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Differential assessment of retrograde amnesia produced by Hypothermia following one-trial avoidance conditioning

Duane E. Brookhart

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DIFFERENTIAL ASSESSMENT OF RETROGRADE AMNESIA PRODUCED
BY HYPOTHERMIA FOLLOWING
ONE-TRIAL AVOIDANCE CONDITIONING

BY

DUANE E. BROOKHART

A Thesis Submitted in Partial Fulfillment of the
Requirements for the Degree of Master of Arts in
Psychology in the Graduate School of the
University of Richmond

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ACCEPTANCE

This thesis has been accepted in partial fulfillment of the requirements for the Degree of Master of Arts in Psychology in the Graduate School of the University of Richmond.

July 11, 1973
Date

Frederick J. Kazub
Chairman

Edward H. Miller
Member

James Tromater
Member

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ABSTRACT

Two different measures of the retention of avoidance conditioning, frequently used in the investigation of retrograde amnesia (RA), were examined. Rats were given a single training trial on either passive or active avoidance in a conventional black-white discrimination apparatus and step-through latencies were recorded. Task sequence, footshock and hypothermia were experimentally varied across eight groups of subjects. Each S was returned to the experimental apparatus 24 hours after training and given retention tests on both passive and active avoidance tasks. Test trial latencies failed to demonstrate any retention deficits attributable to RA, and both passive and active avoidance test latencies significantly increased. The results were interpreted in terms of the suppression of behavior resulting from conditioned emotionality, and serious questions were raised concerning the validity of single-trial avoidance latency measures.

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Theories of perseverating neural processes were first introduced during the initial decade of the Twentieth Century to account for certain newly discovered phenomena of learning and forgetting. The emerging theoretical position concerning neural perseveration postulated the continuation of neural activity associated with learning beyond the specific learning situation. The first clear statement on neural perseveration theory is attributed to Muller and Pilzecker (1900). They interpreted the impaired retention of a learned list of nonsense syllables, following the learning of a second similar list, as resultant of "retrograde inhibition" produced by the disruptive effects of second list learning on perseverating neural processes associated with initial list learning. Although a good descriptive example is offered, Muller and Pilzecker refrain from any postulation about the relationships among learning, perseveration and retention.

McDougall (1901), in extension of Muller and Pilzecker, suggested that neural perseveration may provide an explanation of retrograde amnesia (RA) following cerebral trauma. Burnham (1903) espoused a similar position concerning the relationship of neural perseveration and cerebral trauma-induced RA, and further hypothesized the existence of certain physiological processes which account for a memory becoming part of

a permanent store. Burnham's most provocative contention, introducing the consolidation theme which has continued through the present literature, was that these physiological processes may require a considerable amount of time to effect completion.

DeCamp (1915) offered a perceptive example of neurological speculation:

From the neurological standpoint, in the learning of a series of syllables, we may assume that a certain group of synapses, nerve cells, nerve paths, centres, etc., are involved. Immediately after the learning process the after-discharge continues for a short time, tending to set associations between just learned syllables. Any mental activity engaged in during this after-discharge, involving or partially involving the same neurological group, tends, more or less, to block the after-discharge, and give rise to retroactive inhibition (p. 68).

Technical limitations prevented such hypotheses from being tested for more than four decades. The most notable addition to perseveration and consolidation theory during this era was the Reverberation Hypothesis offered by Lorente de No (1938). At the molecular level, Lorente de No posited that reverberating neural activity maintained a newly acquired memory in an integrated, though disruptable form, until permanent fixation of the information had been completed. This analysis provided significant explicative value concerning the retroactive susceptibility of information to various dis-

ruptive agents. The concept was popularly received, and was subsequently adopted and supported by numerous researchers (Hebb, 1949; Young, 1953; Gerard, 1955). Gerard (1955) expressed what has come to be accepted as the classical position on the perseveration-interference-fixation inter-relationships: any method which effectively lowers neural thresholds thereby decreases interference, and resultingly reduces the time required to accomplish fixation. The reverse of this statement has similarly received extensive support in the literature (Gerard, 1955; Glickman, 1961).

It was not until 1957 that Stellar was able to conclude that a substantial quantity of neurophysiological data had been accumulated to support the existence of system within the brain accounting for the permanent fixation of memory. Burns (1954, 1958) isolated small slabs of cortex from the remainder of the brains of cats while leaving the blood supply systems substantially intact. Electrical stimulation of these slabs was found to produce ongoing neural activity for as much as thirty minutes after stimulation. Burns also found that these continuing chains of neural activity could be effectively blocked by re-introducing electrical stimulation. Such bursts of activity are apparently due, in part to differential rates of depolarization within various

segments of individual neurons.

Milner and Penfield (1955) reported cases of temporal lobe ablation in humans which resulted in severe inability to acquire new material post-operatively. Scoville and Milner (1957) reported similar results, and additionally stated that preoperative retention remained substantially undisturbed: critical brain structures responsible for this function could not yet be identified, yet it did seem that a mass action hypothesis was untenable, and that moreover the amygdala and hippocampus were directly involved in the storage process. Continuing in this line of research, Brady, Schreiber, Geller, and Kling (1954) reported the failure of amygdalized cats to acquire an avoidance response or retain a preoperatively learned response. Such anatomical data suggest the existence of several potentially requisite neural pathways involved in memory storage or retrieval. Much of the recent investigation of the neuronal basis of learning has been directed at examining the role of acetylcholine (Bennett, Diamond, Krech, and Rosenzweig, 1964), ribose nucleic acids (Hyden, 1961; Gaito, 1963), and glial cells (Galambos, 1961; Egyhazi and Hyden, 1961). Unfortunately the conclusions offered from these orientations are both tenuous and highly contested at present.

The consolidation hypothesis makes no attempt to specify or analyze the molecular physiological changes resulting in consolidation. The actual mechanisms whereby a "memory trace" (whatever this may be) is stored are unknown. Hyden (1961) suggested that repetition of neural impulses resulted in differential stability of certain proteins within the RNA molecule; Gaito (1963) proposed a similar system involving DNA. Rosenzweig, Krech and Bennett (1960) correlated learning with Acetylcholine E concentrations in the brain. Hebb (1949), Eccles (1957, 1964) and others have postulated swelling of presynaptic terminal knobs, growth of new presynaptic terminals, and other comparable processes. Both the complexity of the learning phenomenon and the potential variety of physiological changes seem to indicate that a complete understanding of the physiology of learning and retention is not possible at present.

The Reverberating Circuit hypothesis offered a heuristic starting point; however, additional concepts were found essential in delineating the relationships among learning, perseveration, and retention. This void was most eloquently filled through the development of consolidation theory, which was initially, though crudely, offered by Burnham (1904). The major tenets of consolidation theory offer a good introduction into the direction of pertinent research over the last

thirty years.

Neural consolidation is viewed as synonymous with memory fixation. When the neural impulses associated with learning achieve a state of stabilization, memory is then fixed and no longer subject to the effects of external perturbations. Disruption of this fixation process before stabilization will result in retention deficits.

A second consolidation concept is that of the time-bound property of fixation. Fixation is a dynamic process which operates over time. This implies that the condition of memory traces progresses from an initially vulnerable labile phase to a stabilized state reflecting permanent memory establishment. Estimates of the duration of the labile phase have ranged from several seconds (Chorover and Schiller, 1965) to as much as several days (Pearlman, Sharpless and Jarvik, 1961). Numerous factors, including task, training, familiarity, subject idiosyncrasy, and variety of noxious agent, all are pertinent to the duration of the interval, but clear relationships have not yet been elucidated.

An additional property of consolidation, though not universally accepted, is the relative permanence of disruption during the labile phase. Deutsch and Deutsch (1966) reported that memory deficits, though substantial, could be effectively eliminated through the use of a second learning

trial also followed by administration of an amnesic agent.

It is a quite popular contemporary view to conceptualize memory as being composed of several stages, or phases. The most popular discrimination cites two stages, - an early labile phase during which interference is possible, and a later stable, permanent phase during which memory is impervious to extraneous insult (John, 1967). Barondes and Cohen (1966) outline three stages of consolidation and state that these stages may exist consecutively or concurrently. Lewis (1969) emphasized that what is important about these hypothesized stages and functions is that they all refer to input processes, and implicate fixation as an input effect. In terms of brain function and theoretical lines, retention deficits could be produced at any of several points.

Retention could be affected at the input, or registration level. Interference during registration could prevent whatever brain functions underlie the conversion of immediate information into a permanently retrievable form. Perturbations that increase neural thresholds, impede the normal rate of neural activity, or mask the quality or quantity of information at initial registration would seriously disturb retention.

Secondly, there exists the possibility that registration may be successfully accomplished, but for a period of time,

fixation may be impermanent and resultingly susceptible to interference. This is a popular consolidation position. But there is more to memory than the fixation of a trace, either through one or several stages. It is conceivable that memories are rapidly and permanently fixed, and that interference disrupts the storage system following fixation. The brain may possibly be prevented from properly organizing and integrating the retrieval process.

There are other systems still farther along the memory sequence, past input, fixation, and storage, that may be similarly vital to retention. Dissociation is one such mechanism which reflects interference with retrieval. The organism may be able to retrieve the information quite readily, but he may fail to associate the memory within its original context: memory does not occur when it should, or the organism may have lost the motivation to express the memory. Other possibilities such as suppression, competition or inhibition may become relevant long after the conclusion of the general consolidation model.

It is unfortunate that so much emphasis has been placed on input chains: other points offer potential seats of disruption in the memory sequence. Without denying the very real possibility that it is at the point of fixation that at least some disruptions occur, increasing evidence suggests

some interference in memory is due to other causes than simply failure to consolidate.

The impaired retention of responses learned prior to the introduction of a traumatic agent is commonly referred to as retrograde amnesia (RA). A substantial arena for the laboratory examination of RA was provided by the introduction of electroshock therapy (EST) in 1937. Early observations indicated a substantial post-shock amnesia which eventually subsided to a shorter RA effect. Zubin and Barrera (1941) were the first to systematically investigate the phenomenon. Ten patients were trained in a series of paired-associate lists to a criterion of two consecutive errorless repetitions. With no intervening shock between learning and retention trials, significant savings resulted. The introduction of EST between learning and retention trials resulted in no significant savings. A comparison of EST effects on material learned twelve hours before EST and material learned immediately prior to EST indicated that recently acquired material was more severely affected than was more remote material. The latter differences were based on rather small deficits in savings scores with insufficient data provided to permit adequate statistical evaluation. Williams (1960) and Cronholm and Molander (1958) confirmed the findings of Zubin and Barrera.

Another area of research provided similar support for the consolidation interpretation of RA. Russell and Nathan (1946) surveyed 1,209 cases of head injury and found that 133 reported RA effects for events occurring 30 minutes or more prior to trauma. Seven-hundred seven cases acknowledged amnesia for events occurring several seconds to thirty minutes prior to injury. The records for fifty-six cases in the sample were unavailable. Russell and Nathan concluded that the duration of RA is momentary in most cases, and that the loss of information is due to a blocking of the perseveration process. A quite unavoidable early assumption was that the basic neurochemical events associated with memory storage would be fairly similar across different types of learning and that the amnesic effects associated with differing amnesic agents (AA) would have fairly similar effects. This assumption, never strongly held and certainly not crucial to the consolidation hypothesis, has been found to be lacking in a number of aspects. It is now clear that the amount of amnesia is a function at least of the species used, the type of AA, the intensity and duration of the AA, the learning situation and the learning-AA interval.

Pearlman, Sharpless and Jarvik (1961), for example, found amnesia due to ether and phenobarbital limited to a learning-AA interval of ten minutes. Metrazol produced

amnesia at least four days following learning. They were disposed to hypothesize, however, that the response deficits produced by Metrazol were due to other influences than interference with consolidation.

Paolina, Quartermain and Miller (1966) compared different durations of CO₂ exposure with different intensities of ECS. They found no RA when CO₂ was administered for less than ten seconds. When CO₂ was administered for fifteen seconds, the RA gradient extended to one minute, and it extended to four minutes for twenty-five seconds of CO₂ treatment. The effective RA gradient for ECS extended for only thirty seconds. The intensity of ECS current in milliamperes produced no differential effects. They concluded that there are qualitatively different steps in consolidation which are differentially affected by different amnesic agents.

McGaugh (1966) reported that he could get RA at a learning-ECS interval of one hour using a current of 200 milliamperes. This interval could be extended to three hours with a current of 400 milliamperes but no further extension of the interval occurred at 600 milliamperes. This finding was later confirmed by Alpern and McGaugh (1968) and Miller (1968).

These findings clearly indicate that the duration of the AA can be a determiner of the amount of RA. It now seems, in fact, that some of the conflicts noted above are due either

to the degree of original learning (Ray and Bivens, 1968), or to the intensity of the AA (Jarvik and Kopp, 1967; Lee-Teng, 1969; Miller, 1968; Ray and Barrett, 1969). The amnesia gradient can probably be described most accurately by a family of curves in which these variables determine the parameters.

Probably the least researched of all amnesia agents is hypothermia. The first laboratory investigations of lowered body temperature were directed at reducing retrograde inhibition following learning by reducing general body activity. Hypothermia-induced RA offers two theoretical advantages over other methods of investigation. Firstly, all other amnesia agents commonly used bypass the peripheral nervous system and produce their effects by directly influencing the central nervous system. Hypothermia provides for a more natural succession of physiological responses involving both the peripheral and central nervous systems. Secondly, the investigation of learned behavior as affected by hypothermia carries importance because it permits the examination of the mechanisms associated with memory storage at greatly decreased metabolic levels. This provides an important test of the consolidation viewpoint, as the passage of memory traces into more permanent storage forms should require greater lengths of time at reduced levels of metabolic functioning.

French (1942) made the initial attempt at demonstrating facilitated retention following hypothermia. Goldfish were trained to traverse a four-blind linear maze to a criterion of five consecutive errorless trials. Water temperature during acquisition was maintained at 22° C. on four successive days. The average number of errors was found to be a decreasing function over time for all groups. The group maintained a 4° C. during the inter-trial intervals exhibited the fewest number of errors, and a group maintained at an inter-trial temperature of 28° C. evidenced the greatest number of errors. The comparison of these data with a control group was interpreted as demonstrating the facilitative effects of decreased body temperature on retention. It must be noted that subjects used in this research were poikilotherms (cold-blooded thermoregulators), which seriously prohibits the direct generalization of these findings to homeothermic subject investigations.

Jones (1945) trained rats to a criterion of two consecutive errorless trials on a 14-unit T-maze. Experimental Ss were then immersed in an ice-water bath for 24 hours with rectal temperatures approximating 18° C. Ss were returned to the maze 24 hours after hypothermia and retrained. Measures of trials to criterion and numbers of errors before reaching criterion were analyzed. Control Ss received the

same treatment with the exception of immersion. The data indicated that hypothermia had no effect upon the retention of the maze habit. It was concluded that 24 hours of hypothermia does not significantly interfere with retention, nor does it inhibit normal forgetting. The authors do not specify the attrition rate, though they acknowledge it to be substantial; final data were reported on 25 experimental and 48 control Ss.

Essman and Sudak (1962) examined the relationship between water-maze escape acquisition and body temperature reduction in mice. Ss were maintained either in a 33° C. or 2.5° C. ambient chamber for 30 minutes prior to acquisition. Ss were then given three trials of water escape training, with inter-trial intervals of 15 minutes during which Ss were returned to their appropriate ambient temperature cages. Normal mean colonic temperatures approximated 39° C. The results indicated that Ss whose rectal temperatures were maintained below 37° C. failed to exhibit significant learning over trials. This moderate hypothermia, only 2° C. from normal temperature, could not be explicitly attributed to impaired learning ability, as the absolute temperature to which the Ss were reduced may have had associated debilitating properties.

To examine the latter possibility, Essman and Sudak repeated their previous design, however, they maintained all

Ss in a normothermic inter-trial environment and varied water temperatures of 2, 13, 20, and 34° C. across groups. Results indicated that the 2, 13, and 20° C. groups all evidenced a significant decrease in latencies over trials, and that the 34 degrees C. control group showed no such facilitation. The experimenters concluded that the extent of body temperature reduction, at least in situations of brief exposure, probably contributed more to the motivational level of the subject thereby facilitating escape performance from aversive stimulation, than it contributed to the production of physical debilitation.

Essman and Sudak (1963) also demonstrated the failure of mice to retain a single-trial conditioned avoidance response. Using a modification of the traditional step-down apparatus (Jarvik and Essman, 1960), they compared the step-down latencies of hypothermic trained Ss and normothermic trained Ss on a normothermic test trial 30 minutes after conditioning. A significant retention deficit resulted from only a 2.8° C. reduction in core body temperature. It was also determined, in a control experiment, that escape responses could be elicited from similarly hypothermic mice at temperatures in excess of 10° C. body temperature reduction. It thus appears unlikely that the former results were an artifact of sensory or motor dysfunction.

Richman, Parrett, Black-Schaffer and Senter (1967) trained rats to traverse a complex maze, under 48 hours food deprivation, to a criterion of five minutes or less, with no more than ten errors on two successive trials. All Ss then received deep hypothermia (core temperature of 5 to 10° C.). Analyses of both number of errors and running speed on pre- and post-hypothermia trials failed to yield any significant differences. These results indicated that hypothermia has no deleterious effects upon a well-learned maze habit, and that the introduction of hypothermia late in training has no effect upon motivation.

Several general statements concerning the effects of hypothermia on acquisition can be offered:

1. Moderate hypothermia has no deleterious effect on the retention of a previously conditioned response provided the hypothermia is of short duration and is not continued during the inter-trial intervals of cumulative tasks.
2. Moderate or severe hypothermia can produce retention deficits if introduced following the training of a single-trial task.
3. Mild hypothermia can facilitate performance by increasing behavior of avoidance or escape from hypothermia.
4. Cumulative tasks appear more resistant to the amnesic effects of hypothermia than do single-trial tasks.

Riccio, Hodges and Randall (1968) have provided the

most definitive investigation of the effect of the training-hypothermia interval on task retention. They trained rats in a single-trial passive avoidance task using a conventional black-white discrimination chamber. Ss were placed in the white half of the chamber and individual step-through latencies were recorded. Hypothermia was then induced by immersion in an ice-water bath for ten or eleven minutes (mean colonic temperature achieved was 20° C.) with immersion occurring either .33, 5, 15, or 60 minutes after training. Immersion control Ss were immersed in a bath approximating body temperature. Retention control Ss received no immersion. Results indicated that initiation of hypothermia at both .33 and 5 minutes following training resulted in significant retention deficits when tested on the passive avoidance task 24 hours after training. Their investigation indicated that stress accompanying water immersion could not be responsible for the behavioral deficits since immersion control Ss performed equally as well as retention control Ss. Their data clearly demonstrated that the effectiveness of hypothermia as an AA bears a strong inverse relationship to the temporal proximity between learning and hypothermic induction. It also may be concluded that the effects of learning, in this specific task situation, are relatively impervious to the amnesic effects of hypo-

thermia after a post-training interval of five minutes.

Beitel and Porter (1968) similarly investigated the temporal gradient effects of hypothermia in mice employing another one-trial passive avoidance task. They measured step-down latencies to an electrified grid floor both during training and testing 24 hours later. Training immersion intervals were experimentally varied .5, 5 and 20 minutes, and duration of hypothermia was factorially varied at .16, 5, 15 or 30 minutes. Test results indicated that the duration of hypothermia exhibited no reliable effects after a learning-immersion delay of 20 minutes; duration of hypothermia did significantly affect retention at both .5 and 5 minute delay intervals, with increased hypothermic exposure producing more pronounced retention deficits. They concluded that the consolidation associated with this task is secured within a post-learning interval of 20 minutes.

There is research which suggests that the effects of severe hypothermia may extend far beyond the 24 hour period conventionally examined in the literature. Beitel and Porter (1968) observed that weight losses in post-hypothermic mice continue to produce altered ingestion patterns or motivational levels long after the immediate hypothermic effects have disappeared. Beitel and Porter trained mice in a water maze

escape task to discriminate between a white arm, offering a ladder to escape the maze, and a black arm, offering no such escape. Discrimination training consisted of five trials per day for twelve days. Numbers of errors and latencies were recorded for each trial. A comparison of retention, as measured by mean number of errored trials per block of five trials, revealed that Ss who received hypothermia prior to the initiation of the twelve day training period continued to exhibit only chance-level discrimination during the training phase as well as on retention tests three days after completion of training. Ss who received hypothermia prior to training evidenced partial habit recovery when tested sixty days after training. There were no differences in retention between Ss receiving hypothermia prior to training and Ss receiving hypothermia at termination of training. The results indicated that the disruptions caused by severe hypothermia may be relatively permanent proactive effects, at least in comparison to results of other known amnesic agents.

The permanence of the disruption produced by hypothermia has not been wholly supported in the literature. Riccio and Stikes (1969) found that rats trained on a passive avoidance task (black-white step-through apparatus) showed substantial memory loss when tested 1, 5 or 10 days

later. The proactive effects of hypothermia were investigated by immersing one group of Ss in ice-water for 20 minutes, 24 hours before training. Non-cooled controls and hypothermic Ss showed comparable acquisition of the avoidance habit. Since memory loss was incomplete, though substantial, there existed the possibility that repetition of the passive avoidance task may demonstrate increased savings. After two presentations of the learning situation, with experimental Ss receiving foot-shock and hypothermia on each trial, experimental Ss evidenced significantly greater retention than did naive Ss who received only footshock. The authors interpreted these data as supportive of a relatively permanent residual memory build-up which permits accelerated retrieval with additional training experience. The results seem to warrant a slightly more cautious interpretation, since it must be remembered that the experimenters' methodology failed to produce a complete amnesic effect, when such findings are quite prevalent in the literature (Beitel and Portar, 1968; Mrosovsky, 1963; Mrosovsky, 1967; Sudak and Essman, 1962b). This effect has been replicated by other researchers (Nachman and Meinecke, 1969; Pfingst and King, 1969). Jenson and Riccio (1970) have identified three treatments which similarly attenuate the amnesic effects: familiari-

zation with the apparatus prior to training; employment of a training, extinction, and retraining paradigm; or the aforementioned training, hypothermia, retraining paradigm. Each of these methods has been demonstrated to significantly diminish amnesic effects from the magnitude generally associated with a single-trial task followed by hypothermia.

Several investigators have examined the role of seizure activity. Jacobs and Sorenson (1969) demonstrated retention impairment produced by subjecting mice to strong peripheral stimulation. Ss were trained in a single-trial passive-avoidance task in a modified step-through apparatus. All Ss received a one second foot shock upon completion of the step-through response. Experimental Ss fell into ice-water baths ten seconds after termination of foot shock and control Ss similarly fell into sawdust. Six hours after training, conditions for each group were reversed: experimental Ss were dropped into sawdust while control subjects were dropped into ice-water, thus controlling for the differential effects associated with training. Experimental group retention was found to be significantly impaired when tested on the task 24 hours after training. A 1° C. decrease in core body temperature was observed to be produced by ten seconds of immersion in ice-water. Jacobs and Sorenson concluded that the retention of deficits exhibited by the experi-

mental Ss did not appear to be the result of hypothermia, since a 1° C. core body temperature reduction is not sufficient to disrupt neural activity. They interpreted their data as supporting the hypothesis that strong peripheral stimulation immediately following training is sufficient to disrupt memory. Immersion in hot water (48° C.) was found to produce comparable experimental results to immersion in ice-water.

Grosser and Percy (1971) contradicted the findings of Jacobs and Sorenson, failing to find retention deficits upon repetition of the initial design. Grosser and Percy further demonstrated that the previously reported deficits were, in reality, an artifact resulting from the positioning of the Ss in the training and testing situations. Subsequent research (Kane and Jarvik, 1970) has similarly supported the necessity of convulsion activity to effect the impairment of retention of a single-trial passive avoidance task.

The generality of the hypothermic RA effect demonstrated in single-trial passive avoidance tasks has received little attention. Carew (1970), working with ECS, has demonstrated that RA evidenced in passive avoidance tasks may not be reflected in simultaneously gathered measures, operant measures or active avoidance measures. Although the pre-

ponderance of passive avoidance single-trial research strongly supports the RA effects of hypothermia, it would substantially benefit from demonstrated generality across tasks. The objective of the present research was the investigation of the effects of hypothermia on Ss whose training and testing situations offered differential assessment of acquisition and retention on both passive and active avoidance tasks. Analysis of these differential measurements provided important information as to any task-specific limitations of the RA produced by hypothermia.

METHOD

Subjects. Forty male hooded rats served as Ss in the experiment. Ss were received from a commercial supply house at 80 to 100 days of age and were housed in gang cages until three days prior to training, at which time they were transferred to individual wire mesh cages. All Ss were housed in a colony room which was continually lighted, of relatively constant humidity and temperature, and maintained on ad libitum access to food and water. Ss were approximately 100 days of age at onset of experimentation.

Apparatus. The major apparatus consisted of a black-white avoidance chamber measuring 6 x 9 x 18 in. with a grid floor consisting of 3/16 in. stainless steel rods spaced 1/2 in. apart. Transparent plexiglas covered the top of the chamber, with hinged lids allowing access to either side. A central plexiglas partition divided the chamber into two equal compartments measuring 6 x 9 x 8 3/4 in. with a central 3 x 6 in. guillotine door allowing access between the compartments. Black or white cardboard inserts were attached to the proper sides of the partition, guillotine door and floor to effect completely white or completely black compartments. A shock source capable of delivering a 1.6 milliamperes electric shock (UCS) was connected to the grid floor of the white compartment. A conventional 110 volt home door buzzer (CS)

was attached to the center of one wall on the outside of the white chamber. The experimental apparatus was positioned in a 8 x 8 x 10 ft. acoustically insulated laboratory room.

A Yellow Springs 43TC thermometer with a flexible rectal probe was used to record core body temperatures. Plexiglas restraining tubes measuring 7 x 2.5 in i. d., with numerous holes to allow water circulation, were used to confine Ss during immersion in the water baths. Water baths were either non-hypothermic (NHYP: 39° C., approximately body temperature) or hypothermic (HYP: 1° C., an ice-water mixture).

Time latencies, to the nearest one hundredth of a second, were recorded by E on two Marietta 14-15 digital .01 second timers.

The rewarming chamber consisted of a wooden box 12 x 12 x 24 in. with a removable screen lid. A perforated Masonite false floor was fitted 6 in. above the floor of the chamber. Three 100 watt light bulbs were mounted to the floor and served as a source of heat.

Procedure. All Ss were individually housed in wire mesh cages three days prior to experimentation with ad libitum access to food and water. Each S received five minutes of handling on each of three consecutive days prior to onset of

experimentation. Experimentation was divided into three phases occurring at consecutive 24 hour intervals. The phases of the experiment included pre-training, training, and testing.

During the pre-training phase (Day 1), Ss were randomly assigned to either Group PA (passive avoidance response preceding the active avoidance response) or to Group AP (active avoidance response preceding the passive avoidance response). Each S in Group PA was placed in the white chamber with his head directed away from the closed guillotine door. The guillotine door was raised 2 sec. after placement, and step-through latencies (STL) were determined for each subject. STL for Group PA (passive task) reflect the latency of each S to enter the black chamber with all four feet after placement in the white compartment. STL for Group PA (active task) indicate the latency of each S to re-enter the white chamber after placing all four feet in the black chamber. Any S who failed to make the step-through response during the 300 sec. interval was immediately forced into the opposite chamber. Ss in Group PA were removed from the apparatus immediately upon re-entry into the white chamber and returned to their home cages. Pre-training latency measures were determined on three consecutive days prior to training. No CS or UCS was presented during pre-training.

During the pre-training phase for Group AP, each S was placed in the black chamber with his head facing away from the closed guillotine door. The guillotine door was raised 2 sec. after placement. Active task latencies were recorded as the STL of each S to enter the white chamber (all four feet), and passive task latencies reflect the amount of time each S remained in the white compartment before re-entering the black compartment. A 300 second upper limit on latencies was employed; any S who failed to make a step-through response during the 300 sec. interval was immediately forced into the opposite chamber. Ss in Group AP were removed from the apparatus immediately upon re-entry into the black chamber and returned to their home cages. Pre-training latency measures were determined on three consecutive days prior to training. No CS or UCS was presented during this phase.

During training (Day 4), Groups PA and AP were randomly divided into the following experimental groups. The experimental conditions are presented in Table 1.

PA-FS-HYP. Each S was placed in the white compartment facing away from the open guillotine door. Five seconds after completion of the STR the guillotine door was closed, and CS was presented. Foot shock (UCS) was delivered through the grid floor of the black compartment beginning one second

TABLE I

EXPERIMENTAL CONDITIONS

C (Sequence)	PASSIVE TASK				ACTIVE TASK			
A (Footshock)	FOOTSHOCK				NO FOOTSHOCK			
B (Hypothermia)	Hypothermia	No Hypothermia	Hypothermia	No Hypothermia	Hypothermia	No Hypothermia	Hypothermia	No Hypothermia
Experimental Groups	PA	PA	PA	PA	AP	AP	AP	AP
	FS	FS	NFS	NFS	FS	FS	NFS	NFS
	HYP	NHYP	HYP	NHYP	HYP	NHYP	HYP	NHYP

after initiation of CS. The guillotine door was opened two seconds after initiation of UCS. CS and UCS were terminated when Ss escaped to the white chamber. Passive and active task STL measures were recorded. Ss were removed from the apparatus immediately upon returning to the white chamber. Each S was then restrained in a plexi-glas cylinder and immersed in an ice-water bath until core body temperature was decreased 39° C. to 20° C. Ss were then removed from the ice-water baths, taken from the restraining tubes, and placed in the rewarming chamber until normal ambulatory activity was observed. Ss were then returned to their home cages.

PA-FS-NHYP. Each S in this group received treatment identical to Group PA-FS-HYP, with the exception that Ss were immersed for ten minutes in a 39° C. water bath.

PA-NFS-HYP. Each S in this group received treatment identical to Group PA-FS-HYP, with the exception that no UCS was administered during training.

PA-NFS-NHYP. Each S in this group received treatment identical to Group PA-FS-HYP, with the exception that no UCS was delivered during training and immersion was of ten minutes duration in a 39° C. water bath.

AP-FS-HYP. Each S was placed in the black compartment

facing away from the closed guillotine door. CS was initiated five seconds after placement in the black compartment. UCS was introduced through the grid floor of the black compartment three seconds after initiation of CS. The guillotine door was opened two seconds after introduction of UCS, and both CS and UCS were terminated upon subject's escape to the white compartment. Passive and active task latencies were recorded. Ss were removed from the apparatus upon return to the black chamber, or, if the latency ceiling was attained, subjects were forced into the white chamber then removed. Each S was then restrained in a plexiglas cylinder and immersed in an ice-water bath until core body temperature was decreased to 20° C. Ss were then removed from the ice-water bath, taken from the restraining tubes, and placed in the rewarming chamber until normal ambulatory activity was observed. Ss were then returned to their home cages.

AP-FS-NHYP. Each S in this group received treatment identical to Group AP-FS-HYP, with the exception that Ss were immersed for ten minutes in a 39° C. water bath.

AP-NFS-HYP. Each S in this group received treatment identical to Group AP-FS-HYP, with the exception that no UCS was administered during training.

AP-NFS-NYYP. Each S in this group received treatment identical to Group AP-FS-HYP, with the exceptions that no UCS was delivered during training and immersion was of ten minutes duration in a 39° C. water bath.

On Day 5 all Ss were tested for retention of the one-trial learning avoidance tasks. Ss were placed either in the white compartment (Groups PA) or in the black compartment (Groups AP) and CS was presented in the same manner as during the training phase. No UCS was presented during testing. The guillotine door was manipulated for the groups on contingencies identical to those used in training. STL were recorded by E on each subject.

Subjects in Group PA received an additional active avoidance test trial following the passive avoidance test. Testing procedures and contingencies were identical to active avoidance testing for Groups AP. Ss in groups AP received an additional passive avoidance test trial following active avoidance testing, with testing procedures and contingencies identical to passive avoidance testing for Groups PA. STL were recorded on each S by E.

RESULTS

The 300 sec. ceiling imposed on all latency measurements warranted the use of a reciprocal data transformation to normalize within-cell variance (see Appendix A). Independent analyses of pre-training mean reciprocal step-through latencies for subjects were analyzed by analysis of variance for both passive and active avoidance responses.

Analysis of mean reciprocal step-through latencies for pre-training passive task responses is summarized in Table 2. No significant effects due to footshock, hypothermia or any interaction of these factors were obtained. The analysis indicated a significant main effect due to task sequence ($F=29.36$, $df=1, 32$; $p < .05$), and demonstrate significantly increased mean pre-training passive task latencies for AP task sequence subjects. A graphical representation of this effect is provided in Fig. 1.

Analysis of a mean reciprocal step-through latencies for pre-training active task latencies is summarized in Table 3. No significant effects due to footshock, hypothermia or any interaction of these effects were obtained. A significant main effect due to task sequence ($F=32.87$, $df=1, 32$; $p < .05$) was obtained. Graphical representation of this effect is provided in Fig. 2 and indicates significantly increased mean pre-training active task latencies

TABLE 2

ANALYSIS OF VARIANCE:
PASSIVE TASK PREDICTOR

Source	df	MS	F
Footshock (A)	1	.000	.09
Hypothermia (B)	1	.001	.18
Sequence (C)	1	.174	29.36*
A X B	1	.013	2.35
A X C	1	.000	.00
B X C	1	.001	.28
A X B X C	1	.015	2.54

* $p < .05$

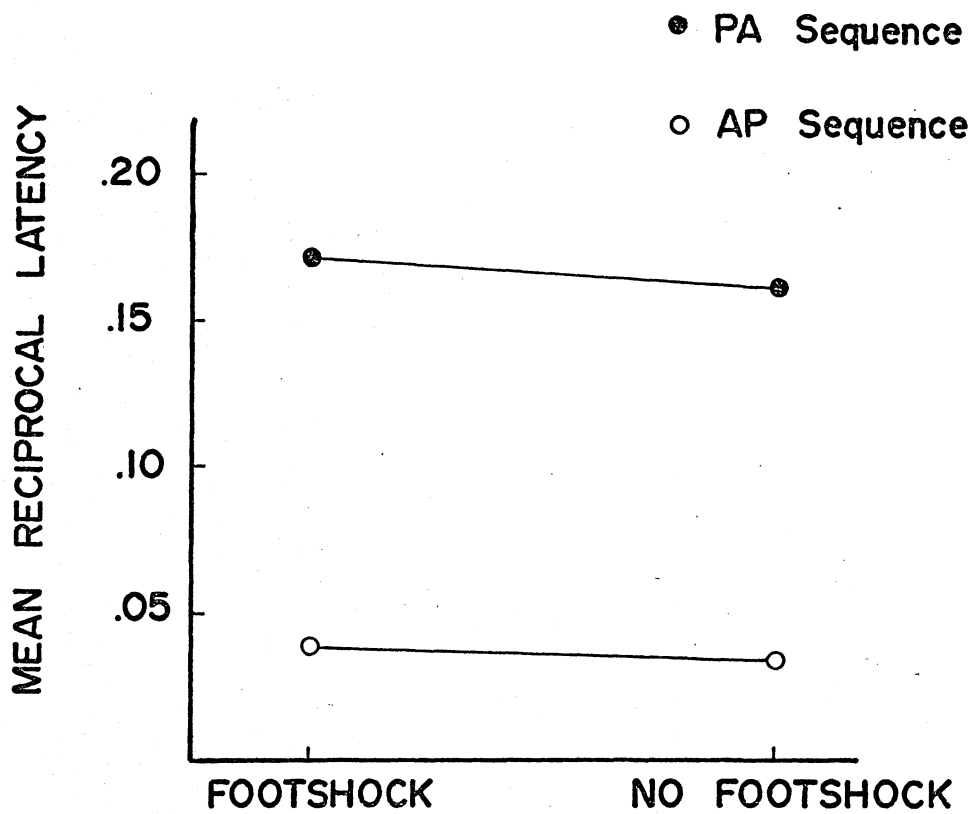


Fig. 1. Mean reciprocal latency for passive response predictors as a function of task sequence and footshock.

TABLE 3

ANALYSIS OF VARIANCE:
ACTIVE TASK PREDICTOR

Source	<i>df</i>	<i>MS</i>	<i>F</i>
Footshock (A)	1	.005	1.17
Hypothermia (B)	1	.008	1.68
Sequence (C)	1	.166	32.87 *
A X B	1	.000	.05
A X C	1	.011	2.35
B X C	1	.001	.30
A X B X C	1	.002	.55

* $p < .05$

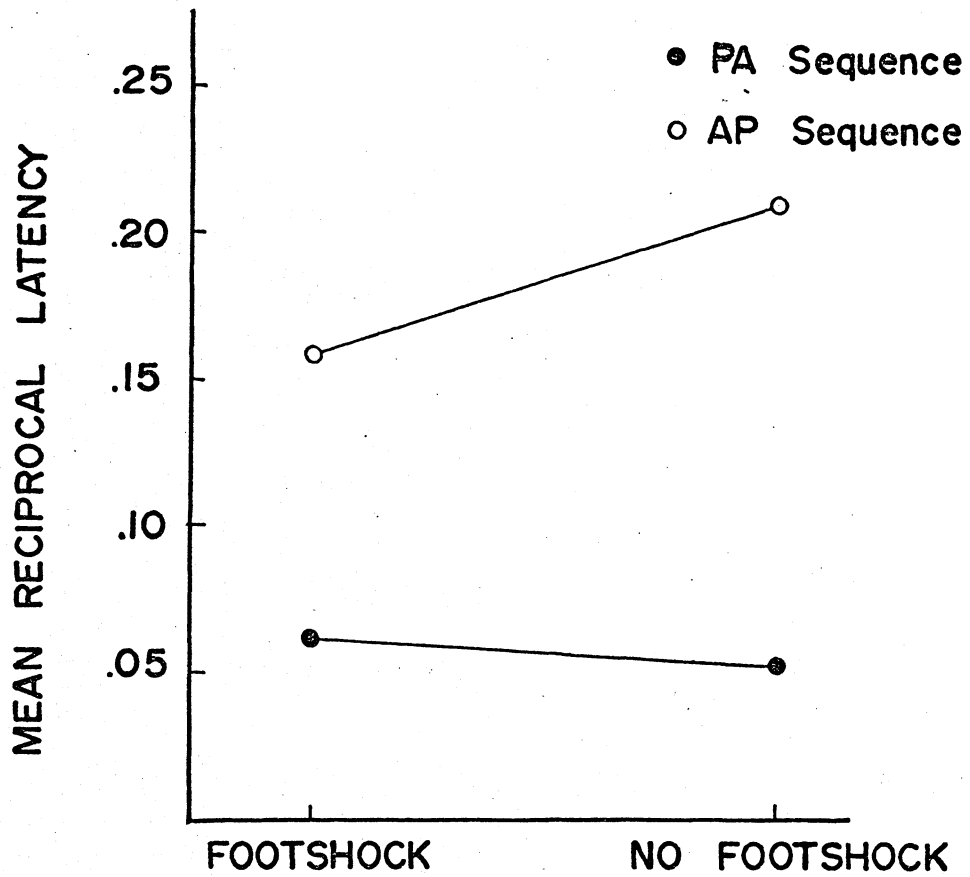


Fig. 2. Mean reciprocal latency for active response predictors as a function of task sequence and footshock.

for PA task sequence.

The correlation of pre-training mean reciprocal latencies and retention test reciprocal latencies was used as an index of the potential effectiveness of analyzing retention test data in terms of the predictable variation attributable to the covariate pre-training latencies. The use of such a covariate would provide statistical control of variability due to idiosyncratic differences in subjects' latencies. The correlation between mean reciprocal passive task pre-training latencies and passive task test reciprocal latencies was negligible ($r = -.09$); the correlation between mean reciprocal active task pre-training latencies and active task test reciprocal latencies indicated a more substantial relationship ($r = -.54$). Based on these findings, it was judged that the additional power provided by the use of a covariate in the analysis of test data would be of minimal value over unadjusted data analysis.

The results of the analysis of reciprocal latencies for the passive task test are summarized in Table 4, and indicated significant main effects due to footshock ($F = 25.77$, $df = 1, 32$; $p < .05$) and task sequence ($F = 11.92$, $df = 1, 32$; $p < .05$). A significant footshock X task sequence interaction ($F = 8.95$, $df = 1, 32$; $p < .05$) was also evidenced. Because no performance differences in passive task test latencies could be attri-

TABLE 4

ANALYSIS OF VARIANCE:
 PASSIVE TASK

Source	<i>df</i>	<i>MS</i>	<i>F</i>
Footshock (A)	1	.164	25.77 *
Hypothermia (B)	1	.005	.92
Sequence (C)	1	.075	11.92 *
A X B	1	.000	.11
A X C	1	.056	8.95 *
B X C	1	.002	.37
A X B X C	1	.000	.05

* $p < .05$

buted to the effects of hypothermia, hypothermia was ignored in the graphic presentation of the data (Fig. 3).

The analysis of simple effects for the footshock X task sequence interaction was reported in Table 5. The analysis indicated significant differences in the effects of footshock for task sequence PA ($F=34.58$, $df=1, 32$; $p<.05$), with No Footshock-PA group latencies significantly lower than those of the Footshock-PA group.

The significant increase in latencies for group FS-PA passive avoidance tests seems to indicate that single-trial passive avoidance training was effectively accomplished. The STL for the passive response for FS-PA could not be shown as being different from groups FS-AP or NFS-AP. Group NFS-PA latencies approximated pre-training latency levels, indicating no spurious latency changes which could be specifically attributed to the additional trial.

The analysis of reciprocal latencies for the active task test are summarized in Table 6. The analysis indicated significant main effects due to footshock ($F=6.65$, $df=1, 32$; $p<.05$) and task sequence ($F=8.19$, $df=1, 32$; $p<.05$). A significant footshock X task sequence interaction ($F=10.57$, $df=1, 32$; $p<.05$) was noted. The effects attributed to hypothermia were negligible, and therefore ignored in the graphic presentation of the data (Fig. 4).

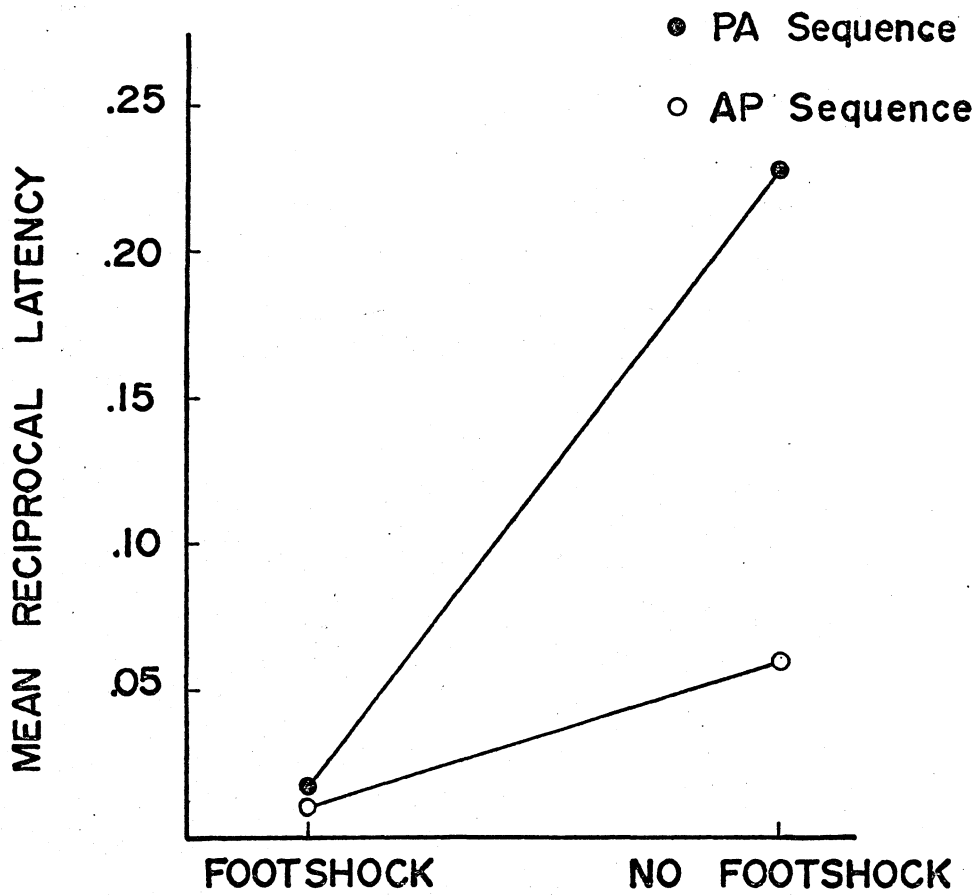


Fig. 3. Mean reciprocal latency for passive response test as a function of task sequence and footshock.

TABLE 5
ANALYSIS OF VARIANCE FOR SIMPLE EFFECTS:
PASSIVE TASK

Source	<i>df</i>	<i>MS</i>	<i>F</i>
A for C ₁ (Footshock for Sequence PA)	1	.2075	34.58*
A for C ₂ (Footshock for Sequence AP)	1	.0001	.02
Within cell	32	.0060	

* $p < .05$

TABLE 6

ANALYSIS OF VARIANCE:
ACTIVE TASK

Source	df	MS	F
Footshock (A)	1	.292	6.56*
Hypothermia (B)	1	.019	.45
Sequence (C)	1	.364	8.19*
A X B	1	.061	1.37
A X C	1	.470	10.57*
B X C	1	.058	1.31
A X B X C	1	.005	.12

* $p < .05$

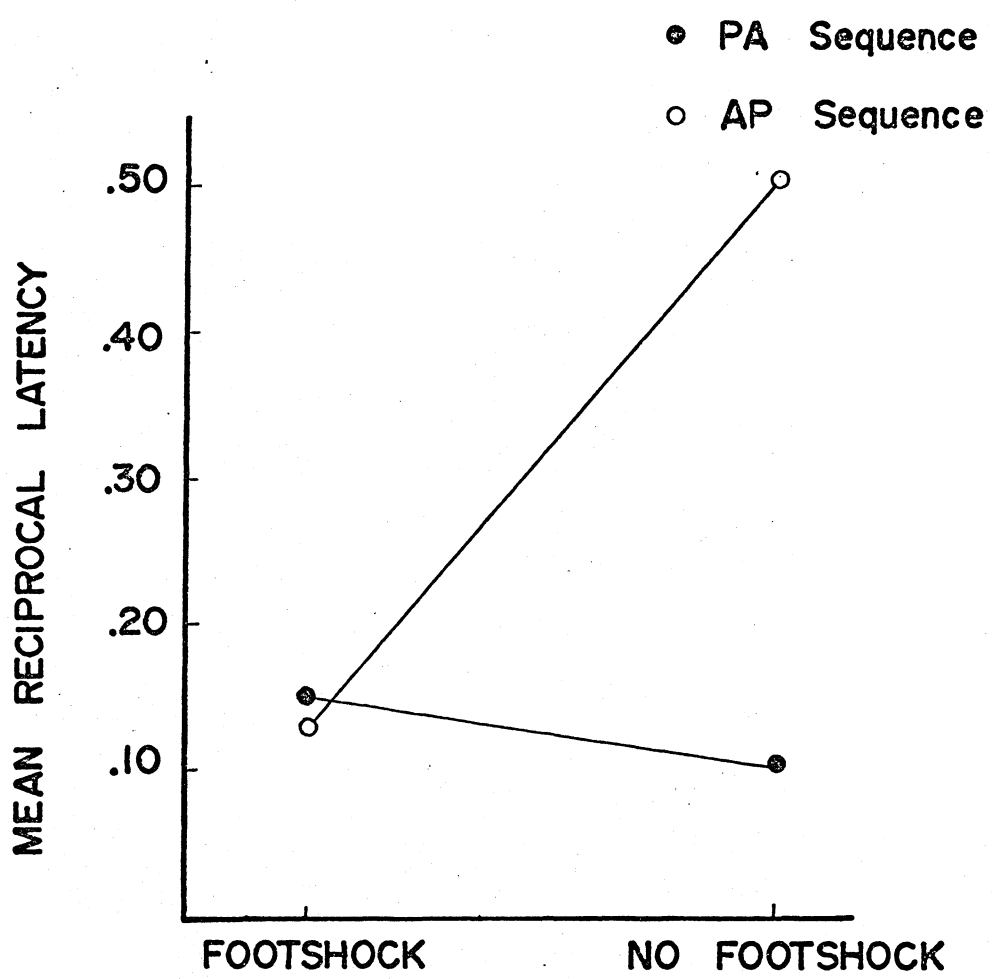


Fig. 4. Mean reciprocal latency for active response test as a function of task sequence and footshock.

The analysis of simple effects associated with the footshock X task sequence interaction for active task test latencies is reported in Table 7. The analysis indicated a significant difference in the effect of footshock for task sequence AP ($F=17.09$, $df=1, 32$; $p<.05$), with No Footshock-AP task sequence group latencies significantly lower than Footshock-AP group latencies.

Active-task test latencies for groups FS-PA, NFS-PA, and NFS-AP similarly did not differ from pre-training performance levels. However, the significantly increased latencies of group FS-AP indicates that active avoidance was not facilitated, as successful active avoidance would be demonstrated by decreased latencies in this group.

TABLE 7

ANALYSIS OF VARIANCE FOR SIMPLE EFFECTS:
ACTIVE TASK

Source	<i>df</i>	<i>MS</i>	<i>F</i>
A for C_1 (Footshock for Sequence PA)	1	.0105	.24
A for C_2 (Footshock for Sequence AP)	1	.7520	17.09*
Within cell	32	.0440	

* $p < .05$

DISCUSSION

The primary question examined in the present research is whether single-trial passive and active avoidance tasks permit accurate assessment of retrograde amnesia. The present investigation is handicapped in answering this question, as the data failed to demonstrate retention deficits which could be attributed to the amnesic effects of hypothermia.

Riccio, Hodges and Randall (1968) and Riccio and Stikes (1969) employed essentially identical passive avoidance training and testing paradigms to those of the present design :no hypothermic RA investigations have used an active avoidance paradigm. The present study similarly employed hypothermia according to conventional criteria (Riccio, Hodges and Randall, 1968) reliably demonstrated to produce RA. An identical method of hypothermic induction was utilized with rigorous attention directed towards compliance with the established parameters. The present failure of Ss to demonstrate amnesic phenomena, at least concerning the passive avoidance test, cannot be attributed to methodological incongruencies with previous research.

The choice of the hooded strain of rats for the present study represents one remarkable difference from other investi-

gations, which have typically employed albino rats and mice. Although present Ss were comparable in weight and sex to Ss employed by other investigators, it was noted that present Ss failed to exhibit any debilitating effects or attrition following hypothermia, and resuscitated quite rapidly. By comparison, present Ss did not appear to experience the severity of hypothermia reported by other researchers. The present results may provide evidence concerning the existence of strain-specific differences in resistance to the effects of hypothermia. The generality of the parameters requisite to hypothermic amnesia have not yet been satisfactorily established, and continued efforts at demonstration of the generality of the amnesic phenomena within strains of the present species merits attention.

The present results remain difficult to reconcile with theories employing the concept of RA. RA would have been a tenable explanation if Ss receiving avoidance training and hypothermia evidenced retention test latencies approximating pre-training performance levels. The observed significant increases in latencies for avoidance-trained hypothermic Ss, in direct opposition to a RA interpretation, cannot be interpreted as disproof of the RA phenomenon. Rather, attention must be directed towards identification of a potentially

stronger effect than RA present in the experimental situation.

Pre-training data indicated that Ss remained significantly shorter periods of time in the compartment of the apparatus in which they were placed as compared to the non-placement compartment. Such behavior could be adequately explained either in terms of exploratory activity or escape from any mildly aversive stimuli associated with handling or placement. During the testing phase, however, Ss receiving avoidance training demonstrated significantly increased latencies in the compartments of placement, regardless of the compartment in which footshock had been administered. The seemingly significant passive avoidance conditioning cannot, therefore, be interpreted in terms of discriminated passive avoidance. Any passive avoidance responses which became conditioned, during the single training trial, were effectively generalized to the entire experimental apparatus environment.

The increased test latencies for Ss receiving footshock in both the PA and AP task sequences on both passive and active avoidance tests evidenced unusual similarity. Increased passive avoidance test latencies may readily be interpreted as successfully retained passive avoidance conditioning; but, what interpretation can then be given to

the same subject's failure to demonstrate active avoidance, and moreover, evidence active avoidance task test latencies in the identical direction and of similar magnitude to passive avoidance task test latencies? If a common, sufficiently powerful effect can account for increased test latencies on both avoidance tasks, when in fact active avoidance was not facilitated but debilitated, serious questions as to the actual occurrence of any conditioned avoidance exist.

The aforementioned incongruencies can be eliminated by interpreting the present results in terms of a fear hypothesis (Coons & Miller, 1960). According to this interpretation, strong emotional responses (e.g., fear) may become conditioned through a single experimental presentation of intense aversive stimulation; such conditioned emotional responses will again be elicited upon presentation of stimuli associated with the conditioning environment.

No aversive effects can be attributed to hypothermia in the present research, however, footshock was demonstrated to exhibit significant influence on test latencies. Footshock, administered in the black compartment and generalized to the entire experimental environment, could easily have been the source of such conditioned emotionality. Suppression of normal locomotor activity, due to debilitating levels

of conditioned emotionality, readily accounts for both increased test latencies on both passive and active avoidance tests for the same S. No measures of typical behavioral correlates of fear in rats (urination, defecation, etc.) were recorded during the present experimentation, and it is with substantial reluctance that the term fear be employed to describe this hypothesized conditioned emotionality. Regardless of the specific qualities of the conditioned emotionality, what is important is that experimental measurements, specifically conventional time latencies, fail to reliably assess one-trial avoidance conditioning. Perhaps the primary question of the present research should properly have asked if measurements of one-trial avoidance conditioning accurately measure one-trial avoidance behavior.

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APPENDIX A

The employment of a 300 sec. ceiling on latency response measures warranted the employment of a reciprocal transformation of data in an attempt to normalize within-cell variation. The F_{\max} test for homogeneity of error variance was used to assess the effectiveness of this transformation in normalizing within-cell variation. Reciprocal scores for passive task predictor, active task predictor and active task test latencies failed to violate the parametric assumption of normal distribution of error variance. Passive task test reciprocal latencies were found to significantly violate the assumption of normal distribution of error variance ($F_{\max} = 2031.25$, $df = 8,4$; $p < .05$). Inspection of the scores comprising the cells of interest in this analysis indicated that four of the five scores were identical in cell with minimal variation. The actual values of these identical scores reflect that four Ss were scored as 300 sec. responses. The violation of the parametric assumption of normal distribution of error variance, in the analysis of the passive task test reciprocal latencies, is therefore an artifact of the imposition of the latency ceiling.

VITA

Duane E. Brookhart, the author, was born in Harrisburg, Pennsylvania on 5 August 1948. He resided in the community of Liverpool, Pennsylvania throughout his youth, and was graduated from Greenwood High School in 1966. He entered Susquehanna University at Selinsgrove, Pennsylvania in September, 1966 and was awarded a Bachelor of Arts degree from that university in June, 1970. In September, 1970 he began work toward the degree of Master of Arts in Psychology at the University of Richmond. The author has been employed by the Virginia State Penitentiary since May, 1971, and presently holds the position of psychologist at that institution.