RECOVERY FROM ECS-INDUCED AMNESIA WITH KINESTHETIC FEEDBACK

A Thesis

Presented to

the Faculty of the Department of Psychology

University of Houston

In Partial Fulfillment

of the Requirements for the Degree

Master of Arts

Terry G. Shaw

May, 1975

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ABSTRACT

Rats were used in two experiments where recovery of an otherwise "unretrievable" memory for a passive avoidance step-down task was demonstrated. The results indicate that animals rendered "amnestic" with ECS treatment are capable of reversing that amnesia when kinesthetic feedback is given during retention testing. The second experiment showed that the kinesthetic feedback also increased the latencies of foot shock only animals. Results suggest that recovery of memory from ECS occurs given a step-down "reminder" at the time of testing and that the effects of ECS are probably on the retrieval rather than the storage or consolidation processes.

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A "dual trace" model of memory (e.g. Hebb, 1949) maintains that recently received information is uniquely encoded in short-term memory in the form of electrochemically active, neural reverbratory circuits that are ultimately terminated by decay and or interference processes. The continued action of these circuits is believed to give rise to a more permanent, stable long-term memory of an energetically passive, chemical-structural format.

Support for this model came from early experimental evidence provided by Duncan (1949), Thompson and Dean (1955) Thompson and Pryor (1956) and Thompson (1957). Using electroconvulsive shock (ECS) these investigators were able to demonstrate retrograde amnesia (RA) for previously learned information. Interpretation of these results posited that the amnestic effect was due to a consolidation failure.

The length of time that a memory trace existed in a labile state then became an issue. If consolidation time could be measured, then some clue as to the physical identity of the trace might be found. However the results of subsequent experiments produced ambiguous findings (e.g. Chorover and Schiller, 1965; Kopp, Bohdanecky and Jarvik, 1966). The inconsistency of the results may, in part be a function of task, species, and treatment parameter differences. Nevertheless, the overall disagreement contributed heavily to the formation of other explanations for the RA effect (Coons and Miller, 1960; Lewis and Maher, 1965; Pinel and Cooper, 1966).

Even though these alternative views failed to explain all of the RA phenomena (McGaugh and Madsen, 1964; Herz, 1969; Spevack and Suboski, 1969), they have led to extremely useful experimental techniques (e.g., one-trial tasks for appetitive as well as aversive situations and single ECS treatments).

Amnesia induced by ECS, hypothermia, hypoxia, metabolic inhibitors and other various treatments have been attributed to consolidation failures (Glickman, 1961; McGaugh, 1966; Barondes and Cohen, 1968). And RA does appear to be a product of interference with memory processes. However, as noted in Miller and Springer's review article (1973), disruption of either the consolidation or the retrieval stage will result in apparent memory failure. Therefore the lack of memory on any one retention test does not necessarily support a consolidation failure hypothesis.

Retrieval Failure Hypothesis

Lewis (1969), Miller and Springer (1972a, 1972b), Springer and Miller (1972), Chute and Wright (1973) and many other investigators have recently argued that the observed amnesia might be accountable in terms of the disruption of a memory sequence subsequent to consolidation.

The availability of previously acquired information for short periods following an amnestic treatment (McGaugh and Landfield, 1970; Miller and Springer, 1971; Geller and Jarvik, 1968a) and the apparent spontaneous recovery from amnesia (Zinkin and Miller, 1967; Young and Galluscio, 1971), suggest that the effect of an amnestic treatment may be on retrieval processes.

The assumption in the consolidation model is that disruption of the "active storage phase" will produce an animal devoid of any memory for the previous event, in which case the amnesia would necessarily be permanent. On the other hand, a retrieval failure notion would imply that the information is present but unavailable at the times of testing. Thus, the issue of permanence becomes the primary distinction between the two explanations of retrograde amnesia.

Permanence of Amnesia

The permanence of induced amnesia is typically measured through behavioral analysis and as such, it is poorly suited for determining the existence of a memory trace. Evidence from human and animal studies show that even when simple retention tests give little indication of memory, other tests such as recognition and relearning often reveal considerable savings. Mendosa and Adams (1969) and Hine and Paulino (1970) present evidence that ECS-induced amnesia is effective for only certain types of responses, (e.g. skelatal motor vs. autonomic).

The issue of recovery from amnesia (i.e. permanence) was first raised by Zinkin et al (1967) and later extended by Koppenaal, Jagoda, and Cruce (1967), Geller and Jarvik (1968b), Quartermain, McEwen and Azmitia (1970) and Miller et al (1972a, 1972b). The net result of these studies demonstrated that, given certain "reminder" treatments, recovery from amnesia occurred. However the effects produced by the various reminders were subject to other interpretations (Cherkin, 1972; Gold, McGaugh, Haycock, and Macri, 1973). If these treatments constitute additional training sessions, then recovery may be explained by the "partial consolidation" notion.

The Role of Contextual Cues

The importance of contextual cues to the retention of information is implied by any model of memory retrieval which assumes that all attributes of memory, when aroused, have the capacity to retrieve other attributes. Then potentially any contextual cue may serve as an effective agent of memory retrieval. Underwood (1969) indicated that contextual stimuli may play a role in retention as lower animals are probably quite dependent upon this type of information to identify what must be remembered.

If memory is viewed as a collection of attributes representing the events noticed during learning, then there is reason to expect that "arousal of a sufficient number, proportion or kind of other attributes belonging to the same memory is both necessary and sufficient for retrieval of a particular target behavior" (Spear, 1973). Although refering to simple retention decrements due to forgetting and/or interference, a similar view may also apply to the RA paradigm.

A taxonomy of the various recovery agents used in the retrograde amnesia literature aids in the analysis of the studies reporting recovery. The various reactivation treatments are organized under two general headings; Overt Induced Recovery and Covert Induced Recovery. Overt refers to those "reminders" which are externally supplied by the experimenter whereas covert implies reminder cues supplied by the subject.

Overt-Induced Recovery

Overt treatments refer to those "reminders" which are externally supplied, in known quantities, by the experimenter. This category would include such recovery agents as a weak and/or non-contingent foot shock, injections of psychopharmacological agents, and the readministration of ECS.

Reinforcer Cues as Recovery Agents: When appetitive conditioning is tested, response decrements (warm-up effects) disappear if the organism is re-exposed to the reinforcer associated with original learning (Spear, 1967). Warm up effects may not be typical of the decrements produced by ECS, but a second presentation of the reinforcer has been effective in producing recovery within the RA paradigm (Koppenaal, et al 1967; Geller et al 1968b).

Other investigators were able to attenuate memory loss using a non-contingent foot shock (lewis, Misanin, and Miller, 1968; Quartermain, McEwen and Azmitia, 1970; Miller et al 1972a, 1972b; Young and Fuselier, 1973). Miller, Springer and Vega (1972) have shown that in appetitive tasks, recovery could be accomplished through a second, non-contingent presentation of the appetitive reinforcer.

Readministration of the primary reinforcer appears to be a sufficient recovery agent but "reminder" treatments may not release a memory from an ECS-produced retrieval block. Instead, application of a second foot shock may provide an extra learning experience that adds to the performance of animals who would otherwise have poor retention of avoidance training. Gold et al (1973) present data suggesting such an effect for a second foot shock.

Internal Physiological Cues: Perhaps the critical memory attribute aroused by the reactivation operation, the agent for recovery, is the animals internal physiological response to any severe stress, rather than the specific reinforcer. If physiological consequences of the stress were represented as attributes of the memory their presence during test could serve as agents for recovery even when elicited by quite different aversive stimuli. Springer and Miller (1972) have shown recovery when ice water emersion was the reinforcer and footshock was the recovery agent.

By administering amphetamine, corticosteriods or foot shock, Barondes and Cohen (1968) were able to block the amnestic effect of ECS. One common feature of these treatments is physiological arousal.

Davis, Thomas and Adams (1971) showed that memory could be largely restored with scopolamine injections just prior to testing. Similarly, Duncan and Hunt (1972) were able to reduce the effects of ECS with strychnine.

In a related study, Robbins and Meyer (1970) trained rats on a series of appetitive and aversive discrimination tasks. Interference with memory was seen with subsequent ECS treatment, but the amnesia was restricted to those earlier problems that had been learned under the same motivational state as the problem immediately preceding the ECS.

In conclusion, the evidence implicates internal physiological cues as effective "reminders". However the theoretical constructs representing such underlying processes are too vague for exact manipulation.

ECS Induced State Cues: Internal cues associated with the state of the animal at the time of learning, do have retrieval value. One interpretation is that the use of ECS to produce RA simply functions within a state dependent learning paradigm. At present, there are two lines of evidence dealing with this notion, the first assumes that the ECS treatment has anterograde influences on memory and the second implies that the properties of this treatment primarily involve retrograde effects.

<u>Anterograde Effects as an Induced State Cue</u>: Nielson (1968) and later DeVietti and Larson (1971) and DeVietti and Hopfer (1974a) present evidence suggesting that the interaction of foot shock and ECS serve as an agent to produce a dissociation from the normal brain state that existed during the acquisition/consolidation phase. As the dissociative effects of the treatment wear off with the passage of time, retention is observed, in as much as a 96 hour post-treatment test (enough time to recover) yields some partial return of memory.

Cogent arguments have been raised against the intero-grade dissociative learning interpretation of RA effects on the grounds that it predicts spontaneous recovery and dissociative states lasting up to 96 hours (Luttges and McGaugh, 1967; Miller, Malinouski, Puk and Springer, 1972).

<u>Retrograde Effects as an Induced State Cue</u>: A second approach to the dissociative effects of ECS assumes that the physiological state necessary for effective retrieval is the state present during the consolidation phase of memory. Consequently, the memory is stored in an altered state produced by the amnestic treatment (Thompson and Neely, 1970; Thompson and Grossman, 1972; Wright, Chute and Weber, 1973). By delivering a second ECS just prior to retention testing, amnesia was partially reversed.

There are problems with ECS treatment as a recovery agent due to its debilitative effect on the animals motor performance. Recent work by Manthei, Wright and Kenny (1973) suggests that the effective state produced by ECS may not last longer than 10 or 15 minutes and any retention testing during this period would probably be confounded. Nevertheless, the ECS-induced state of the organism does seem to be a significant attribute of memory capable of attenuating amnestic effects.

Each treatment considered thus far may be said to serve as a significant attribute of memory with the capacity to partially attenuate memory loss. But by their very nature, these types of recovery agents are subject to other interpretations (McGaugh and Herz, 1972; Gold et al, 1973). If a distinction between the consolidation and retrieval failure view is to be clearly and unambigiously made, then recovery must occur as a function of the endogenous cues supplied by the subject (e.g. covert-induced recovery).

Covert-Induced Recovery

Covert induced recovery deals with those reactivation treatments whose exact values are generally unknown and are endogenous cues supplied by the subject. Examples of this type of recovery treatment would include pretraining exposure in the form of multiple, non-reinforced test trials or simple placement in the test chamber prior to retention testing. Kinesthetic cues associated with the task as well as other kinds of sensory information might also be categorized under this heading.

<u>Apparatus Cues as Agents for Recovery</u>: Pre-treatment exposure to the apparatus (familiarization/habituation) appears to be a common feature in many of the studies reporting recovery; presumably the net effect is an increase in the probability of arousing a sufficient number or kind of memory attributes. Familiarization may be analogous to stacking the "deck" in favor of recovery. More likely, exposure is a necessary component of the "optimal conditions" for recovery.

Keppel (1972) discusses evidence that retention of verbal materials may decline as a consequence of changes in surprisingly mundane features of the environment, such as the nature of the experimental room and the color of the ink used to print the words. Although the response decrements in Keppel's study were not due to an amnestic agent, clearly the passive features of the external environment defined in the context of learning make themselves evident as attributes of memory.

Several other studies have found related results within the ECS-induced amnesia paradigm. Essentially any study reporting spontaneous recovery from amnesia is subject to the interpretation that the presence of apparatus cues is sufficient to induce recovery (Zinkin et al, 1968; Quartermain et al, 1970; Young et al, 1971). Also, within the "implicit reactivation" paradigm (see Spear, 1973), it is assumed that mere re-exposure to the apparatus is sufficient to activate a memory (Lewis, Bregman and Mahan, 1972).

Quartermain et al (1970) report results which showed recovery from amnesia with a foot shock. The interesting aspect of this study is that foot shock alone was ineffective unless preceded by exposure to the apparatus (a retention test). The authors indicate that, although the first test had not been considered by others to be a crucial factor in reactivating a memory it was in fact included in the experimental procedures of two other reports of memory recovery after a "reminder" foot shock, (Koppenaal, et al 1967; Geller et al.1968).

Other investigators have observed that simple re-exposure to the apparatus was sufficient in producing recovery (DeVietti and Hopfer, 1974b; Sara, 1973). Azmitia, McEwen and Quartermain (1972) found that simply allowing the animal to recover in the apparatus after the administration of ECS proved to be sufficient in reversing the usual amnestic effect. Moreover, Miller, Springer and Vega (1972) present data showing that memories may be recovered with brief exposure to the punishment compartment or somewhat longer exposure to the start compartment. However, the data suggested that overexposure to either compartment not only failed to further improve retention but could actually impair it. Seemingly, a restored memory is vulnerable to extinction like any other memory.

The evidence overwhelmingly implicates the apparatus as a valuable source of cues that serve as potent attributes of a memory. The fact that simple re-exposure to the apparatus, in the absence of any other treatment, is effective in alleviating amnesia supports a retrieval failure interpretation of RA.

<u>Kinesthetic Cues as Agents for Recovery</u>: Kinesthetic feedback could serve as an important attribute of memory for some tasks. As Boring (1950) stated "the dog who remembers a location by pointing. . . carries some of his memory in his posture." Similarly, Piaget (1970) suggested that young children carry memory in their actions. Rand and Wapner (1967) found a retention deficit in humans when the subjects' physical position during learning differed from that during the retention test (standing versus laying down).

Isolated conditions for accessing the effect of kinesthetic cues on retrieval do not at present exist within the RA literature. Problems are inherent with the availability of kinesthetic information at the time of testing because the subject must first produce the response used to access memory loss before the cue is present.

To determine the potency of kinesthetic cues as attributes capable of an "amnestic" memory reversal, the motor information must be made available without producing the target behavior. By converting a single platform to a double platform in a passive avoidance step-down task, the subject should have access to kinesthetic feedback prior to reaching the grid. If kinesthetic cues are effective recovery agents then one would expect to see a typical "amnestic" effect for the first step-down response followed by longer latencies to a second response. If RA is truly a retrieval problem, considerable recovery should be evident.

EXPERIMENT 1

METHOD

Subjects: Ninety Holtzman strain rats between 100 and 120 gm. in weight were housed 2 per cage and allowed free access to water and lab chow. Upon arrival, all animals were anesthethitized with ether and implanted with stainless steel "pigtail" electrodes inserted immediately behind the ears.

Apparatus: The training-testing apparatus is similar to one first described by Jarvik and Essman (1960). This version of the apparatus consisted of a 40 cm square compartment with 30 cm high walls made of translucent plexiglas. The floor was constructed of .64 cm dia. stainless steel rods placed 1.90 cm apart. Located in the center of the compartment was an electrically insulated double platform (Fig. 1) consisting of a 11 cm dia. top platform which stood 5.5 cm above the second platform (22 cm dia.). The second platform was 5.5 cm above the grid. Both platforms operated independently and were removable. For the single platform condition, another platform, the size of the first (11 cm dia.) was constructed to stand 5.5 cm above the grid.

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Insert Figure # 1 here

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The grid was connected to a Lafayette Operant Control system set to deliver a 200 V at 2.0 ma foot shock (FS) for a duration of 2 seconds. Following the offset of the foot shock, some S's were given an Electroconvulsive Shock (ECS).

A Lehigh Lectronics electro shock therapy unit was set to deliver a 200 V, 200 msec. charge through alligator clips attached to the pigtail electrodes. All animals displayed full clonic-tonic convulsions after an ECS treatment.

Group 1 and 2: Subjects in these groups were trained and tested using the single platform (SP). Group 1 animals (FS+ ECS, SP) received foot shock and ECS on training day while Group 2 subjects were given no foot shock followed by no ECS (NFS+NECS, SP).

Group 3, 4 and 5: These groups were trained and tested using the double platform (DP). Group 3 S's were given foot shock upon completion of the double step-down response and returned to their home cages (FS+NECS, DP), Group 4 animals received ECS 2 sec. after stepping off the second platform and returned to their cages (NFS+ECS,DP) and Group 5 S's were removed from the grid 2 sec. after responding without the administration of any treatment (NFS+NECS,DP).

Training for all other groups consisted of foot shock followed by ECS. Application of differential recovery treatments made up the remaining conditions.

Group 6 and 7: In an attempt to isolate the effects of kinesthetic and apparatus feedback, these S's were given only one source of information on test day. Group 6 animals were trained and tested onthe double platform (FS+ECS,DP) and thus received kinesthetic cues on test day.

The rats in Group 7 were trained and tested using the single platform (FS+ECS,A,SP), but were placed on the grid for a 30 sec. "exposure" period just 5 minutes prior to testing, providing Apparatus cues (A).

Group 8 and 9: Animals in Group 8 were trained and tested on the double platform (Kinesthetic Cue) and were given the apparatus "exposure" prior to retention testing (FS+ECS,A,DP). Group 9 S's were treated in the same manner as Group 8 except they were tested 96 hours after training (96) in an effort to provide the "internal state" cues associated with ECS amnesia, (FS+ECS,A,S,DP). The various treatment conditions are shown in Table 1.

RESULTS

Training day latency scores were analyzed with a Kruskal-Wallis One-Way ANOVA, with Groups as the factor. The results indicated no significant differences between groups implying equality across samples ($H=1.36$, $p>.05$).

Test day scores were separated into latencies to the first step-down response and total latency scores. For Groups 1,2,and 7, the latency to the first step-down response was the same as the total latency score. For each subject tested on the double platform, the total latency score on test day was corrected by subtracting the amount of time spent on the second platform during training.

Test day latencies to the first step-down response (Figure 2) were analyzed with a Kruskal Wallis One-Way ANOVA using Groups as the factor. A significant difference between groups ($H=16.12$, $p<.05$) indicated the need for inter group comparisons. Since there are problems with using multiple paried comparison tests, further analysis was attempted only when observed differences were large enough to indicate the need for individual testing.

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Insert Figure 2 Here

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For the first platform latencies shown in Figure 2, there were only small observed differences between the groups except for the foot shock only animals (FS+NECS,DP), and they differed significantly from every other group.

Comparison of the foot shock only group (FS+NECS,DP), with the foot shock group given ECS and tested on the single platform (FS+ECS,SP) yielded a significant difference ($U=13, p<.01$). This FS+ECS,SP group in turn did not differ from the non-treated controls (NFS+NECS,SP or NFS+NECS,DP), suggesting a rather large amnestic effect. The same result occured when the FS+NECS,DP Group was compared with the FS+ECS animals tested onthe double platform (FS+ECS,DP). Since all FS+ECS groups performed essentially the same as the NFS+NECS animals, no more comparisons were made on the first step-down latency scores.

Group performance on the total latency scores are presented in Figure 3. A Kruskal Wallis ANOVA indicated overall group differences ($H=20.63$, $p<.01$). However, upon subsequent analysis, no significant differences between the foot shock only animals (FS+NECS,DP) and the foot shock plus ECS S's given only the Kinesthetic cue (FS+ECS,DP) were evident ($U=33$, $p>.05$). This implies that reversal from an "amnestic effect" did occur and more importantly, that recovery was due to kinesthetic feedback provided by the first step-down response. Further

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Insert Figure 3 Here

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analysis showed that the FS+ECS,DP animals (Kinesthetic Cue) were significantly different from the non-treated controls (NFS+NECS, SP and NFS+NECS,DP) as well as having significantly longer latencies ($U=23$, $p<.05$) than the

FS+ECS group tested on the single platform (FS+ECS,SP). Only small observed differences were evident between the rest of the groups and analysis of the data was halted.

DISCUSSION

Kinesthetic feedback supplied by the first step-down response significantly alters behavior on the second platform such that the total latency scores of the group given only kinesthetic feedback (FS+ECS,DP) are statistically indistinguishable from the group given only foot shock (FS+NECS,DP). However, this recovery was not evident on the first step-down response in which the FS+ECS,DP animals showed the typical "amnestic effect". These findings lend much support to the idea that recovery is possible under appropriate conditions, and that the "amnesia" evidenced on the first response represents a true retrieval failure.

When S's are given exposure to the apparatus (Apparatus Cues, prior to retention testing their performance is apparently unaltered. The lack of significant recovery due to apparatus exposure (A) is supported by the small latency scores seen in the animals who were given only the apparatus cue (FS+ECS,A,SP). This treatment seems to reverse the effect of the kinesthetic information as inddcated by the poor retention of the animals treated with both retrieval cues (FS+ECS,DP,A). Exposure to the apparatus may also account for the small latencies of the animals tested at 96 hours (FS+ECS,DP,A-96).

Although Sara (1973) reported recovery with exposure to the apparatus, her results are actually based on the use of two test trials. How exposure by means of an extra testing session is still unresolved. Perhaps direct placement has a stronger extinguishing effect, or perhaps 30 seconds allowed for "overexposure" as indicated by Miller, Springer and Vega, (1972).

EXPERIMENT 2

In a second experiment, we decided to take a closer look at the "reversal" effect of kinesthetic information. Rather than concern ourselves with the interactions of multiple cues all other recovery agents were eliminated from the study.

Subjects: Fifty Sprague Dawley rats (300-350 gm) previously used in an appetative task were housed and implanted as before.

Apparatus: The same apparatus was used except that the single platform was present on training day for all subjects and the foot shock parameters were changed to 250 V at 2.0 ma. All other treatment parameters and criterion were the same.

Procedure: On the first day of the experiment, the animals were brought to the experimental chamber and given 2 minutes of handling followed by 1 minute of exposure to the apparatus with the electrodes attached. The same procedure was repeated on the second day of the experiment. On Day 3, the subjects were randomly divided into 5 equal groups of 10 S's each and differential treatments were administered. All groups were tested 24 hours later.

Group 1 and 2: These S's were given foot shock upon completion of the single step-down response and immediately returned to their home cage. Group 1 (FS,SP) was tested

24 hours later with the single platform in place and Group 2 (FS,DP) with the double platform.

Group 3 and 4: After responding these animals received foot shock followed immediately by ECS. Group 3 S's (FS+ECS, SP) were tested on the single platform and Group 4 animals (FS+ECS,DP) were tested with the double platform in position.

Group 5: To serve as a baseline control from which to correct the double platform scores, these animals were given no treatment on the single platform and tested on the double platform (NFS,NECS,DP). All latencies were automatically recorded as in experiment 1.

RESULTS

Acquisition latencies were gathered and analyzed by means of a Kruskal Wallis One-Way ANOVA with Groups being the factor. The results indicate that all groups had essentially the same latencies on training day ($H=1.19$, $p > .05$).

Latency data from retention testing was separated into the response time on platform 1 (first step-down response) and the total platform latencies for the groups tested on the double platform. Each double platform latency was corrected by subtracting the mean latency on platform two of the untreated controls (NFS+NECS,DP) from every response time.

Using the latencies to the first step down response, a Krusdal Wallis test yielded significant differences between groups ($H=17.28$, $p < .01$) as shown in Figure 4. Independent Mann-Whitner U tests showed significant differences between the foot shock only (FS) and the FS+ECS animals for both the single and double platforms (U=13, U=11 respectively, $p < .01$). There were no significant differences between the FS+ECS,SP and FS+ECS,DP S's when compared to the non treated controls (NFS+NECS,DP), suggesting a rather large "amnestic" effect.

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Insert Figure 4 Here

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When the total platform data was subjected to the same analysis, the Kruskal Wallis was again significant (H=26.36, $p < .001$) for an overall group difference. Independent Mann-Whitney U tests verified the overall effect due to ECS but also pointed out significant differences due to the platform type (i.e. single vs. double). As Figure 5 shows, there was a significant difference between the FS,SP and the FS,DP groups (U=26, $p < .05$) as well as for the FS+ECS,SP snd FS+ECS,DP groups (U=24, $p < .05$). The only non-significant difference was between the "amnestic control group" tested on the single platform (FS+ECS,SP) and the non-treated controls (NFS+NECS,DP) as indicated by the analysis on the first step-down response latencies.

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Insert Figure 5 Here

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The "amnestic" effect was again reversed when kinesthetic feedback was made available prior to emitting the target behavior. Furthermore, the observed recovery occured only when the animals were allowed the motor cue associated with the first step-down response.

DISCUSSION

Kinesthetic cues at the time of testing are effective in retrieving an otherwise "amnestic" memory, for a step-down task. Furthermore, the recovery doesn't occur until after the S has already produced the first step-down response. This is supported by the fact that first step down latencies for the FS+ECS animals tested on the double platform (FS+ECS,DP) are indistinguishable from the non treated controls (NFS+NECS,DP) and the FS+ECS group tested on the single platform (FS+ECS,SP). However, the FS+ECS,DP group shows significant increases in response latencies on the total platform scores. Also, the effect of kinesthetic feedback appears to be effective in increasing latencies whether the memory is "amnestic" or not, as evidenced by the performance of the FS,DP group who avoided significantly longer than their single platform counterparts (FS,SP).

The recovery effect of kinesthetic information appears to be a generalized phenomenon as 9 of the 10 S's in Group 4 and 6 of the Group 2 sample had higher second platform scores. Two more animals in Group 2 might have produced the same result but their tiime on the first platform limited the amount they could spend on the second platform before reaching criterion.

In conclusion, the permanence of RA is the primary issue at hand. If recovery from apparent "amnesia" can be demonstrated, then the consolidation failure position as well as the notion of a liable, perseverative first stage of memory looses much support.

Recovery from ECS-induced amnesia has been shown to occur (Lewis, 1969; Miller et al; 1972a, 1972b; Springer et al, 1972; Chute et al, 1973), but these results must be carefully interpreted. It is possible that the recovery agents used in these experiments provided extra learning trials (Gold et al, 1973). At least these examples of recovery are subject to other interpretations. However, as experiment 1 and 2 demonstrate, reversal of an otherwise "amnestic" memory was accompolished through the use of a recovery agent not succeptable to the interpretations proposed by Gold and his associates.

Cherkin (1972) proposed another way to save the consolidation hypothesis from the "recovery" data. Cherkin argues that recovery will occur when moderate amnestic agents are used and that stronger amnestic treatments fail to yield a recovery of memory. At present, there are no guidelines for specifying "strong" amnestic treatments, however, using essentially the same ECS intensity that Cherkin used for "strong ECS" we were able to obtain recovery.

Finally, when complete recovery occurs, as it did in experiment 1 and with other studies, there is no basis for denial of the retrieval failure notion. Partial recovery, implies the lack of sufficient number, kind, or proportion of attributes or irreversable damage due to ECS. In any case, the significant return of an "amnestic" memory can only support a retrieval failure interpretation of retrograde amnesia.

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Table 1

Treatment Conditions

Experiment 1

GROUP	FS	ECS	PLATFORM TYPE	APP. CUES	KIN. CUES	TEST TIME
1. FS+ECS,SP	x	x	SP			24
2. NFS+NECS,SP			SP			24
3. FS+NECS,DP	x		DP			24
4. NFS+ECS,DP		x	DP			24
5. NFS+NECS,DP			DP			24
6. FS+ECS,DP	x	x	DP		x	24
7. FS+ECS,A,SP	x	x	SP	x		24
8. FS+ECS,A,DP	x	x	DP	x	x	24
9. FS+ECS,A,S,DP	x	x	DP	x	x	96

KEY: FS or NFS indicates the presence or absence of foot shock during training; ECS or NECS indicates the presence or absence of ECS; SP means that the animals were trained and tested on the single platform; DP denotes the use of the double platform (Kinesthetic Cue); A indicates the Apparatue Cue and S means the Apparatus cue was applied at 96 hours post-training.

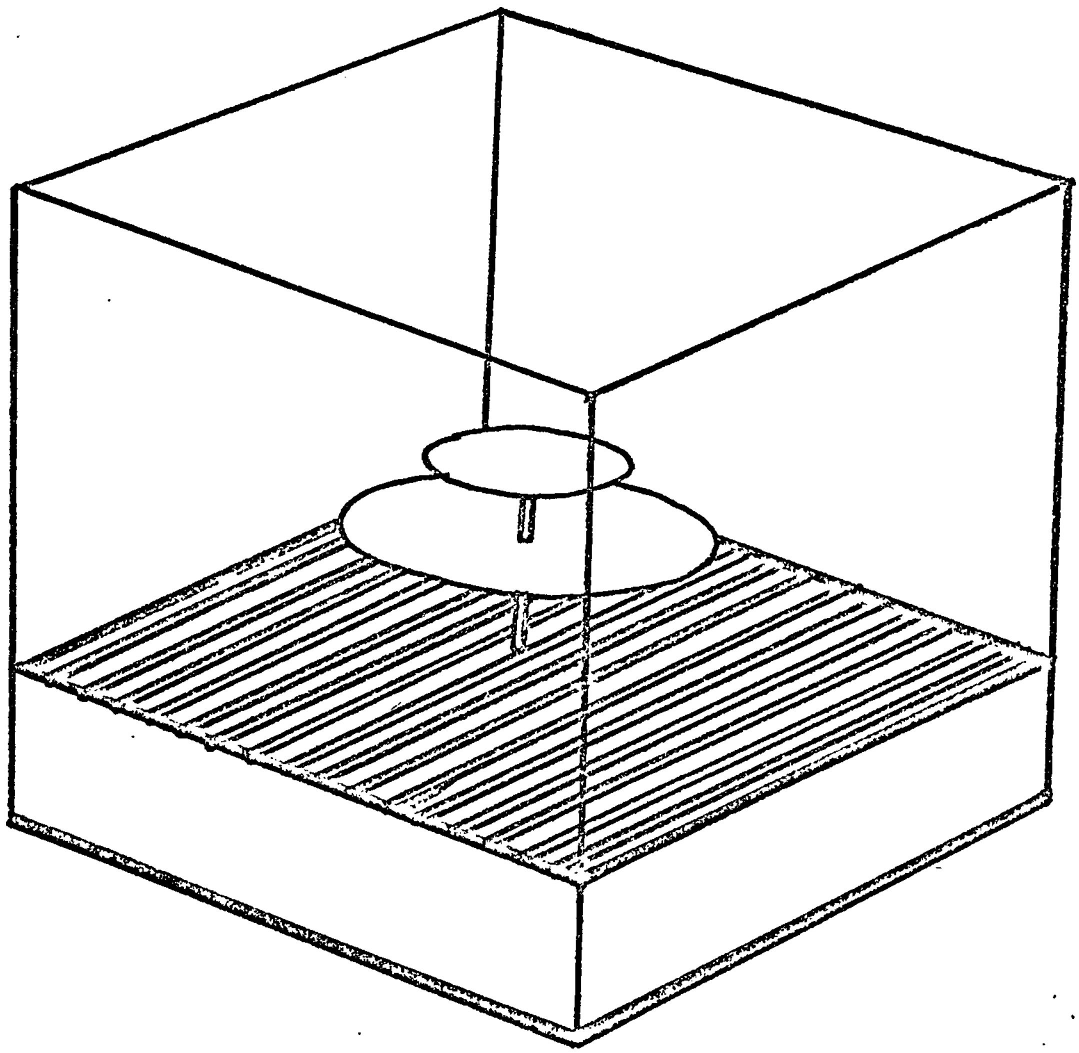

Fig. 1. Illustration of the experimental chamber used in Experiment 1 and 2. (The double platform is shown in position).

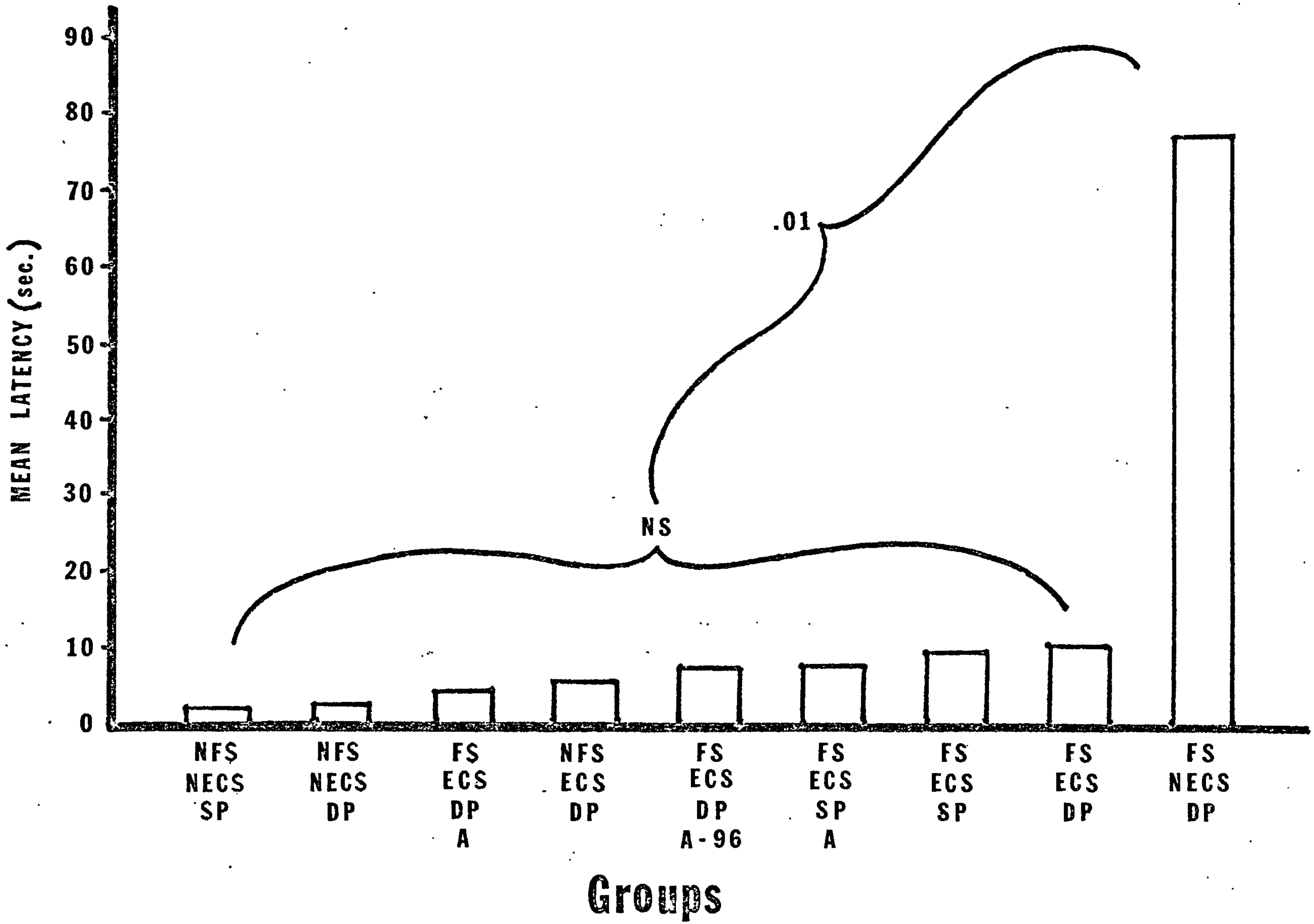

Fig. 2. Mean latency scores to the first step-down response in Experiment 1.

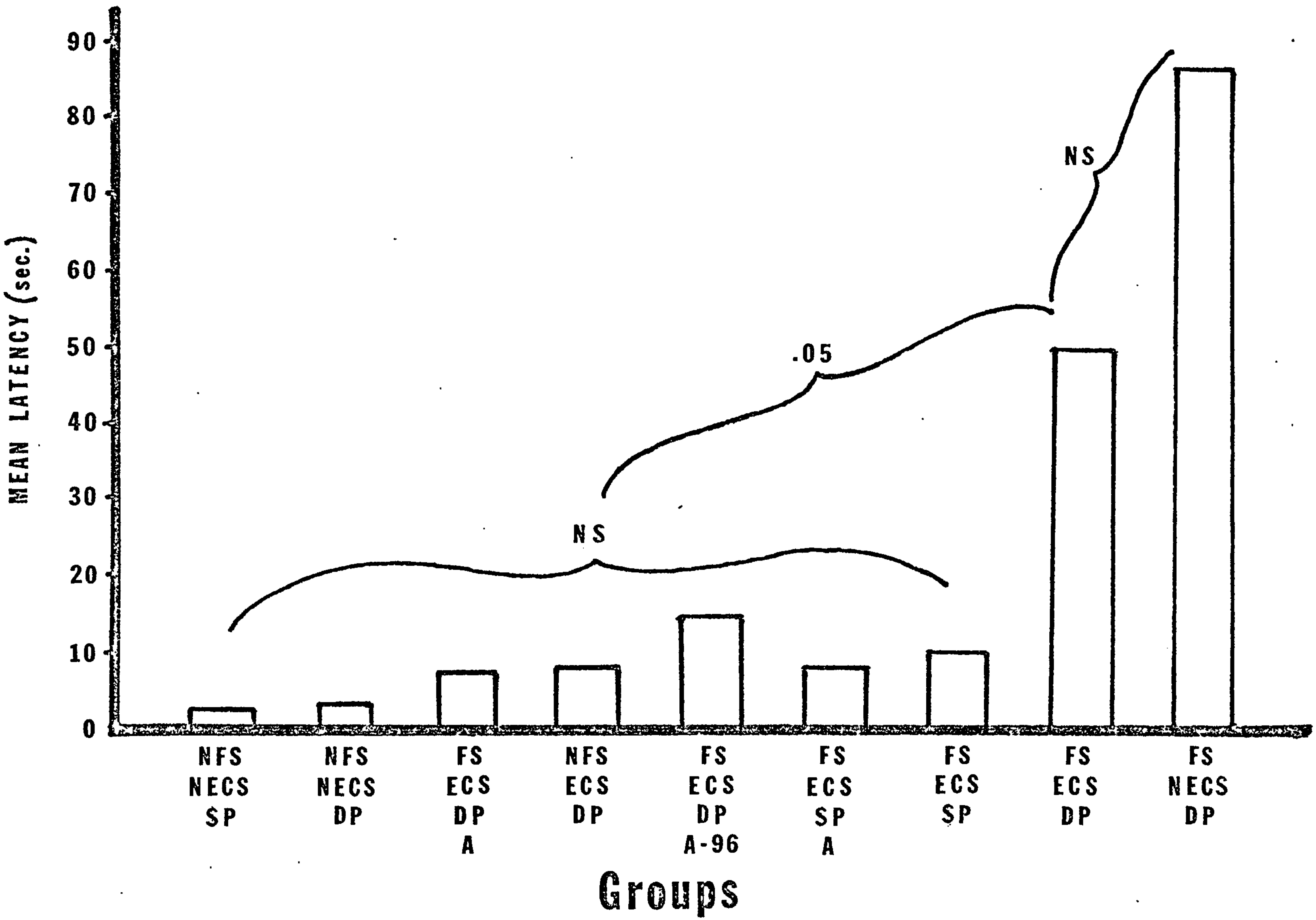

Fig. 3. Mean "Total" latency scores for each group after correction of double platform response time, from Experiment 1.

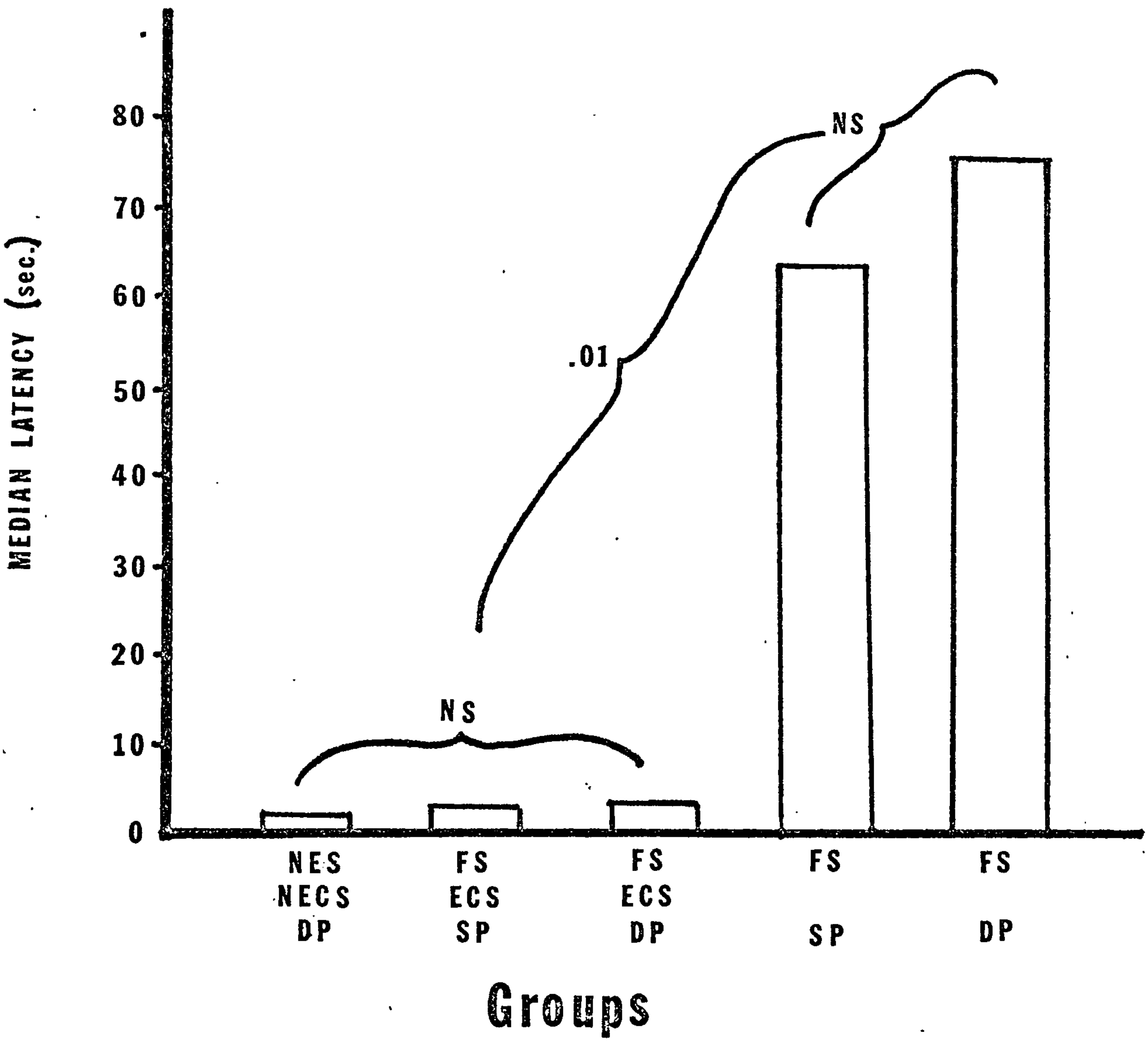

Fig 4. Median response latencies to the first step-down for the groups in Experiment 2.

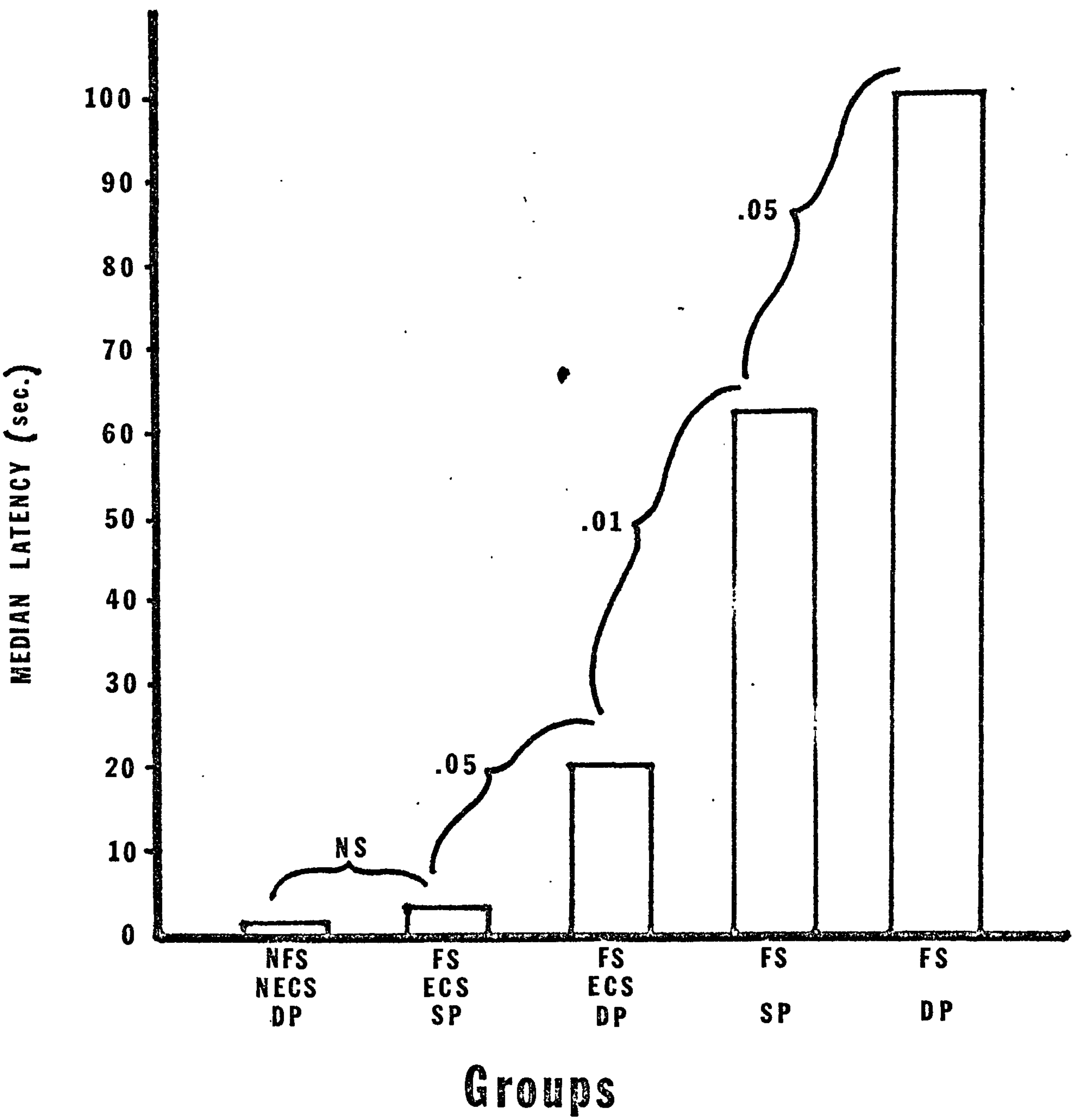

Fig. 5. Group medians for the total latency scores in Experiment 2. Double platform scores have been corrected.