lists to learn prior to ECT; the pairs that were highly associated semantically were remembered better after the treatment than those not associated, and pairs that involved association by mediation were intermediate between these two extremes, showing a pattern of learning and forgetting similar to that of normals. She suggested that much is forgotten unless something happens to make us rehearse and remember; ECT may disrupt the necessary rehearsal.

Recently, alternatives to theories of trace consolidation or storage disruption have been put forward, one being an explanation in terms of interference or some other failure of retrieval of the stored trace (Weiskrantz, 1966). Lewis (1969), in a review of animal literature, suggested that many forms of suppression, competition, and inhibition may actively prevent the recall of stored information, and

Meyer (1972) also supported the idea that ECT suppresses, rather than destroys, engrams. Kesner and D'Andrae (1971), who gave rats shock contingent upon a bar-press, found that those given ECS five minutes after the shock showed permanent amnesia for it, while those given ECS 24 hours after the contingent shock showed only temporary amnesia, and later recovered the memory for the shock. They concluded that information storage is disrupted by ECS within some temporal bounds, but even when information is stored adequately, the retrieval gradient may be affected by ECS.

From the results obtained in the previously mentioned reminder studies, Miller and Springer (1974) have theorized that the RA gradient reflects the vulnerability of an aspect of retrieval that facilitates future access to stored information. Squire (1974, 1975) found that ECT could produce a memory impairment for events 10 to 20 years in the past, which could not be explained in terms of conventional storage malfunction theories. It was suggested that the deficit could reflect a transient impairment in retrieval. Support for this position was gathered in a further study by Squire, Slater, and Chace (1975). Long-term RA was found to occur one hour after the fifth ECT, but the memories were recovered to some extent one to two weeks after the completion of the course of ECT. It was suggested that access to some memories was temporarily disrupted after ECT, but that the memories themselves were not erased.

In summary, although many studies have supported the hypothesis of trace consolidation or storage disruption, it is obvious that the influence of ECT in retrograde amnesia is not limited to such an effect,

and that the use of these theories may have been overextended. Properties central to trace consolidation theory have been challenged in the discovery of evidence that cannot be interpreted in a consolidation disruption framework. This evidence indicates that ECT also produces a malfunctioning of retrieval processes, and that this disturbance is an integral part of the post-ECT memory deficit.

Anterograde Disorder

Anterograde amnesia (AA) may be described as a post-ECT loss of memory for events that occurred after the treatment. This 'loss of memory' may be an actual loss or unavailability of material due to a defect in storage or retention. Such a defect may involve either or both of two processes. First, there may be difficulty in getting material into storage, shown as an impairment in learning and generally defined as increased trials to a criterion. Second, there may be difficulty in retaining learned material in storage, which is generally measured by the decrement in performance on a delayed recall test as compared to immediate recall. However, it is also possible that what the 'loss' actually entails is a loss of accessibility to material that is adequately retained.

Certainly, in the case that ECT administration gives rise to learning difficulties, a memory deficit will be the result, since if material is imperfectly acquired there is a lesser chance of its recovery or subsequent expression (Lewis, 1969). There has been evidence to suggest that there is such a post-ECT impairment in

acquisition (eg. Cohen, Noblin, Silverman, & Penick, 1968). Miller (1970) stated that the learning deficit becomes more apparent with increasing number of electroconvulsive treatments. Although attention and orientation are generally disrupted for a time after ECT, in one study these factors were not able to completely account for the post-ECT impairment of verbal learning (Fromholt, Christensen, & Stromgren, 1973).

However, not all findings point unequivocally to a learning impairment. Cronholm and Ottosson (1961) suggested that, except for the few hours immediately after ECT, anterograde amnesia did not involve a disturbance in learning. Rather, the treatment reduced the learning problem associated with the depression, but at the same time produced a decrease in the recall of material over a three hour period. An increase in forgetting may also, in part, account for the impaired relearning scores in the patients of Costello et al. (1970). These results would imply that whether or not a disturbance in initial acquisition is present after electroconvulsive therapy, the possibility of a disruption of some other memory function still exists. In fact, since it cannot be assumed that what is recalled on even the first trial after learning accurately represents the sum total of what has been 'acquired', and since it cannot be assumed that other factors have not already played a part in diminishing recall, there is certainly a justification for investigating the effect that ECT has on the subsequent fate of material that has been demonstrably acquired.

Some researchers have attempted the assessment of disturbances

in post-ECT memory that are shown after the material has been learned (post-ECT forgetting). In concordance with the results of Cronholm and Ottosson (1961), some studies have shown a positive relationship between the length of the retention interval and the severity of the memory defect (D'Elia, 1971; Ottosson, 1969). However, forgetting is merely the failure to recall, after an intervening period of time, something that was previously learned. This decrement in performance may be the result of either the unavailability of the trace of the item in memory storage at the time of recall, or the inaccessibility of an otherwise intact memory trace (Tulving & Pearlstone, 1966). In order to clarify where the dysfunction lies in the post-ECT recall failure, it is necessary to use a method that will allow for the differentiation between these two possible alternatives.

Explanations of post-ECT anterograde forgetting are conflicting, and this would appear to be due to a great extent to variability in the method used to study the deficit and in the interpretation of the results obtained. Dornbush, Abrams, and Fink (1971) reported that subjects with bilateral ECT treatment, but not those with unilateral right-sided treatment, showed deficits on an auditory short-term memory test; a positive relationship between the extent of the memory impairment and the length of the retention interval was found. Since both treatment groups performed similarly at 0 seconds delay, indicating that ability to perceive and retrieve stimuli was unimpaired, these researchers suggested that ECT affects the storage or retention of material. However, such an explanation is valid only if retrieval

after zero seconds can be considered to be the same as retrieval after a longer period of time. No evidence was cited to suggest that this is so. Stones (1973) in a similar paradigm came to quite different conclusions. He found that STM for verbally presented material was deficient after ECT, but found no evidence to indicate that the extent of this STM deficit was significantly related to the length of the retention interval. As there was no interaction between length of the retention interval and the presence or absence of ECT, it was concluded that the post-ECT STM deficit could not be explained in terms of a defect of retention, but could possibly be accounted for as a defect at encoding or retrieval.

On a long-term level, Fromholt et al. (1973), using a delayed reproduction version of the Wechsler Memory Scale Logical Memory subtest, found that in addition to a reduction in verbal learning capacity after bilateral ECT, the probability of forgetting was greater for the bilateral than for the unilateral non-dominant group; this was true regardless of the level of immediate reproduction achieved. These results confirmed their hypothesis that bilaterally treated individuals are greatly impaired with respect to retention of material, but shed no light on the question of whether the material actually became unavailable, or was really only inaccessible.

A study of Squire and Miller (1974) showed that the ability to retain material over a 24-hour period was significantly poorer and improved more slowly on successive recall trials with increasing number of ECT treatments, as compared to the ability to retain material

for 30 minutes. They concluded that the acceleration in forgetting after ECT reflected a failure to effectively acquire and consolidate new information. However, this explanation cannot fully account for their results, as it was obvious that subjects subsequently 'forgot' material that they had previously possessed. These researchers also rejected a retrieval-failure explanation as subjects did not spontaneously remember things previously forgotten, but they did not use a technique that might help subjects overcome a block in retrieval, if such existed.

In essence then, though electroconvulsive therapy may well have disruptive effects on the rate of acquisition of material, such a disruption cannot be taken as a total account of the post-ECT deficit. Also, since 'acquisition' is itself a complex and difficult process to assess, evaluation of subsequent processes may best be carried out using as a basis for measurement the information that has been demonstrably acquired. In subjects receiving electroconvulsive treatment, investigations into the post-acquisition fate of material have given strong indications that information is forgotten much more rapidly than in subjects not given shock treatments. However, research is somewhat scanty, and the methods used usually have not allowed for a full explanation of the decrease in recall with increase in inter-recall period. That is, the methods used have not been sufficient to determine if there has been an actual loss of stored material, or if there has been a loss of ability to recall that which was retained.

A method that allows for differentiation between failure of retention and failure to retrieve retained material has been used in the assessment of forgetting in normal subjects and in patients with other amnesic disorders. Thus, a review of the work in these areas should allow clearer predictions to be made as to the nature of the post-ECT memory deficit.

Forgetting

When a person recalls or recognizes something at one time, but fails to do so on a subsequent occasion, forgetting is said to have occurred. Electroconvulsive stimulation is just one of a class of stimuli that has been found to be correlated with a disturbance in memory, as manifested by a partial or total inability to recall past experiences. Other trauma that may produce this performance decrement include organic brain disease (Brion, 1969), head injury (Inglis, 1970), senility (Kral, 1969), neurosurgery (Milner, 1966), and certain drugs (Levin, 1959). Of course, so-called 'normal' forgetting over a period of time is also represented by the above definition.

The period of time covered by an amnesia can be quite variable, depending on the extent of the trauma. In a review of literature, Inglis (1970) reported that the ultimate duration of RA is usually less than half an hour, taking into account the shrinkage of the retrograde gradient, but if the injury is severe or the amnesia chronic, the deficit may extend backwards into remote memory. In the case of brain lesions, the associated anterograde amnesia is irreversible;

memory functions never reach their premorbid level.

As methodology and findings vary a great deal in memory research depending on the type of subject studied, the following discussion has two general subdivisions, forgetting in normal subjects and forgetting in amnesic subjects. When referring to traumatic memory loss, all references will deal with the anterograde aspect of memory disorder, since the research described in this thesis is primarily concerned with the failure to recall events that occur after electroconvulsive treatment.

Forgetting in Normal Subjects

As previously stated, forgetting may be the end result of either unavailability of the information in store or inaccessibility of stored information (Tulving & Pearlstone, 1966). In other words, forgetting may be due to a failure of retention or to a failure of retrieval. However, in order for this distinction to be valid in a practical sense, it has to be shown that material can possibly be retained in memory, but not recalled upon demand.

A method that has been used to demonstrate the difference between availability and accessibility is recall cuing, which leads to greater accessibility of stored information and increases the probability of retrieval success. Using taxonomic category names, which were given to all subjects at input, Tulving and Pearlstone (1966) found levels of cued recall to be higher than noncued recall,

the difference varying directly with list length and inversely with number of items per category. Their results suggested that accessibility of higher-order units (in this case, categories), in which a number of items have been clustered together in some way, depends on appropriate retrieval cues and on the total number of stored higher-order units. On the other hand, accessibility of items within the higher-order units is largely independent of these variables. Their explanation for the success of the cued recall was that use of this method reinstated at the time of recall the stimulating situation that had been present at the time of input, thus facilitating retrieval (Tulving & Pearlstone, 1966).

A wide variety of specific retrieval cues that are related to the to-be-remembered (TBR) word (including initial letter, synonymic cues, descriptions of graphemic features, and taxonomic category names) have been reported to facilitate recall (Tulving & Osler, 1968). However, this facilitation occurred if and only if the information about the cues and about their relationship to the TBR words had been stored at the same time as the information about the membership of the TBR words in a given list. In other words, cuing at recall alone was ineffective. Using taxonomic category names, Crouse (1968) also varied the presence or absence of cues at retrieval factorially with the presence or absence of cues at input. Retrieval cues facilitated recall when cues had been present at input, but not when they had been absent. His explanation, like that of Tulving and Pearlstone (1966), was that retrieval cues may assist recall when they are success-

ful in reinstating the storage tags that were formed during input.

Results of many studies reporting no facilitation of recall with response cuing can be explained in terms of the hypothesis of Tulving and Osler (1968) that retrieval cues must be associated with TBR items at input. For example, Slamecka (1968) found no beneficial effects of cues (or context), and in fact, the context situation sometimes exerted a slightly interfering effect upon recall. Similarly, Allen (1968) found no evidence that responses already given serve as cues for items yet to be recalled. The type of cues used in both these studies were intralist cues: The assumption was made that giving items from the list recently learned would trigger recall of other responses. Since no information about the relationship of the cue and the TBR word was stored at the same time as the words themselves, in light of the previous findings of Tulving and Osler (1968) it is not surprising that the use of these cues did not result in increased recall. Freund and Underwood (1970) also argued that a cue could fail because it was not actually a part of the stored memory for the word, just as it could fail if the word itself was not stored.

Freund and Underwood (1969) found no differential effects of cuing over free recall. Their method was to use three instructional conditions at learning, which emphasized serial position, alphabetical organization, interitem associations, and an uninstructed control group. All subjects were given the same list to learn. A free recall trial was then given, followed by a cued recall trial where subjects

were presented with half of the words from the learned list, appropriate to one of the instructional conditions. The cued recall trial resulted in an average increment of less than one-half word, which did not differ across the twelve conditions. Other negative findings were reported by Hicks and Young (1973). They presented their subjects lists of words that were either grouped into categories according to initial letter or were not categorized. Recall was either free or cued by initial letter. The categorized lists were recalled better than uncategorized lists, but free recall was better than cued recall. They concluded that under certain conditions cues hinder recall performance by interfering with the organization that the subject has imposed on the list.

It is not clear that the most effective cuing procedure was used in the above two studies. What is clear in these conflicting results on cuing is that the procedure maximizing the effectiveness of cues requires associating them with the TBR words at input, as done by Tulving and Osler (1968). Since cuing is not an end in itself, but only a means of demonstrating availability of retained information, the strongest possible cue should be used, to provide the highest degree of association.

Allen (1969) suggested that cuing can effectively aid recall only if two conditions are met. The first was that the amount of subjective organization prior to cuing must be optimal. Too little organization may lead to too few functional connections, thus not aiding recall. Too much organization may also lead to a minimal effect

of cuing, as there may be little left to cue.

The second condition for effective cuing was that the cue be related to the organization the subject has imposed on the list. Some researchers (eg. Bilodeau & Blick, 1965; Loess & Harris, 1968) have found cuing at recall to be facilitative when no cues have been given at input. Tulving and Osler (1968) explained this apparent inconsistency with their hypothesis in that appropriate coding at input may take place even if the experimenter does not explicitly suggest to the subject how he is to code the TBR words. Then, the specific retrieval cues provided by the experimenter at output may overlap with the subjective coding responses that have occurred at input. The results of Loess and Harris (1968) confirmed the value of pre-existing subjective organization, as cued performance was superior to noncued performance only if the subject was first given a brief opportunity for free recall. Freund and Underwood (1970) noted that cuing by the use of related nouns and adjectives was effective in increasing the probability of recall, but that when cues were given at input only, recall was lower than if no cues were given. It would appear that the cues disrupted the normal or preferred subjective encoding habits.

Both of the above criteria for effective cuing can be met, as suggested earlier, by using the same cues at retrieval as have been used at input; then the encoding of the list words is controlled to a large degree, and the cues can be used to their full extent. As Tulving and Thomson (1973) stated, specific encoding operations performed on what is perceived determine what is stored, and what is stored determines

which retrieval cues are effective in providing access to the stored material. The potency of the retrieval cue is thought to vary directly with the strength of the pre-experimental associations between it and its associated words (Tulving & Patterson, 1968), so a high probability of recall of related as compared with unrelated words is to be expected. The results of Freund and Underwood's (1970) study with related nouns and adjectives give support to this interpretation. Thus, cues should be even more efficient in overcoming retrieval deficits if they have previously been strongly associated with TBR words for the subject population.

It appears, therefore, from the research that has been conducted that provision of appropriate cues can indeed lead to higher recall of verbal material. This finding of course indicates that nonrecall of some items may reflect the limited capacity of the retrieval mechanism to find access to the information available in store, rather than a failure to get appropriate information about the list items into store or a failure to maintain the information in memory store (Tulving & Patterson, 1968). However, to use cues to validly estimate how much is available but otherwise inaccessible, efforts should be taken to see that the cues are maximally effective. This may be done by using cues that are related in some meaningful way to the TBR words and by associating the cue and the TBR words at input. It may be possible, then, if these conditions are met, to use a cuing technique in order to determine if post-ECT forgetting is a result of a retention failure or a retrieval failure.

Forgetting in Amnesic Subjects

One drawback that had to be overcome in order to investigate forgetting in amnesics is their generally low level of initial learning, since a prerequisite for forgetting is that learning has occurred and that recall or recognition of the stored information was in principle possible at some stage. Attempts to find methods to assure a rate of learning for amnesics comparable to that of normal subjects have suggested that the process of forgetting in these two groups may not be so dissimilar as one would expect.

A study of Warrington and Weiskrantz (1968) demonstrated that their amnesic groups had impaired retention relative to that of controls. This finding did not mean much in itself, though, since by conventional means the amnesics failed to achieve sufficient learning to compare the two groups adequately in terms of rate of forgetting. However, using the technique of partial information with fragmented items (Warrington & Weiskrantz, 1968) and with whole letters (Warrington & Weiskrantz, 1970; Weiskrantz & Warrington, 1970) at learning and recall, patients were able to learn to criterion rapidly, although not as rapidly as controls, and they demonstrated retention savings and faster relearning than by conventional methods. The use of the technique at the input stage alone resulted in no differential efficacy, whereas the use of partial information at retrieval alone resulted in significantly higher retention than either free recall or recognition in amnesics. In other words, the method of learning did not differentially affect the level of recall in the amnesic subjects, but the method of retrieval did; recall

by partial information was a particularly favorable retrieval method for the amnesics but not for the controls.

The above results would suggest that the increased forgetting in amnesics as compared to normals is due to an increase in the disruption of retrieval processes that is normally noted over time. That the retrieval process is disrupted in amnesics was also supported by Buschke and Fuld (1974). Using techniques of selective reminding of those items not recalled on the immediately preceding trial and restricted reminding of each item only until it had been recalled once, they concluded that the patient's impairment in learning was caused mainly by her impaired retrieval from long-term storage. These reminding techniques allowed the subject to show learning by spontaneous retrieval without the confounding variable of continual presentation. Although initial storage appeared to be somewhat impaired, retention in long-term storage was intact, and recall failures were due to retrieval dysfunction.

Results from studies using cuing techniques have also lent support to the notion that an impairment in the retrieval process, more pronounced than that occurring normally over a time delay, is a significant part of the amnesic memory dysfunction. Warrington and Weiskrantz (1971) found no qualitative differences in the performance of controls and amnesics on perceptual classification, perceptual isolation, subjective categorization, and semantic clustering, implying that organization of perceptual and mnemonic information in the amnesic patient is normal. However, cuing by semantic category significantly raised the performance

of amnesic patients as it did the performance of normal subjects. Taxonomic and phonemic clustering of lists at input has also been found to be useful to amnesics, but significantly less so than to controls (Baddeley & Warrington, 1973). However, cuing at retrieval with unrecalled items in lists that were both organized and blocked was useful with semantic, phonemic, and imagery lists. Thus, conditions at input were shown to have much less effect on retention in amnesic patients than did conditions at retrieval, suggesting that in fact it is the retrieval process that is disrupted.

The properties and limits of cued recall in amnesic patients were explored by Warrington and Weiskrantz (1974). Relatively unimpaired retention over long periods of time was possible with phonemic cuing in amnesics. The phenomenon of differential efficacy of cued recall in amnesic subjects over controls was replicated, and restricting the number of response alternatives was shown to be more effective in amnesics than in normal subjects. Cuing by semantic category was effective for both amnesics and normals.

Since the use of these various methods has raised the level of recall to that of normals, increased forgetting in amnesics can be attributed to a failure to access effectively the information in store, rather than a failure to retain this information. An explanation for the disruption of accessibility that has been proposed is that stored information is not inhibited or not dissipated in the proper way, and thus leads to increased interference. This concept is seen more clearly

in light of the finding that the differential effect of partial information and cuing at recall for amnesics over controls increases if the number of response alternatives can be reduced (Warrington & Weiskrantz, 1970, 1974). The fewer the possible alternatives, the less interference there will be when access to the higher-order unit has been gained.

The amnesic retrieval defect can be compared to the loss of memory that is normally observed over a time delay; in fact, it has been shown that memory in amnesic patients tested after one minute is directly comparable to normal memory tested after one week, in both a qualitative and quantitative sense (Woods & Piercy, 1974). Williams (1969) cited data to support her hypothesis that time lapse and cerebral disorganization have parallel effects on the recall of past events. Over a time delay, actions or impressions intervening between perception and retrieval cause interference which disrupts retrieval processes; cerebral disorganization in amnesia may result in increased interference from unnecessary information, which accentuates the difficulty in retrieval of past experiences normally seen after a time lapse. That indeed there is more interference to be overcome in the disorganized state than in the normal state is proposed by the 'rule of forces' (Luria, 1971). Normally, strong stimuli evoke strong cortical reactions whereas weak stimuli evoke weak cortical reactions; in pathological states of the cortex, this rule may break down, leading to 'inhibitory states' in which both strong and weak stimuli may evoke equal reactions or weak stimuli may evoke stronger reactions than strong stimuli. The effect of such increased interference from unnecessary stimuli then is reduced accessibility to stored material.

In summary, partial information and cuing techniques have provided evidence that a significant part of the amnesic memory disorder is a dysfunction of retrieval processes. That these methods have been found to be differentially effective in increasing amnesic recall to a level similar to that of normals suggests that the effect of the cerebral pathology is to increase the interference normally noted after a time lapse, thereby altering control of stored information and decreasing accessibility to it. Using similar cuing techniques it should be possible to determine whether the effect of ECT administration on recall is comparable to that of cerebral disorganization in other amnesias. In other words, one can investigate whether or not the increased post-ECT forgetting is also a result of an increase in interference, shown as a decrease in accessibility to stored material.

Forgetting after Electroconvulsive Therapy

The purpose of this present study is to investigate the post-ECT recall deficit with a view to attributing the increased forgetting to either a failure of retention or to a failure of retrieval. The use of a cued recall procedure should enable the subject to retrieve any material that has been retained. The literature previously reviewed with regard to cuing included both experiments carried out with normal subjects, many of them university students, and experiments carried out with patients with disordered memory (excluding post-ECT amnesics). The latter group is probably less homogenous than the former, with the root of the amnesia, though always organic, quite varied. Some examples

of associated neurological disorders are right temporal lobectomy, alcoholic Korsakoff psychosis, post-encephalitis (Warrington & Weiskrantz, 1974), and chronic alcoholism (Buschke & Fuld, 1974). What all the subjects had in common was a severe memory loss as assessed by clinical methods, unaccompanied by any appreciable deterioration of general intellectual functioning.

However, across all these dissimilar groups of subjects cuing has been noted to increase the amount recalled, showing that in some cases more is retained than is actually demonstrated under conventional test conditions. Therefore it has been concluded that the forgetting over time that has been observed in normals, and the increase in forgetting that has been noted in amnesics, may be to a large extent explained in terms of a failure of retrieval processes, resulting from interference occurring between perception and retrieval. Inasmuch as increased forgetting has been likewise noted in the post-ECT amnesic syndrome, use of an effective cuing technique should allow for an explanation of this deficit either in terms of unavailability of material, or retention failure, or in terms of inaccessibility to retained material, or retrieval failure. In other words, if ECT administration results in cerebral disorganization which increases the interference and inaccessibility normally seen after a time delay, as has been shown with other amnesic disorders, then the use of cues at recall should eliminate that forgetting by providing access to higher-order units of stored material. If, on the other hand, ECT administration results in increased loss of stored material

from memory, cues will certainly have no effect. In other words, effective recall cuing provides a means whereby the two alternatives of unavailability and inaccessibility can be weighed in terms of their influence in the post-ECT amnesic disorder.

In order to evaluate the effect of cuing on post-ECT forgetting, three variables have to be manipulated. First of all, category names serving as cues must either be present at the time of recall (CR subjects) or be absent at the time of recall (NR subjects). Second, amnesia must be absent (pre-ECT condition) or present (post-ECT condition). Third, since the effect of ECT on forgetting is being investigated, there must be two recall periods per subject (R1 and R2) in each condition, separated by a fixed time interval. Forgetting can then be measured as the difference in recall between these two periods, or R1 - R2. However, due to the possibility of variability of the R1 baselines between conditions, such a measure cannot be taken as necessarily reflecting the totality of what has been acquired, and forgetting might be better assessed in terms of a proportion of that baseline. Thus, the proportion of forgetting relative to what has been demonstrably learned can be measured by the following equation:

$$\% \text{ forgetting} = \frac{R1 - R2}{R1} \times 100, \quad \ldots\ldots\ldots\ldots\ldots\ldots \quad \text{(eq. 1)}$$

where R1 and R2 are here the number of words correctly recalled at the first and second recall periods respectively.

Predictions

ECT is predicted to increase forgetting (Cronholm, 1969; Fromholt et al., 1973; Squire & Miller, 1974). Thus, it is expected that word and category recall will be higher before ECT than after, so that the main effect of ECT administration should be significant. The interaction of the time delay and ECT administration should also be significant: The difference between the amount recalled in the first and second recall periods (R1 - R2) will be higher after ECT than before, as will be the percent forgetting measure. Also, of course, recall is expected generally to decline over time (R1 > R2) due to forgetting (Tulving & Pearlstone, 1966; Weiskrantz & Warrington, 1970).

However, the important comparisons are those that will allow this expected forgetting to be explained as either a loss of retention or a loss of retrieval. These comparisons will all involve the variable of cuing. To clarify matters, it may be useful to look at what each of the alternatives, loss of available material versus loss of access to available material, would predict as an account of the failure of recall after ECT.

Loss of retention. It is predicted that ECT increases forgetting by accelerating the loss of material from storage over time. Thus, presentation of cues at recall would be expected to have no differential effect over free recall, as the cues cannot provide access to material that is not available. The main effect of cuing as well as the double and triple interactions of this variable with time delay and ECT administration should have an insignificant effect upon the recall of words,

categories, and words per category, and upon the percent forgetting scores (Cronholm & Ottosson, 1961; Dornbush et al., 1971; Fromholt et al., 1973).

Loss of retrieval. (1) The main prediction is that ECT administration increases the inaccessibility of material that normally occurs over a time lapse. By providing accessibility to retained material, cuing should produce a differential effect on post-ECT scores, resulting in less forgetting being shown after ECT with cued than with free recall; the difference in the decrease in recall over time between cued and free recall should be greater in the post-ECT condition than in the pre-ECT condition. In terms of the number of words and the number of categories recalled, the triple interaction of ECT administration, time delay, and cuing should be significant: Post(NR.R1 - NR.R2) > pre(NR.R1 - NR.R2) > post(CR.R1 - CR.R2) > pre(CR.R1 - CR.R2), where pre and post refer to before and after ECT conditions, CR and NR refer to cued and noncued recall, and R1 and R2 refer to the two recall trials in each condition.

The differential effect of cuing would also mean that the interaction between cuing and ECT with the percent forgetting scores as the dependent measures would also be significant. The difference between the amount forgotten in the cued and noncued conditions should be greater after ECT than before: Post(NR - CR) > pre(NR - CR), where NR and CR refer to percent forgetting in the noncued and cued conditions, respectively. Also, since material should be generally less accessible after ECT in all recall periods, provision of cues should overcome this inaccessibility, bringing post-ECT levels of word and category recall closer

to the pre-ECT levels. In other words, the two-way interaction of cuing by ECT, in terms of the dependent measures of number of words and number of categories recalled, should be significant: Post[(CR.R1 + CR.R2) - (NR.R1 + NR.R2)] > pre[(CR.R1 + CR.R2) - (NR.R1 + NR.R2)].

(2) Cuing is expected to result in significantly higher word and category recall than the free recall procedure, as cues should provide accessibility to material that would otherwise not be retrieved (CR > NR) (Crouse, 1968; Freund & Underwood, 1970; Loess & Harris, 1968; Warrington & Weiskrantz, 1971). However, words within category recall should not be affected by the cuing procedure, since cues are effective by providing access to higher-order units (Tulving & Pearlstone, 1966).

(3) Since the function of the cues is to provide access to material that would otherwise become inaccessible over time, the interaction of cuing with the delay period should be significant (Tulving & Pearlstone, 1966). In terms of both words and categories, decrease in recall over time is expected to be significantly greater for noncued than for cued subjects (NR.R1 - NR.R2 > CR.R1 - CR.R2). Likewise, percent forgetting scores should be smaller for cued than for noncued subjects. Thus, the main effect of cuing should be significant for the dependent measure of percent forgetting (NR>CR).

In the process of testing these hypotheses, it was felt that a further comparison might help determine the limits of recall cuing for post-ECT subjects. Thus, information was gathered to assess the effects on recall of the provision of cues after a period of free recall. This

extra recall (ER) condition applied only to noncued subjects in the second recall (R2) period. Such a variation of cuing technique has been used previously (Allen, 1969; Freund & Underwood, 1970), and in some cases it has been found to be more effective than cuing without the benefit of prior free recall (Loess & Harris, 1968).

Method

Design

A three-factor mixed design was used, with repeated measures on two factors. The between-subjects variable was cuing; the effects of cuing at retrieval were tested by supplying half the subjects with category names at the time of recall (CR subjects), and requiring that the other half recall without the aid of cues (NR subjects). Taxonomic category names were used as retrieval cues for two reasons. First, taxonomic cuing has been used effectively to increase recall of retained material (Baddeley & Warrington, 1973; Crouse, 1968; Tulving & Osler, 1968; Warrington & Weiskrantz, 1974). Second, there are readily available norms for taxonomic categorization (Battig & Montague, 1969). To ensure that the cues selected would be effective, they were presented to the subjects at the time of learning, along with blocked category items. This procedure should allow for optimal organization to facilitate recall cuing, and the recall cues themselves should be related to the subjects' organization of the list (Allen, 1969).

There were two within-subjects variables. The first was the presence or absence of amnesia, with ECT being the amnesic agent. Parallel forms of the experimental procedure were administered to subjects prior to the first electroconvulsive treatment (pre-ECT condition) and following a set number of treatments (post-ECT condition). The number of ECT treatments intervening between the conditions was usually five, but in one case it was six, and in three cases it was four. It would appear that the

amnesia is established by the fourth to the sixth treatments (Cohen et al., 1968; Fromholt et al., 1973; Miller, 1970).

The second within-subjects variable was the time delay between the two recall periods, the first recall test being administered two minutes after the learning task (R1), and the second three hours after the learning task (R2). The timing of the recall trials allows for comparison of two measures from long-term memory, with an intervening time delay. The design of the experiment is shown in Figure 1.

Subjects

The subjects were selected from the inpatient psychiatric population of three St. John's hospitals. For all those selected, a series of bilateral ECT treatments had been prescribed, which served as the amnesic agent in this experiment. Six subjects in each group had never received ECT before, whereas the others had received treatment ranging from two months to twenty years prior to the current series.

A total of 20 subjects were used in the experiment, 10 in the cued and 10 in the noncued condition. The cued group was composed of six males and four females, ranging in age from 28 to 61 years, with a mean age of 41.7 years. The noncued group included five males and five females, ranging in age from 17 to 57 years, with a mean age of 38.6 years. The *t* score for the difference between these means is 0.564,

	Cued Recall (CR) Condition		Noncued Recall (NR) Condition	
	Pre-ECT	Post-ECT	Pre-ECT	Post-ECT
First Recall (R1)	N=10	N=10	N=10	N=10
Second Recall (R2)	N=10	N=10	N=10	N=10

FIGURE 1. Outline of the experimental design. The between-subjects comparison is cued versus noncued recall. The within-subjects comparisons are pre-ECT recall versus post-ECT recall, and recall after two minutes versus recall after three hours.

which is not significant. The groups of available subjects were thus matched closely on the variables of age, sex, and previous exposure to electroconvulsive therapy.

All subjects were administered the Associate Learning subtest of the Wechsler Memory Scale (WMS), to check for initial differences between the two groups. The items on this subtest were derived from the paired-associate list originally used by Wechsler (1945) to assess the retention deficit in Korsakoff patients; any learning or retention difficulty should be shown by this test.

Materials and Apparatus

The stimulus material consisted of two lists of one and two syllable words from the Battig and Montague (1969) norms. Each list consisted of five taxonomic categories, with three instances of each category, for a total list length of 15 words. List A consisted of 10 one-syllable and five two-syllable words, while List B consisted of nine one-syllable and six two-syllable words. Across Lists A and B, categories were matched for the number of instances generated freely by the normative sample. Equivalent categories were chosen such that the number of instances given in the categories were as nearly the same as possible. Words within the equivalent categories were matched for position in frequency of recall; the three words in each category were chosen such that their frequency rankings were the same as those in the equivalent category. Information

about the two lists is presented in Appendix A.

Both lists, along with category names and the instructions to the subject, were recorded at the rate of one second per word with a five second interval between categories, by means of a Sony cassette tape recorder #33052. Record sheets for the Associate Learning subtest of the WMS and for the subjects' recall of Lists A and B (Appendix B) were made.

ECT Administration

Approximately one-half hour prior to the treatment, all subjects were administered Atropine, and, in some cases, a barbiturate or Valium to induce relaxation. At the time of treatment, patients received Succinyl Choline, a short-acting muscle relaxant, and Sodium Pentothal, an anesthetic. Oxygen was administered immediately before and after the shock.

Electrode placement was bitemporal. Two different methods of ECT administration were used; some doctors preferred the 'glissando' method, whereby there is a gradual increase in current until the seizure is noted, then a gradual reduction; others preferred the delivery of a steady current until the seizure was noted to occur. Two models of the ECT machine were employed, one an Ectonustim, serial #517M, and the other an Ectron, serial #R3867. There was no evidence of differences in subsequent performance related to type of apparatus and administration.

Experimental Procedure

Pre-ECT condition. Within 48 hours prior to the first ECT treatment of the present series, each subject who agreed to cooperate in an experiment on memory was given the Associate Learning subtest of the WMS (Wechsler, 1945). The first learning trial was then administered on an individual basis.

The following taped instructions were given to all subjects:

"I want to see how well you can remember words. I am going to read several groups of three words each. After I have read the words in the group, I want you to say the words you heard, so I'll know you are hearing them all correctly. Listen carefully, and be sure to repeat the words you hear, because later you will be asked to tell me what the words you heard were. Here are some examples:

KINDS OF FISH

tuna
cod
shark

BOYS' NAMES

Paul
Harry
Mike

Do you have any questions about what you are supposed to do?"

Half of the subjects were then presented with List A, and the other half with List B, at the rate of one second per word, with five seconds between categories. Each entire list was presented twice to increase the probability of learning.

Recall 1 (R1). After a period of two minutes, which was spent conversing with the examiner, the subjects were placed in either a cued

recall condition (CR) or a noncued recall condition (NR). Half of the subjects who had learned List A were placed in each condition; likewise for those who had learned List B. The CR subjects were then given the category names in a predetermined random order, which was the same for all CR conditions, and asked to say all the words they could recall as each category was named. One minute per category was allowed for recall, or until 30 seconds had elapsed without a response. The NR subjects were simply asked to say as many words as they could remember from those they had just heard. They were allowed five minutes to recall the words, or until one minute had elapsed with no response. All responses were checked by the experimenter as they were spoken.

Recall 2 (R2). Three hours later, the instructions to recall were repeated. The CR subjects were given the category names in the same order as before and the NR subjects were asked to free recall again.

Extra recall condition (ER). After free recall in the R2 period, the NR subjects were given the category names in the same order as the CR subjects had received them, and were asked to recall as many words as possible that they had heard when each category was named.

Post-ECT condition. Three hours after the fifth (in some cases, the fourth or sixth) ECT, the learning task was repeated, with those subjects previously hearing List A being presented List B, and vice versa. As before, recall testing for both CR and NR groups was carried out two minutes later (R1), and was repeated after three hours (R2), with NR subjects again given cues after free recall in an ER trial.

Results

Background Data

WMS scores. The mean scores on the Associate Learning subtest were 10.4 and 11.8 for the cued and noncued subjects respectively. These values do not differ significantly (t = 0.782, df = 18, $p > .2$).

Comparison of the stimulus lists. In the pre-ECT condition, the mean number of words recalled per subject (R1 and R2 combined) on List A was 14.1 and on List B was 15.0 (t = 0.398, df = 18, $p > .2$). In the post-ECT condition, the mean number of words per subject on List A was 8.3 and on List B was 8.2. The difference is not significant (t = 0.038, df = 18, $p > .2$).

Experimental Data

Analyses were carried out using the number of words recalled, the number of categories from which at least one word was recalled, and the number of words per category recalled as dependent measures. Analyses of the percent forgetting scores were also done, with the proportion of decrease in words recalled from the first to the second recall trial as the dependent variable.

Word recall. The total number of words correctly recalled by each subject in each experimental condition was determined. The comparison of the means for these conditions is shown graphically in Figure 2.

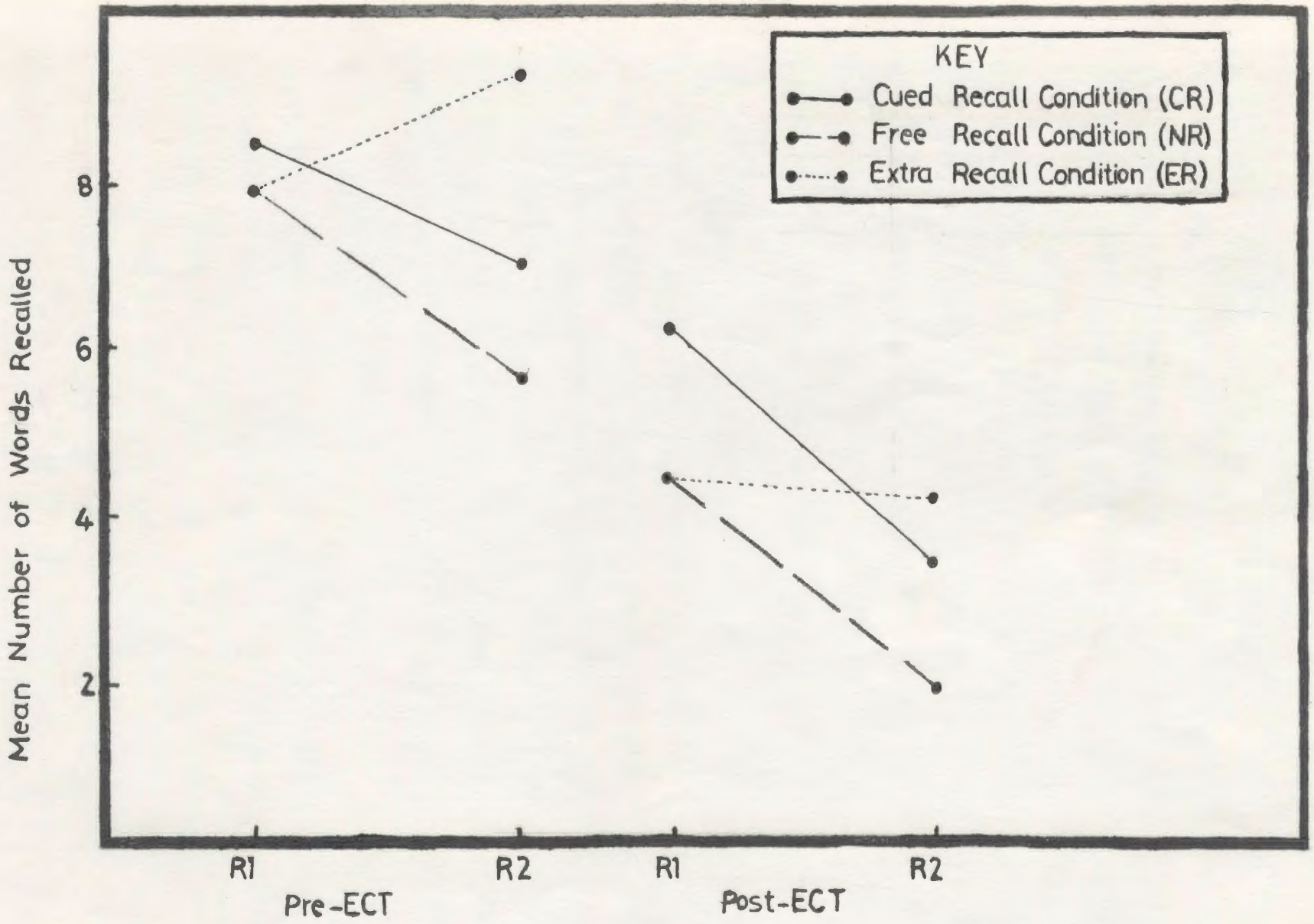

FIGURE 2. The mean number of words recalled in each experimental condition.

The data were submitted to an analysis of variance with cued versus noncued recall as the between-subjects factor, and pre-ECT versus post-ECT and R1 versus R2 as the within-subjects factors. The results of this analysis are shown in Table 1. The administration of ECT significantly decreased recall across all conditions, as did the time lapse of three hours. However, there was no interaction between these two factors, suggesting that recall of words over time dropped about the same amount after ECT as it had before. Neither the main effect of cuing nor any of the interactions with this factor were significant. The use of cuing did not result in significantly greater output than did free recall, and it did not differentially affect the decline in recall over time and after ECT.

Figure 2, page 41, also shows the mean number of words recalled in the extra recall (ER) trial, where noncued subjects were given category names after a period of free recall in the R2 condition. With this procedure, performance improved significantly over free recall alone, both in the pre-ECT condition ($\underline{t} = 5.125$, df = 9, $p < .01$) and in the post-ECT condition ($\underline{t} = 3.214$, df = 9, $p < .05$). There was no significant difference in the amount of the improvement in the pre-ECT and the post-ECT conditions ($\underline{t} = 1.565$, df = 9, $p > .2$).

A similar analysis of variance was carried out, but with ER scores substituted for the NR scores in the R2 recall period. The results are shown in Table 2.

TABLE 1

Analysis of Variance of Words Recalled

Source	SS	df	MS	F
Total	928.8	79	-	-
Between Ss	391.3	19	-	-
Cuing (C)	36.45	1	36.45	1.849
$Error_b$	354.85	18	19.714	-
Within Ss	537.5	60	-	-
ECT (E)	198.45	1	198.45	23.159*
Time delay (T)	92.45	1	92.45	53.255*
C x E	1.80	1	1.80	0.210
C x T	0.80	1	0.80	0.461
E x T	3.20	1	3.20	1.050
C x T x E	0.45	1	0.45	0.148
$Error_w$				
E, C x E	154.25	18	8.569	-
T, C x T	31.25	18	1.736	-
E x T, C x T x E	54.85	18	3.047	-

*$p < .01$

TABLE 2

Analysis of Variance of Words Recalled, with Scores on the Extra Cued Recall Test Replacing Free Recall Scores in the Second Recall Trial

Source	SS	df	MS	F
Total	961.99	79	-	-
Between Ss	355.44	19	-	-
Cuing (C)	0.315	1	0.315	0.016
$Error_b$	355.125	18	19.729	-
Within Ss	606.55	60	-	-
ECT (E)	241.515	1	241.515	21.161*
Time delay (T)	9.115	1	9.115	4.193
C x E	7.810	1	7.810	0.684
C x T	32.51	1	32.51	14.954*
E x T	10.51	1	10.51	3.157
C x E x T	0.615	1	0.615	0.186
$Error_w$				
E, C x E	205.425	18	11.413	-
T, C x T	39.125	18	2.174	-
E x T, C x E x T	59.925	18	3.329	-

*$p < .05$

Again, the administration of ECT significantly decreased recall of words across all conditions. The cuing procedure did not produce significantly greater recall, and the main effect of time delay was not significant; however, the interaction of cuing and time delay was significant, suggesting that presentation of cues after a period of free recall resulted in the demonstration of significantly less forgetting over a three hour lapse than was found in the cued recall condition. Other data in this comparison added nothing to the previous analysis.

Category recall. The total number of words recalled was broken down into its two components, number of categories recalled and mean number of words per category. Tulving and Pearlstone (1966) suggested that accessibility of higher-order units depends in part on appropriate retrieval cues whereas accessibility of items within the higher-order units is largely independent of these cues. Category recall should provide a more sensitive measure of cue effectiveness, as the recall of any element from the category demonstrates that the cue has accessed the higher-order unit. In order, then, to determine the effects of the experimental procedure on the number of categories (higher-order units) from which words were recalled, an analysis of variance was performed. The dependent variable for each subject in each condition was the number of categories from which at least one word was recalled. Table 3 presents the results, and the comparison of the means for all conditions is shown in Figure 3.

All three main effects, but none of the interactions were significant. In other words, presentation of cues resulted in recall of words

TABLE 3

Analysis of Variance of Number of Categories From Which at Least One Word Was Recalled

Source	SS	df	MS	F
Total	169.487	79	-	-
Between Ss	62.737	19	-	-
Cuing (C)	25.312	1	25.312	12.175*
$Error_b$	37.425	18	2.079	-
Within Ss	106.75	60	-	-
ECT (E)	35.112	1	35.112	48.165*
Time delay (T)	17.112	1	17.112	29.251*
C x E	1.013	1	1.013	1.390
C x T	0.613	1	0.613	1.048
E x T	2.813	1	2.813	1.960
C x E x T	0.612	1	0.612	0.426
$Error_w$				
E, C x E	13.125	18	0.729	-
T, C x T	10.525	18	0.585	-
E x T, C x E x T	25.825	18	1.435	-

*$p < .05$

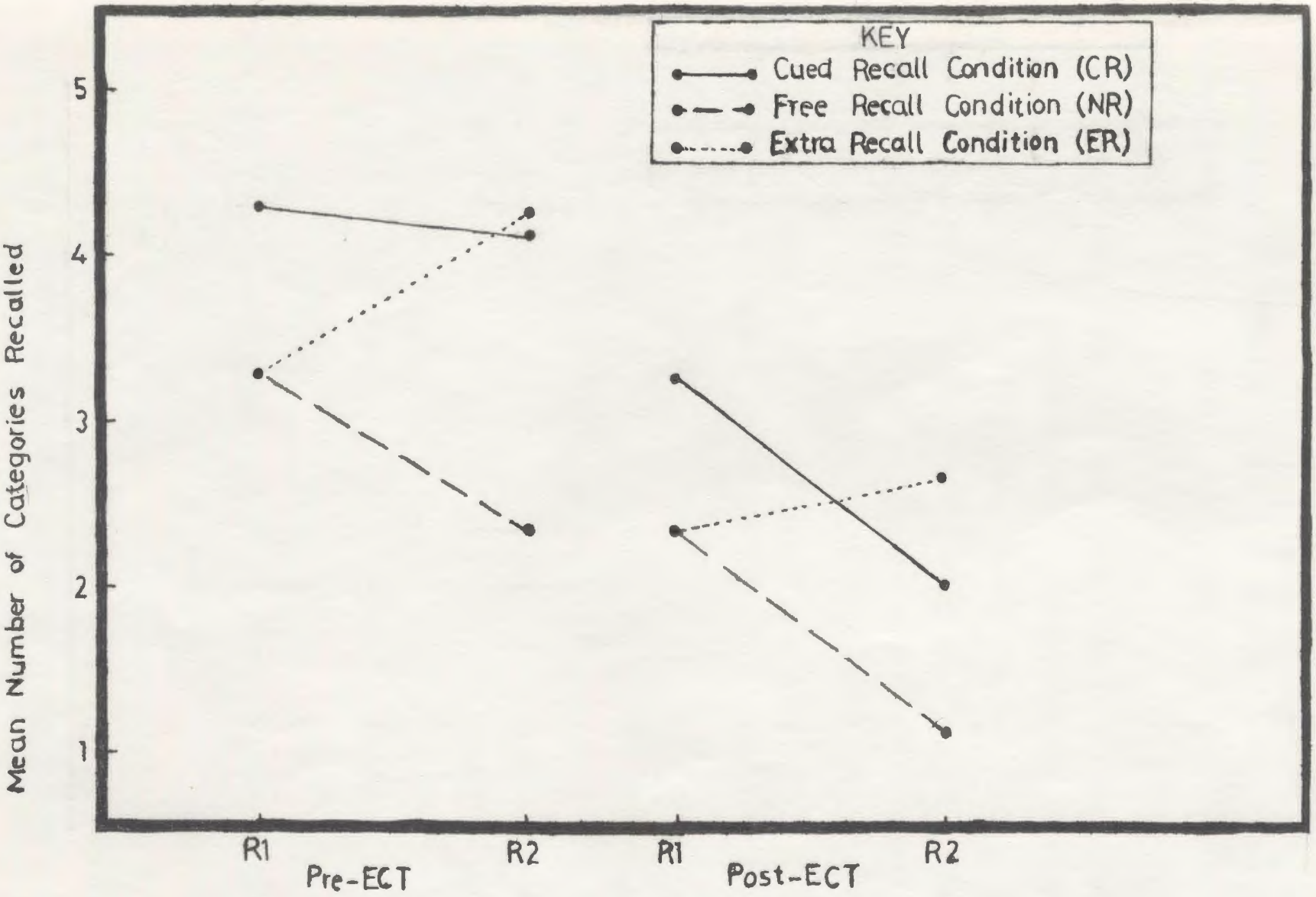

FIGURE 3. The mean number of categories from which at least one word was recalled, in each experimental condition.

from significantly more categories than did free recall, and both ECT administration and the three hour time delay between recall tests had the effect of decreasing the number of categories from which words were recalled. The drop in recall of categories over a three hour period was not significantly different in the pre-ECT and the post-ECT conditions, and cuing did not differentially affect the decrease in recall of categories associated with time delay and ECT administration. However, though category recall generally decreased over time and there were no interactions, a study of Figure 3, page 47, would suggest that time delay did not significantly affect CR subjects in the pre-ECT condition. It would appear that in this specific condition, category recall in R2 was practically as high as it was in R1, which was certainly not so for the other three conditions.

For noncued subjects, the use of an extra recall trial with the benefit of cues in the R2 condition improved recall of categories significantly, both before ECT ($\underline{t} = 6.042$, df = 9, $p < .01$), and after ECT ($\underline{t} = 3.308$, df = 9, $p < .05$). However, there was no significant difference in the degree of this improvement before and after ECT ($\underline{t} = 0.802$, df = 9, $p > .2$). The means for the ER trials are shown in Figure 3, page 47.

Table 4 shows the results of the analysis of variance in the comparison of cuing alone (CR) to cuing after free recall in the R2 period (ER). Here, as well, ECT administration had an overall effect of reducing the number of categories from which words were recalled. The two types of cuing were not significantly different in their overall effect, and, due to the nature of the interactions with the cuing and ECT conditions, recall

TABLE 4

Analysis of Number of Categories Recalled, with Scores of the Extra Cued Recall Test Replacing Free Recall Scores in the Second Recall Trial

Source	SS	df	MS	F
Total	158.387	79	-	-
Between Ss	41.637	19	-	-
Cuing (C)	1.512	1	1.512	0.678
$Error_b$	40.125	18	2.229	-
Within Ss	116.75	60	-	-
ECT (E)	40.612	1	40.612	18.587*
Time delay (T)	0.112	1	0.112	0.183
C x E	0.313	1	0.313	0.143
C x T	9.113	1	9.113	14.866*
E x T	4.513	1	4.513	6.986**
C x E x T	0.112	1	0.112	0.173
$Error_w$				
E, E x C	39.325	18	2.185	-
T, C x T	11.025	18	0.613	-
E x T, C x E x T	11.625	18	0.646	-

*$p < .01$

**$p < .05$

scores did not drop significantly over the three hour interval. Since the cuing by time delay interaction was significant, it would appear that with cuing alone, recall of categories decreased slightly over the three hour period, but with cuing after free recall performance increased from what it had been with no cues three hours earlier (Figure 3, page 47). The time delay by ECT interaction was also significant since there was a slight increase in category recall from R1 to R2 in the pre-ECT condition, but there was a drop in recall in the post-ECT condition.

Words per category recall. The measure of words per category (WPC) recall was obtained by dividing the number of words recalled in a given experimental condition by the number of categories from which words were recalled in that condition, for each subject (Mathews & Tulving, 1973). Table 5 presents the results of the analysis of variance with the same independent variables as in previous analyses, but with WPC as the dependent variable.

The two main effects of ECT administration and time delay significantly decreased the number of words per category recalled, but the main effect of cuing did not quite reach significance. Although Figure 4 would suggest that WPC recall was much lower for the CR than for the NR subjects in the pre-ECT condition, and that cued and noncued recall declined at different rates in the pre-ECT and post-ECT conditions, there is no statistical evidence to support this in terms of the ANOVA as no interactions were significant.

TABLE 5

Analysis of Variance on Words per Category Recalled

Source	SS	df	MS	F
Total	45.536	79	-	-
Between Ss	11.528	19	-	-
Cuing (C)	1.659	1	1.659	3.027
$Error_b$	9.869	18	0.548	-
Within Ss	24.008	60	-	-
ECT (E)	6.985	1	6.985	11.899*
Time delay (T)	2.291	1	2.291	8.392*
C x E	0.874	1	0.874	1.489
C x T	0.017	1	0.017	0.062
E x T	0.460	1	0.460	1.108
C x E x T	0.440	1	0.440	1.060
$Error_w$				
E, E x C	10.557	18	0.587	-
T, C x T	4.915	18	0.273	-
E x T, C x T x E	7.469	18	0.415	-

*$p < .01$

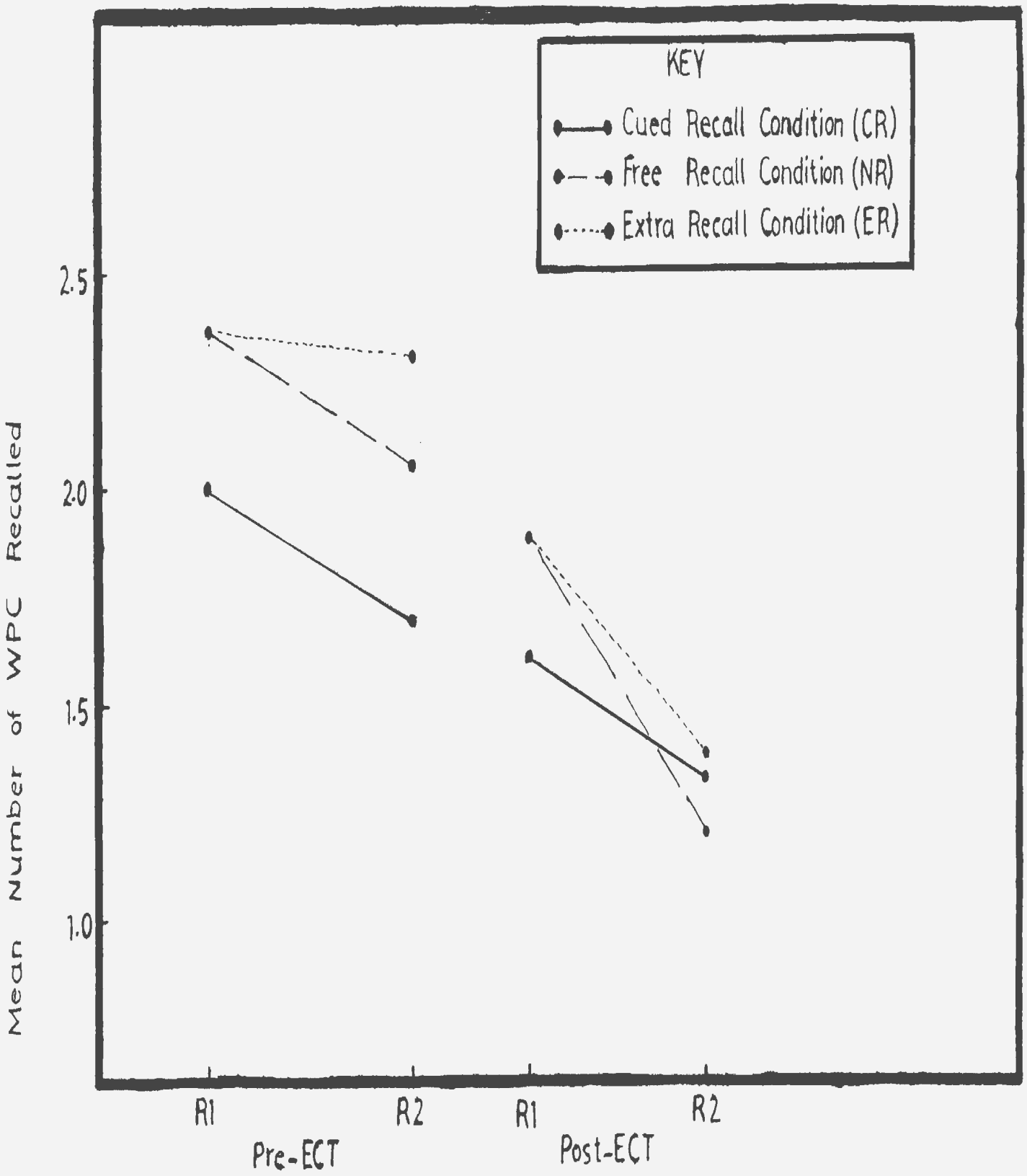

FIGURE 4. The mean number of words per category recalled, in each experimental condition.

As illustrated in Figure 4 on page 52 presentation of cues after a period of free recall had only a very slight effect on WPC recall, relative to noncued recall. Results of the analysis of variance were basically the same as above, with the main effects of ECT and time delay reaching significance (Table 6, page 54).

Percent forgetting scores. The percent forgetting from the first to the second recall period was determined by equation 1, page 28, for all subjects. The comparison of the means is shown in Figure 5, page 55.

Data for the subjects were submitted to an analysis of variance for a two-factor mixed design, with cuing as the between-subjects variable and ECT administration as the within-subjects variable. Results are shown in Table 7, page 56. It can be seen that the administration of ECT significantly increased the degree of forgetting as so defined, but cues had no effect whatsoever on the forgetting.

As suggested by Figure 5, page 55, the use of an extra cued recall trial after free recall significantly decreased the forgetting shown by noncued subjects, both in the pre-ECT condition ($t = 5.35$, $df = 9$, $p < .01$), and in the post-ECT condition ($t = 3.08$, $df = 9$, $p < .05$). Results of the analysis of variance comparing cuing after free recall (ER) to cuing alone (CR) are shown in Table 8, page 57. The variation on the cuing technique resulted in significantly less forgetting over time than standard cuing, but in this comparison, ECT administration did not increase forgetting.

Percent forgetting data were also analyzed in view of possible sex differences. There was no difference found in performance between males and females, either quantitatively or qualitatively.

TABLE 6

Analysis of Words per Category Recalled, with Scores on the Extra Cued Recall Test Replacing Free Recall Scores in the Second Recall Trial

Source	SS	df	MS	F
Total	32.496	79	-	-
Between Ss	11.625	19	-	-
Cuing (C)	1.607	1	1.607	2.885
$Error_b$	10.018	18	0.557	-
Within Ss	20.871	60	-	-
ECT (E)	4.637	1	4.637	13.925*
Time delay (T)	2.353	1	2.353	13.369*
C x E	0.198	1	0.198	0.595
C x T	0.022	1	0.022	0.125
E x T	0.035	1	0.035	0.142
C x E x T	0.031	1	0.031	0.129
$Error_w$				
E, E x C	5.992	18	0.333	-
T, C x T	3.174	18	0.176	-
E x T, C x T x E	4.429	18	0.246	-

*$p < .01$

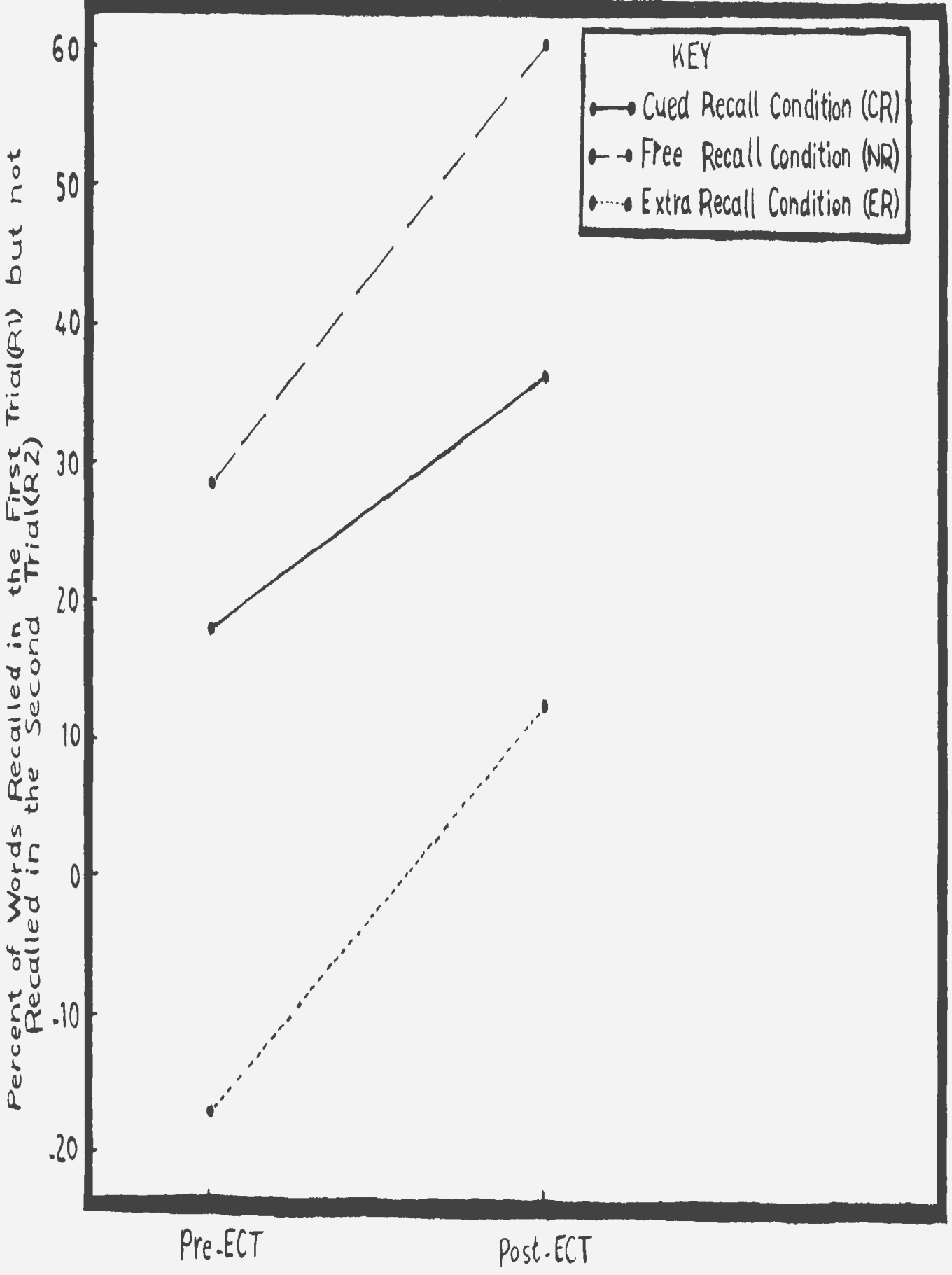

FIGURE 5. The mean percent of words forgotten from R1 to R2, for each experimental condition.

TABLE 7

Analysis of Variance of Percent Forgetting Scores

Source	SS	df	MS	F
Total	52655.79	39	-	-
Between Ss	22630.84	19	-	-
Cuing (C)	2921.76	1	2921.76	2.67
$Error_b$	19709.08	18	1094.95	-
Within Ss	30024.95	20	-	-
ECT (E)	6507.60	1	6507.60	5.11*
E x C	615.92	1	615.92	0.484
$Error_w$	22901.43	18	1272.30	-

*$p < .05$

TABLE 8

Analysis of Percent Forgetting Scores, with Scores on the Extra Cued Recall Test Replacing Free Recall Scores in the Second Recall Trial

Source	SS	df	MS	F
Total	75900.70	39	-	-
Between Ss	39370.88	19	-	-
Cuing (C)	8723.16	1	8723.16	5.12*
$Error_b$	30647.72	18	1702.65	-
Within Ss	36529.20	20	-	-
ECT (E)	5772.00	1	5772.00	3.42
E x C	364.22	1	364.22	0.216
$Error_w$	30393.60	18	1688.53	-

*$p < .05$

Discussion

As predicted, bilateral ECT administration depressed recall significantly, resulting in an anterograde memory deficit as measured by a decrease in recall from pre-ECT levels. This finding is in conformity with results of previous research pointing to the disruptive effect of bitemporal shock on the level of recall (Costello et al., 1970; Fromholt et al., 1973; Squire & Miller, 1974). Post-ECT performance was significantly impaired whether memory loss was measured in terms of absolute number of words recalled (Table 1, page 43), absolute number of categories from which words were recalled (Table 3, page 46), or mean number of words per category recalled (Table 5, page 51), or whether it was measured in terms of proportion of forgetting of that which was originally recalled (Table 7, page 56).

The post-ECT deficit was evident in the initial recall period following ECT (R1). For each group, the R1 level of recall was significantly lower than its equivalent in the pre-ECT conditions (see Figure 2, page 41, Figure 3, page 47, and Figure 4, page 52). General depression of recall by electroconvulsive therapy may suggest a disruptive effect of the stimulation on the acquisition of material (Cohen et al., 1968; Miller, 1970). However, it cannot be assumed that what was recalled at R1 represents the totality of what was learned, since other factors interfering with retention or retrieval phases of memory may have already played a part in diminishing recall. Thus, the performance deficit at R1 may be due to disruption in learning, retention, retrieval, or all of

these. The impairment at R1 cannot be satisfactorily clarified, but its presence does not rule out the existence of a disruption in the recall of what has been demonstrably acquired, which requires a comparison of the R2 levels of recall to those of three hours earlier.

Forgetting and the Post-ECT Deficit

Significant loss of recall over a three hour period was noted to occur, in terms of words (Table 1, page 43), categories (Table 3, page 46), and words per category (Table 5, page 51). That there has been forgetting, then, is obvious. But what is the effect of ECT administration on this forgetting, on the failure to recall this material that was once demonstrably acquired?

Increased percent forgetting scores in the post-ECT tests (Figure 5, page 55) would suggest that after ECT, subjects recalled proportionately less of what they had recalled three hours previously than they did before ECT. This finding supports the hypothesis that forgetting is accelerated after ECT, as has been noted in other amnesic disorders (Warrington & Weiskrantz, 1968; Weiskrantz & Warrington, 1970). However, measurement of words and categories recalled does not support the idea of increased forgetting over time after ECT as there was no interaction between the ECT and time variables (Table 1, page 43 and Table 3, page 46). Whereas proportional decrease in recall was significantly greater after ECT, the absolute decrease in scores from R1 to R2 was approximately the same for

both pre-ECT and post-ECT conditions (see Figure 2, page 41 and Figure 3, page 47). This discrepancy is not surprising in view of the drop in overall levels of recall subsequent to the ECT series, and it may be that, as originally proposed, the relative decline in recall is a better measure of forgetting than the absolute decline. On the other hand, the significant proportional decrease may merely be an artifact of the same low post-ECT R1 baseline that it was supposed to circumvent, since these baselines were so low (a mean of 5.4 words at R1 post-ECT and 8.2 in the pre-ECT condition) that even a slight decrease could be a significant proportion of that initial recall. Because of the conflicting results, no conclusions can be reached on the basis of these data alone.

The words per category data are relevant here in determining if forgetting of acquired material over a three hour time lapse is accelerated after ECT, or if, in fact, the post-ECT recall decrement is no more affected by time than the pre-ECT drop in recall. Table 5 on page 51 indicates that WPC recall dropped significantly over a three hour period. This must be interpreted as a loss of information from storage over time, since items were lost from categories which had been retrieved during the initial test. WPC recall also dropped significantly after ECT (Table 5), indicating that there was a decrease in the amount of material in storage after electroconvulsive therapy. However, though both ECT and time lapse resulted in decreased availability of material, the lowered availability after ECT does not appear to be significantly related to the loss of information from storage over time, since the interaction of ECT administration and time delay did not reach significance. Rather, after

ECT the unavailability was as marked in R1 as it was in R2, both being evidenced by a decrease in words per category recall from the pre-ECT level (Figure 4, page 52). Since it was as noticeable before the time lapse as after, the post-ECT deficit cannot be interpreted as increased loss of retention; rather, the more likely interpretation is in terms of some factor having more or less equal bearing on all post-ECT conditions, likely concerned with an aspect of getting the material into storage.

In summary, the data on the post-ECT forgetting are rather ambiguous. Percent forgetting measures suggest that forgetting does in fact increase after ECT, while measures of absolute decrease in recall over time and of words per category recall suggest that though there is less material available in storage after ECT, this decrement is not significantly time-related. That material is less available after ECT is clear, but it would appear that there is insufficient reason to believe that this is a result of once remembered material becoming less easily remembered over time, as in other post-traumatic amnesias. The generally lowered recall levels after electroconvulsive therapy is more probably a result of decreased post-ECT storage of information.

The Effect of Cuing

It has been seen that in all likelihood, material is less available after ECT than before, and that this post-ECT unavailability does not increase over time, but is present at the initial recall. As there is no proof of accelerated forgetting after ECT, there is no possibility of

attributing the post-shock deficit to a decrease in accessibility of retained material. This being the case, it is not surprising that the main hypothesis of greater efficacy of cuing after ECT than before was not supported, as cuing could naturally not overcome the unavailability of material at recall (Nelson & Brooks, 1974). Neither the cuing by ECT interaction nor the triple interaction between cuing, ECT, and time delay was significant. This held true whether the dependent variable was words recalled (Table 1, page 43), categories recalled (Table 3, page 46), or percent of words forgotten over a three hour interval (Table 7, page 56). Cuing did not assist post-ECT recall any more than it did pre-ECT recall, and produced no more of an effect on the decline of recall over time after ECT than it did prior to the shock. Even if increased post-ECT forgetting had been demonstrated, such results with cuing would still give no evidence that the effect of electroshock in decreasing recall is to increase the disruption of retrieval of intactly stored material, such as that reported to occur normally over time. Results from the extra recall trial also support the conclusion that the post-ECT deficit does not result from an increased inaccessibility of available material. As shown in Figure 2, page 41 and Figure 3, page 47, though ER scores were significantly higher than NR scores, the proportion of increase was approximately the same in the pre-ECT and post-ECT conditions, with no differential effects evident. The findings of Warrington and Weiskrantz (1974) were thus not replicated for this specific amnesia.

If forgetting had been demonstrated to increase after ECT, however, it would have to be considered that a cue may fail to increase recall for

a reason other than the obvious one that the word to be recalled is itself not in storage. When the information needed is not in storage, either because it was lost over time or because it was never there, naturally a cue cannot access it. The other possibility, when post-ECT forgetting is found to increase but where there is no evidence for increased disruption of retrieval is that the cue used was not a part of the stored memory for a word, and thus was not effective in providing accessibility to the retained material. Under these circumstances, before a retrieval-failure hypothesis can be discounted, it must be determined that the cues used were effective in carrying out the function for which they were intended, that function being to provide a means of accessibility to adequately retained material.

Table 3 on page 46 would suggest that the cues used were effective, since in terms of higher-order units or categories, the main effect of cuing was significant, indicating that when cues were given to subjects at recall, they recalled words from significantly more categories than with free recall. Cues would thus seem to have been effective in their control function of providing access to the higher-order unit, and according to the explanation of Tulving and Pearlstone (1966) they should have provided access to material adequately retained. However, unlike the data of Tulving and Pearlstone, category recall was not here accompanied by the expected correlated increase in word recall. Cuing did not significantly increase the total number of words recalled, as is evident in Table 1, page 43, and Table 2, page 44. There is no obvious

methodological reason why this relationship did not occur, and the most likely explanation is that the data on category recall were influenced by subjects' guessing, since category items were fairly high associations to the category names. Thus, increase in category recall with cuing cannot be taken as an unambiguous demonstration that cues were effective in their function.

Another piece of data that might ostensibly indicate that cues were effective in their function of providing access to retained material are the ER scores (Figure 2, page 41 and Figure 3, page 47). Presentation of cues after free recall did lead to recall of words and categories not previously recalled in R2. The improvement in recall suggests that noncued subjects had retained material not previously recalled by conventional means, and the use of cues demonstrated that there had been some difficulty in retrieval of retained material for these subjects.

Though the presentation of cues after free recall did significantly increase subjects' recall, the failure of the cuing by time interaction (Table 1, page 43, and Table 3, page 46) indicates that the standard cuing used here had no effect on the decline in recall over time. Forgetting was as high for cued subjects as it was for noncued subjects, so if we are to believe that the cues were indeed effective in their function, then it must be accepted that subjects' decline in recall over time was not at all affected by retrieval dysfunction. That such a dysfunction does occur normally to some extent has been satisfactorily documented (Crouse, 1968; Tulving & Pearlstone, 1966; Tulving & Thomson, 1973), and

that it does not happen here probably reflects on the failure of the cuing procedure for these subjects. When the ER scores were used in the analysis of variance, the cuing by time interaction was significant, showing that in this condition recall dropped less over time than it did with the standard cuing procedure (see Table 2, page 44, and Table 4, page 49). Similarly, when percent forgetting scores represented the proportion of decline in recall from the R1 (noncued) trial to the R2 (extra recall) trial, the main effect of cuing was significant (Table 8), showing that the ER trial did lead to a closer reinstatement of R1 recall levels than the standard cuing procedure did. However, since they involve comparison of free recall in the R1 period and cued recall in the R2 period, which are basically two different treatments, to cued recall at both R1 and R2, no valid interpretation can be made of these significances. What is important is that the ER scores are not different from the CR scores at R2, and the CR scores are not different from the NR scores. Therefore, all that can really be said about the ER trial is that when subjects were given cues after free recall, their performance level increased significantly over what it had been with no cues in the R2 period.

The ER trial demonstrated that subjects had retained material not previously retrieved, but the evidence is that the cuing procedure used was not effective since it did not show difficulty in retrieval that would be expected over time for all subjects. Therefore, even if increased forgetting after ECT had been shown, the lack of sufficient effectiveness of the cues would have presented a difficulty in the discrimination of the

forgetting as being due either to increased interference with retrieval processes as a result of cerebral disorganization, or to accelerated loss of stored information. Since, however, the presentation of cues in the ER trial did seem to show some efficacy, as instruments to access material not retrieved in the free recall trial, this type of method might be incorporated into future research attempting to assess the post-ECT memory disorder. As an alternative to the method employed here, the comparison could be made between cued recall after free recall and a second free recall after free recall. The results of this comparison could then serve to distinguish a decline in recall over time after ECT as one of failure of retention or failure of retrieval.

Conclusions: The Nature of the Post-ECT Deficit

Though the administration of a series of electroconvulsive treatments resulted in substantially lowered levels of recall in the present subjects, insufficient evidence was found to conclude that subjects did forget learned material faster after ECT than before the treatment, as has been suggested by Cronholm and Ottosson (1961) and by Fromholt et al. (1973). In view of this finding, it would not have been possible to support a retrieval-failure explanation of the post-ECT amnesia, since cue utilization could not possibly succeed in removing the memory impairment caused by unavailability that followed the ECT series. It would appear that the post-ECT defect can be compared neither qualitatively nor quantitatively to that which occurs normally over a time delay or to that

which is associated with other amnesias (Warrington & Weiskrantz, 1974). Though both pre- and post-ECT memory seem to be affected somewhat by unavailability through loss of material as well as by inaccessibility of stored material under normal conditions, decreased recall levels after ECT cannot be fully accounted for as an increase in either of these types of forgetting. Rather, the particular unavailability seen after ECT would seem most likely to result from an increase in difficulty in getting material into storage, since the deficit is evident in the initial post-ECT recall trial and is not affected by time.

That is not to say, of course, that a retention or a retrieval deficit may not also coexist as a part of the anterograde memory dysfunction. It merely suggests that the predominating difficulty, resulting in lowered post-ECT baselines, precludes the demonstration of such a deficit. Use of methods to increase the initial post-ECT baseline, for example, having subjects learn the list to a set criterion, would probably allow the demonstration of a retention or retrieval deficit if such existed. However, such methods would introduce the danger that the resulting differential efficacy of cues may be merely an artifact of the procedure used. This may actually be the case in studies that have shown differential efficacy of recall cuing for amnesics over controls (eg. Warrington & Weiskrantz, 1974), in which event a conclusion of increased blocking of retrieval in the post-traumatic condition would be unwarranted. For the present subjects, the interruption of retention and retrieval processes has a negligible effect on the overall pattern of responses, and it would appear that the effect of the ECT current is to produce an anterograde

amnesia by impairing the process of acquisition, such that the material to be learned does not become properly stored.

Doubts were also cast on the efficacy of the cuing procedure used in that it may have been insufficient to discriminate a retrieval failure from a retention failure even if increased forgetting had been shown. Though an extra cued recall trial for noncued subjects indicated that retained material was not always recalled, use of the cues did not show the disturbance of accessibility over time that has been noted in the general population. If the cues used could not demonstrate inaccessibility of retained material, it could be that we are in fact dealing with a different population of subjects, to whom previous methods and results cannot be generalized. It may well be that taxonomic cuing is not the most effective type for the present group of subjects, though it certainly has been effective in demonstrating retention in normals and in other amnesic conditions; there is no data to confirm or deny that it is likewise effective for psychiatric patients undergoing electroconvulsive treatment. In view of the fact that the subjects performed somewhat differently when tested by cued recall after free recall as opposed to cued recall in all trials, it may be profitable to explore this variation on the cuing method with these particular subjects.

A major source of the failure to demonstrate increased forgetting over time and in the low efficacy of the cuing procedure is the lack of statistical power - a result of too few subjects relative to the large variability of the sampling group. This can be seen by looking at the

large sampling errors shown in the Tables. The only restriction placed on the selection of subjects were those previously listed. Thus, there is the likelihood of the patients used as subjects varying widely along the dimensions of psychiatric disorder, other treatments being received concurrently, motivation for the task, and premorbid intellectual and memory functions. The effects of these interrelated influences on performance in a memory task has not been assessed here, but doubtlessly, such important variables must play a part in the end results. Use of a larger sampling group might help overcome this drawback, as might attempts to restrict the heterogeneity of the sample, and provide greater statistical power.

In conclusion, it must be accepted that for the present sample of psychiatric patients the major effect of cerebral disorganization associated with ECT administration is to generally depress recall levels, so that less material is available after ECT. The low initial baselines would suggest that the post-ECT anterograde amnesia occurs as a result of impairment in the acquisition of material, so that information is not available in storage. Neither disruption of accessibility to adequately stored material, nor the loss of that material itself appear to be a significant part of the memory disorder; failure of retention or retrieval plays such a small part so as to be negligible, in view of the overall decline in recall and the low post-ECT baselines. In this regard, the post-ECT deficit is parallel neither to that normally occurring over a time delay nor to other amnesias.

References

Abrams, R. & Fink, M. Clinical experiences with multiple electroconvulsive treatments. Comprehensive Psychiatry, 1972, 13, 115-21.

Allen, M. Rehearsal strategies and response cueing as determinants of organization in free recall. Journal of Verbal Learning and Verbal Behavior, 1968, 87, 58-63.

Allen, M. Cueing and retrieval in free recall. Journal of Experimental Psychology, 1969, 81, 29-35.

Baddeley, A. & Warrington, E. Memory coding in amnesia. Neuropsychologia, 1973, 11, 159-65.

Battig, W. & Montague, W. Category norms for verbal items in 56 categories: A replication and extension of the Connecticut category norms. Journal of Experimental Psychology, 1969, 80, Monograph Supplement.

Bilodeau, E. & Blick, K. Courses of misrecall over long-term retention intervals as related to strength of pre-experimental habits of word association. Psychological Reports, 1965, 16, Monograph Supplement 6.

Brion, S. Korsakoff's syndrome: Clinico-anatomical and physiopathological considerations. In G. Talland & N. Waugh (Eds.), The pathology of memory. N. Y.:Academic Press, 1969.

Brody, M. Prolonged memory defect following ECT. Journal of Mental Science, 1944, 90, 51.

Buschke, H. & Fuld, P. Evaluating storage, retention, and retrieval in disordered memory and learning. Neurology, 1974, 24, 1019-25.

Cohen, B., Noblin, C., Silverman, A., & Penick, S. On the functional asymmetry of the human brain. Science, 1968, 162, 475-77.

Costello, C., Belton, G., Abra, J., & Dunn, B. The amnesic and therapeutic effects of bilateral and unilateral ECT. British Journal of Psychiatry, 1970, 116, 69-78.

Cronholm, B. Post-ECT amnesias. In G. Talland & N. Waugh (Eds.), The pathology of memory, N. Y.: Academic Press, 1969.

Cronholm, B. & Molander, L. Memory disturbances after electroconvulsive therapy: 5. Conditions one month after a series of treatments. Acta Psychiat. Scand., 1964, 40, 212-16.

Cronholm, B. & Ottosson, J. Memory functions in endogenous depression. Archives of General Psychiatry, 1961, 5, 101-07.

Crouse, J. Storage and retrieval of words in free-recall learning. Journal of Educational Psychology, 1968, 59, 449-51.

Dawson, R. & McGaugh, J. Electroconvulsive shock effects on a reactivated memory trace: Further examination. Science, 1969, 525-27.

D'Elia, G. Unilateral ECT. Acta Psychiat. Scand., 1971, Supplement 215.

Dornbush, R., Abrams, R., & Fink, M. Memory changes after unilateral and bilateral convulsive treatment (ECT). British Journal of Psychiat., 1971, 119, 75.

Ebtinger, R. Aspects psychopathologiques du post-electrochoc. Colmar: Imprimerie Asatie, 1958.

Freund, J. & Underwood, B. Storage and retrieval cues in free recall learning. Journal Experimental Psychol., 1969, 81, 49-53.

Freund, J. & Underwood, B. Restricted associates as cues in free recall. Journal Verbal Learning & Verbal Behavior, 1970, 9, 136.41.

Fromholt, P., Christensen, A., & Stromgren, L. The effects of unilateral and bilateral electroconvulsive therapy on memory. Acta Psychiat. Scand., 1973, 49, 466-78.

Gold, P. & King, R. Retrograde amnesia: Storage failure versus retrieval failure. Psych. Review, 1974, 81, 465-69.

Hicks, R. & Young, R. Input and output variables in the recall of categorized and uncategorized lists. Journal General Psychol., 1973, 89, 109-14.

Hurwitz, T. Electroconvulsive therapy: A review. Comprehensive Psychiat., 1974, 15, 303-14.

Inglis, J. Electrode placement and the effect of ECT on mood and memory in depression. Canadian Psychiat. Assoc. Journal, 1969, 14, 463-71.

Inglis, J. Memory disorder. In C. G. Costello (Ed.), Symptoms of psychopathology. New York: Wiley, 1970.

Janis, I. Psychological effects of electric convulsive treatments. Journal of Nervous and Mental Disorders, 1950, 111, 359.

Jarvik, M. Effects of chemical and physical treatments on learning and memory. Annual Review Psychol., 1972, 23, 457-86.

Kesner, R. & D'Andrae, J. ECT disrupts both information storage and retrieval. Physiology & Behavior, 1971, 7, 73-76.

Kral, V. Memory disorders in old age and senility. In G. Talland & N. Waugh (Eds.), The pathology of memory. New York: Academic Press, 1969.

Levin, M. Toxic psychoses. In S. Arieti (Ed.), American handbook of psychiatry, Vol. 2. New York: Basic Books, 1959.

Lewis, D. Sources of experimental amnesia. Psychol. Review, 1969, 76, 461-72.

Lewis, D., Miller, R., & Misanin, J. Selective amnesia in rats produced by electroconvulsive shock. Journal Comp. & Physiol. Psychol., 1969, 69, 133-35.

Loess, H. & Harris, R. Short-term memory for individual verbal items as a function of method of recall. Journal Exp. Psychol., 1968, 78, 64-69.

Luria, A. Memory disturbances in local brain lesions. Neuropsychologia, 1971, 9, 367-75.

Luttges, M. & McGaugh, J. Permanence of retrograde amnesia produced by electroconvulsive shock. Science, 1967, 156, 408-10.

Mah, C. & Albert, D. Reversal of ECS-induced amnesia by post-ECS injections of amphetamine. Pharmacology, Biochemistry, & Behavior, 1975, 3, 1-5.

Mathews, R. & Tulving, E. Effect of three types of repetition on cued and noncued recall of words. Journal Verb. Learning Verb. Behavior, 1973, 12, 707-21.

Mayer-Gross, W. Retrograde amnesia: Some experiments. Lancet, 1944, 2, 603.

McGaugh, J. Facilitation of memory storage processes. In S. Bogoch (Ed.), The future of the brain sciences. New York: Plenum Press, 1969.

McGaugh, J. & Dawson, R. Modification of memory storage processes. Behav. Science, 1971, 16, 45-63.

McGaugh, J. & Krivanek, J. Strychnine effects on discrimination learning in mice: Effects of dose and time of administration. Physiology & Behavior, 1970, 5, 1437-42.

McGaugh, J. & Landfield, P. Delayed development of amnesia following electroconvulsive shock. Physiology & Behavior, 1970, 5, 1109-14.

Meyer, D. Access to engrams. American Journal Psychol., 1972, 27, 124-33.

Miller, E. Psychological theories of ECT: A review. British Journal of Psychiatry, 1967, 113, 301-11.

Miller, E. The effect of ECT on memory and learning. British Journal of Medical Psychol., 1970, 43, 57-62.

Miller, R. & Springer, A. Implications of recovery from experimental amnesia. Psychol. Review, 1974, 81, 470-73.

Milner, B. Amnesia following operation on the temporal lobes. In C. Whitty & O. Zangwill (Eds.), Amnesia. London: Butterworths, 1966.

Misanin, J., Miller, R., & Lewis, D. Retrograde amnesia produced by electroconvulsive shock after reactivation of a consolidated memory trace. Science, 1968, 160, 554-55.

Nelson, D. & Brooks, D. Retroactive inhibition of rhyme categories in free recall: Inaccessibility and unavailability of information. Journal Exp. Psychol., 1974, 102, 277-83.

Slamecka, N. An examination of trace storage in free recall. Journal Exper. Psychol., 1968, 76, 504-13.

Squire, L. Amnesia for remote events following electroconvulsive therapy. Behavioral Biology, 1974, 2, 119-25.

Squire, L. A stable impairment in remote memory following electroconvulsive therapy. Neuropsychol., 1975, 13, 51-58.

Squire, L. & Miller, P. Diminution of anterograde amnesia following electroconvulsive therapy. British Journal Psychiat., 1974, 125, 490-95.

Squire, L., Slater, P., & Chace, P. Retrograde amnesia: Temporal gradient in very long-term memory following electroconvulsive therapy. Science, 1975, 187, 77-79.

Stones, M. Electroconvulsive treatment and short-term memory. British Journal Psychiat., 1973, 122, 591-94.

Tulving, E. & Madigan, S. Memory and verbal learning. American Review Psychol., 1970, 21, 437-84.

Tulving, E. & Osler, S. Effectiveness of retrieval cues in memory for words. Journal Exper. Psychol., 1968, 77, 593-601.

Tulving, E. & Patterson, R. Functional processes in free recall. Journal Exper. Psychol., 1968, 77, 239-48.

Tulving, E. & Pearlstone, Z. Availability versus accessibility of information in memory for words. Journal Verbal Learning & Verbal Behavior, 1966, 5, 381-91.

Tulving, E. & Thomson, D. Encoding specificity and retrieval processes in episodic memory. Psychol. Review, 1973, 80, 352-73.

Valentine, M., Keddie, K., & Dunne, D. A comparison of techniques in electroconvulsive therapy. British Journal Psychiat., 1968, 114, 989-96.

Warrington, E. & Weiskrantz, L. A study of learning and retention in amnesic patients. Neuropsychol., 1968, 6, 293-91.

Warrington, E. & Weiskrantz, L. New method of testing long-term retention with special reference to amnesic patients. Nature, 1968, 217, 972-74.

Warrington, E. & Weiskrantz, L. Amnesic syndrome: Consolidation or retrieval? Nature, Lond., 1970, 228, 628-30.

Warrington, E. & Weiskrantz, L. Organisational aspects of memory in amnesic patients. Neuropsychol., 1971, 9, 67-73.

Warrington, E. & Weiskrantz, L. The effect of prior learning on subsequent retention in amnesic patients. Neuropsychol., 1974, 12, 419-28.

Wechsler, D. A standardized memory scale for clinical use. Journal of Psychology, 1945, 19, 87-95.

Weiskrantz, L. Experimental studies of amnesia. In C. Whitty & O. Zangwill (Eds.), Amnesia. London: Butterworths, 1966.

Weiskrantz, L. & Warrington, E. Verbal learning and retention by amnesic patients using partial information. Psychonomic Science, 1970, 20, 210-11.

Williams, M. Traumatic retrograde amnesia and normal forgetting. In G. Talland & N. Waugh (Eds.), The pathology of memory. New York: Academic Press, 1969.

Woods, R. & Piercy, M. A similarity between amnesic memory and normal forgetting. Neuropsychol., 1974, 12, 437-45.

Zinkin, S. & Miller, A. Recovery of memory after amnesia induced by electroconvulsive shock. Science, 1967, 155, 102-104.

Zubin, J. & Barrera, S. Effect of ECT on memory. Proc. Soc. Exper. Biol. & Med., 1941, 48, 556.

Appendix A

Below are the word lists used in the memory task. The number in parentheses after the category name refers to the number of different instances generated for that category by the normative sample; the number after the category item refers to the frequency ranking of the item in that category (Battig & Montague, 1969).

LIST A

KINDS OF TOOLS (35)

hammer (1)
wrench (9)
square (15)

KINDS OF TREES (39)

pine (3)
birch (6)
spruce (9)

KINDS OF COLORS (26)

green (3)
yellow (4)
purple (7)

KINDS OF FRUIT (31)

apple (1)
grape (6)
cherry (7)

KINDS OF CLOTHES (37)

blouse (5)
coat (7)
socks (2)

LIST B

KINDS OF FLOWERS (34)

rose (1)
pansy (9)
lilac (15)

KINDS OF ANIMALS (42)

horse (3)
tiger (6)
bear (9)

KINDS OF VEGETABLES (27)

corn (3)
bean (4)
lettuce (7)

KINDS OF INSECTS (32)

fly (1)
beetle (6)
roach (7)

KINDS OF FURNITURE (37)

desk (5)
couch (7)
table (2)

Appendix B(i)

Score sheet for subjects presented List A in the pre-ECT condition and List B in the post-ECT condition. Words were checked by the examiner as they were recalled by the subject.

Subject: _____ Condition: _____

WORD	PRE-ECT		WORD	POST-ECT	
	R1	R2		R1	R2
green			corn		
yellow			bean		
purple			lettuce		
blouse			desk		
coat			couch		
socks			table		
hammer			rose		
wrench			pansy		
square			lilac		
apple			fly		
grape			beetle		
cherry			roach		
pine			horse		
birch			tiger		
spruce			bear		
Total R1			Total R1		
Total R2			Total R2		
Total Pre-ECT			Total Post-ECT		
% Forgetting			% Forgetting		

Appendix B(ii)

Score sheet for subjects presented List B in the pre-ECT condition and List A in the post-ECT condition. Words were checked by the examiner as they were recalled by the subject.

Subject: _____ Condition: _____

WORD	PRE-ECT		WORD	POST-ECT	
	R1	R2		R1	R2
corn			green		
bean			yellow		
lettuce			purple		
desk			blouse		
couch			coat		
table			socks		
rose			hammer		
pansy			wrench		
lilac			square		
fly			apple		
beetle			grape		
roach			cherry		
horse			pine		
tiger			birch		
bear			spruce		
Total R1			Total R1		
Total R2			Total R2		
Total Pre-ECT			Total Post-ECT		
% Forgetting			% Forgetting		