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Hypothermia: Amnesic agent, punisher, and conditions sufficient to attenuate amnesia

CHARLES F. HINDERLITER
Kent State University, Kent, Ohio 44240

In three experiments, the effects of prior experience with hypothermia as a manipulation to attenuate amnesia were examined. In Experiment 1, prior experience with one hypothermia treatment did not appear to influence the amnesic effects of hypothermia. In Experiment 2, hypothermia treatment was made contingent upon stepping into a darkened chamber of a passive avoidance apparatus daily for 8 days. By Day 8, latencies indicated that subjects could remember the previous hypothermia treatments. The values of hypothermia found to produce learning in Experiment 2 were then used to evaluate several retrieval hypotheses concerning the prevention of amnesia in Experiment 3. Rats which received eight daily hypothermia treatments, as well as rats restrained without immersion, showed little evidence of amnesia following a train-hypothermia treatment. Results were interpreted as supporting the view that amnesia may be produced because important contextual cues necessary for memory retrieval are usually absent during testing.

Literally hundreds of experimenters have demonstrated that traumatic treatments such as electroconvulsive shock (ECS), pharmacological suppression of protein synthesis, and extreme reduction of body temperature result in forgetting of events preceding these amnesic treatments. (Barraco & Stettner, 1976; Gibbs & Mark, 1973). Because events that closely precede the amnesic treatment are the most severely affected, the memory loss is referred to as retrograde amnesia. Although the effects of treatments such as ECS are quite robust, there are a variety of manipulations that can attenuate amnesia (e.g., Hinderliter, Smith, & Misanin, 1973; Kesner, McDonough, & Doty, 1970). Systematically manipulating the amount of familiarization with the apparatus where a train-ECS treatment was to be given, Lewis, Miller, and Misanin (1968) found that a minimum of three ½-min exposures was sufficient to attenuate amnesia. Hinderliter et al. (1973) have shown that prior experience with ECS or the aversive stimulus used in training (footshock) also can effectively attenuate the amnesic effect of ECS. These findings, along with those of Kesner et al. (1970) and Lewis et al. (1968), indicate that sufficient experience with almost any aspect of the train-amnesic treatment may reduce amnesia. The three experiments described in this paper were designed to explore the generality of prior experience effects. Experience with the amnesic agent was the manipulation examined in all three experiments. Because prior experience manipulations have most often been examined with the use of ECS, hypothermia was used as the amnesic agent in an effort to extend these findings.

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EXPERIMENT 1

The purpose of the first study was to determine if hypothermia, like ECS, could effectively prevent or attenuate amnesia when given prior to a train-hypothermia treatment. Although Jensen and Riccio (1970) and Riccio and Stikes (1969) have shown that several types of prior experience manipulations can attenuate hypothermia-produced amnesia, the effects of hypothermia per se as a prior experience treatment have not been reported. By contrast, Hinderliter et al. (1973) and Kesner et al. (1970) have reported that ECS given 24 h prior to a training-ECS treatment effectively attenuates amnesia.

Method

Subjects. The subjects were 28 Holtzman, albino, male rats (280-470 g). The rats were caged in groups of four to six at least 1 week after receipt from the supplier, and were maintained on ad-lib food and water at all times. Twenty-four hours prior to any experimental manipulation, each rat was handled 2-3 min, weighed, ear punched for identification, and placed in an individual cage.

Apparatus. The passive avoidance apparatus consisted of a 38.1 × 16.5 × 20.3 cm Plexiglas box divided into two equal-sized chambers by a partition with a 7.5 × 6.4 cm opening. A wooden door was placed inside the partition to block the opening when necessary. One chamber of the apparatus was white and had a

clear Plexiglas lid and a solid metal floor; the other was black and had a black Plexiglas lid and a grid floor consisting of 0.25 cm stainless steel rods spaced 1.27 cm apart. A Foringer Model SC-901 scrambler and a matched impedance ac shock source were used to deliver a 150-V shock through the grids (Campbell and Teghtsoonian, 1958). The room containing the passive avoidance apparatus was illuminated by a 15-W bulb suspended 38 cm above the white chamber. A Model 2095 Forma Temp. Jr. bath and circulator containing tap water maintained at 3°-5°C was used to produce hypothermia (HYPO). Rectal temperatures were taken with a Model 43TD Yellow Springs Tele-Thermometer, with probes inserted approximately 3.2 cm into the rat's anus.

Procedure. On Day 1, 21 rats were restrained in hardware cloth cylinders and immersed in 3°-5°C water until colonic temperatures reached 20°C. Following HYPO, the rats were dried with paper towels and returned to their home cages. On Day 2, 14 of these rats and an additional 7 other rats which had not received hypothermia were given passive avoidance training. During training, each rat was placed in the white side of the passive avoidance apparatus facing away from the door separating the two chambers. Five seconds later, the door was opened. After a rat placed all four feet into the black chamber, the door was dropped and a 1-sec, 150-V inescapable shock was delivered. The amount of time taken to cross into the black chamber (training latency) was recorded to the nearest 0.1 sec.

The remaining seven rats, which had received prior hypothermia, were treated identically to the three groups of trained rats except that no footshock was administered when they crossed into the black chamber. The latencies to enter the black chamber for the no-train group were also referred to as training latencies. Only this no-train group and one of the prior hypotherma-train groups received hypothermia immediately after being removed from the black chamber. Subjects in the other two groups were returned to their cages immediately after training. To summarize, three prior hypothermia groups received footshock-HYPO, footshock-no HYPO, or no footshock-HYPO on Day 2. A fourth group, which had no prior experience with hypothermia, received only footshock during training.

Testing occurred on Day 3. Latency to enter the black chamber was recorded to the nearest 0.1 sec. Rats failing to cross within 10 min were assigned test latencies of 600 sec.

Results

Training and test latencies are shown in Figure 1. Unless otherwise stated, all comparisons involved two-tailed nonparametric tests. A Kruskal-Wallis one-way analysis of variance performed on training latencies revealed no significant differences. In contrast, the same analysis performed on test latencies revealed significant effects, $H(3) = 16.0$, $p < .01$. As indicated in Figure 1, and by Mann-Whitney U comparisons, amnesia was produced. Latencies of rats in the footshock-HYPO group were significantly shorter than either of the no-HYPO groups' latencies; $Us(1,7) \leq 10.5$, $ps < .05$, one-tailed test. Likewise, rats in the no-footshock-HYPO group had latencies shorter than either of the no-HYPO groups' latencies, $Us(7,7) = 0$, $ps < .001$. Latencies of the two footshock-no HYPO groups did not differ significantly. That amnesia was produced by the HYPO treatment was further indicated by the fact that only for rats of the two footshock-no HYPO groups were test latencies longer than training latencies, $Ts(7) \leq 1$, $ps \leq .05$, Wilcoxon matched-pairs signed-ranks test.

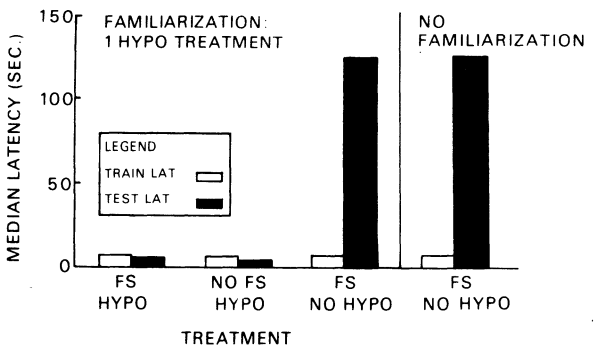

Figure 1. Median train (Day 2) and test latencies (Day 3) for rats given footshock (FS) and/or hypothermia (HYPO) on Day 2. All rats, except for the no-familiarization group, had received a HYPO on Day 1.

Discussion

Empirically, it appears that one hypothermia treatment is not sufficient to attenuate the amnesia effects of a further cooling treatment. This failure to find protection from amnesia has important theoretical implications. For example, DeVietti and Hopfer (1974) suggested that storage of a trained event occurs in a "normal state" even when followed immediately by ECS. Accordingly, testing 24 h after train-ECS treatment results in amnesia because subjects are still in the state produced by ECS. Extending this notion, it would be predicted that inducing that altered state 24 h prior to the train-amnesic treatment should permit memory to be stored in the changed state, and amnesia should not be produced. The results of this first experiment contrast with this state-dependent retrieval notion of DeVietti and Hopfer, since amnesia was not attenuated by inducing the hypothermic state 24 h prior to a train-hypothermia treatment.

Others have also reported a failure to attenuate amnesia with prior experience manipulations such as familiarization with the training apparatus (e.g., Galosy & Thompson, 1971). It is important to note, however, that in all of the studies failing to lessen the effects of an amnesic treatment, the amount of prior experience subjects had received was not systematically manipulated (e.g., Nachman & Meinecke, 1969). If prior experience with amnesic treatments is a process similar to the familiarization techniques used by Lewis et al (1968) to prevent amnesia, the reported failures may result from insufficient exposure during the prior-experience manipulations.

As Lewis et al. (1968, 1969) have shown, there evidently is a critical amount of prior experience necessary if amnesia is to be attenuated. Whether several exposures with hypothermia prior to train-hypothermia treatment will attenuate amnesia has not been determined. The effects of such manipulations may provide information relevant to differ-

entiating a state-dependent interpretation proposed by Thompson and Neely (1970) and a contextual cues model proposed by Hinderliter, Webster, and Riccio (1975); these are discussed below.

EXPERIMENT 2

According to Thompson and Neely (1970), the storage of a trained event occurs in the state produced by an amnesic treatment. When tested 24 h after train-amnesic treatment, subjects show evidence of amnesia because the amnesic-treatment cues have dissipated by the time of testing. In a similar interpretation, Hinderliter et al. (1975) emphasize the importance of contextual cues largely produced by the amnesic treatment. According to this latter interpretation, amnesia occurs because the training and the amnesic treatment constitute relatively undifferentiated events. It does not follow, from the Thompson and Neely hypothesis, that increasing the number of prior experiences should change the state-altering properties of the amnesic treatment. Because a contextual-cues notion implies that learning to differentiate training- and amnesic-treatment cues is a prerequisite to preventing amnesia, more than one experience with hypothermia may be necessary to produce such a discrimination. It follows that if subjects are given sufficient experience with an amnesic treatment to differentiate the training- and amnesic-treatment events, amnesia should be attenuated. According to a contextual cues interpretation, the number of experiences with hypothermia necessary to produce evidence of remembering the "traumatic" experience should at least be sufficient to differentiate a train-hypothermia treatment and, thus, prevent amnesia. No systematic investigation has been reported, however, concerning the effectiveness of deep hypothermia as a punishing stimulus used to support learning. Accordingly, testing of the necessary condition to differentiate a train-hypothermia treatment took place in two stages. Experiment 2 was conducted to determine whether repeated hypothermia treatments made contingent upon stepping into the black side of a passive avoidance apparatus would increase rats' latencies to reenter the black chamber, while Experiment 3 incorporated these values into an experimental manipulation evaluating attenuation of amnesia.

Method

Subjects and Apparatus. Ten Holtzman, male, albino rats were used as subjects. Treatment of subjects prior to the experiment, as well as the apparatus, were the same as described in Experiment 1.

Procedure. The general plan of this experiment included two phases. Phase 1 involved passive avoidance training similar to that of Experiment 1, except that hypothermia was used as a punishing stimulus instead of footshock. Considering the debilitating effects of hypothermia (e.g., convulsions), it seemed important to obtain evidence that daily hypothermia treatments were not impairing a subject's ability to make the required step-through response. Thus, latency to escape from the chamber that had been more closely paired with hypothermia (the black chamber) was obtained in Phase 2 in addition to a passive avoidance latency.

More specifically, training during Phase 1 involved placing each rat facing away from the door on the white side of the passive avoidance apparatus and 10 sec later, raising the door. During the passive avoidance training, the time taken to place all feet into the black chamber was recorded to the nearest 0.1 sec. Rats then were removed from the black chamber and immersed in 3°-5°C water until their rectal temperatures reached 21°C. Thus, hypothermia alone was examined as a punisher. Following hypothermia, rats were dried with paper towels and returned to their home cages. Each day, one trial was given as a test for previous training and also as a training trial for subsequent testing. The procedure of making hypothermia contingent on a step-through response each day continued for 8 days, when latencies were significantly higher than those on Day 1, $T(10) = 8$, $p = .05$, Wilcoxon matched-pairs, signed-ranks test. On any day that the rats failed to step into the black chamber within 1,800.0 sec, they were gently forced through by the experimenter and then given hypothermia.

In Phase 2, escape latencies from the black side were recorded on Day 9. Although amounting to an extinction trial, escape tests were given to determine a subject's ability to perform the step-through response. Escape latencies were obtained by placing a rat in the black chamber and measuring the time taken to place all feet into the white chamber. Rats then were removed from the white chamber, and approximately 3 min later were placed into the white compartment and given a passive avoidance test. Latencies to enter the black chamber during the passive avoidance test were recorded as on Days 1-8.

Results and Discussion

Median step-through latencies obtained on Days 1-9 are shown in Figure 2. Because 6 of the 80 scores were 1,800.00 sec, and also because variability between cells was large, nonparametric tests were performed on Day 1-8 passive avoidance latencies. A Friedman two-way analysis of variance by ranks revealed a significant Days effect, $\chi^2_r(7) = 28.9$, $p < .001$. Wilcoxon matched-pairs, signed-ranks

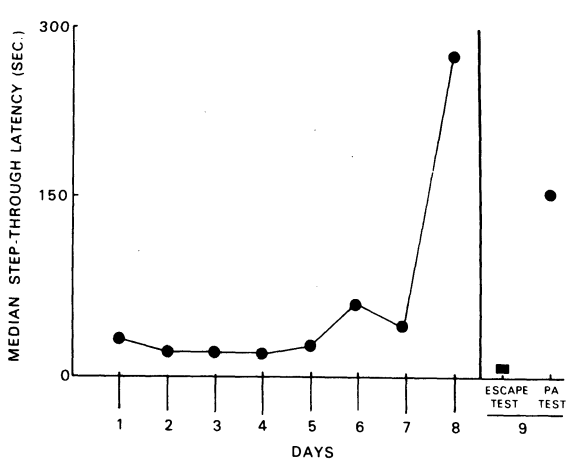

Figure 2. Median latencies for rats given daily response-contingent hypothermia treatments on Days 1-8. Latencies to escape from and passively avoid (PA) the chamber more closely associated with hypothermia were obtained on Day 9.

tests also revealed that latencies on Day 8 were significantly longer than latencies on any of the other 7 days: Ts(10) $\leq$ 8, ps $\leq$ .05, Day 1, 2, 3, 4, or 6 vs. Day 8; Ts(9, correction for tie scores) $\leq$ 4, ps $<$ .05, Day 2 or 4 vs. Day 8. Although nonhypothermia controls were not included in this experiment, other data from this laboratory indicate that rats simply removed following a step-through response continue to show short latencies over a number of trials (Schulenberg, Riccio, & Stikes, 1971). Thus, the results indicate that a hypothermia treatment of sufficient intensity to produce amnesia can also serve as punishing stimulus when given repeatedly, as can ECS (Hudspeth, McGaugh, & Thompson, 1964).

Escape latencies from the black chamber and passive avoidance latencies obtained on Day 9 are also shown in Figure 2. Wilcoxon matched-pairs, signed-ranks test revealed that escape latencies on Day 9 were significantly shorter than passive avoidance latencies obtained on Days 8 and 9, Ts(10) = 4, ps $<$ .02. These relatively short step-through latencies obtained during escape testing on Day 9 and then the significant increases when passive avoidance behaviors were recorded indicate that the increase in step-through latencies on Day 8 was not due to inactivity per se.

It should be noted that a significant weight loss occurred over the 9-day period, $F(8,72) = 11.02$, $p < .01$. Mean weights ranged from 385 g on Day 1 to 357 g on Day 9. Whether this weight loss affected step-through performance cannot be determined from the experiment. However, the relatively short escape latencies obtained on Day 9 indicate that rats certainly were capable of making the step-through response.

To summarize, it appears that repeated experience with hypothermia can be incorporated into a "memory system," as evidenced by the increase in step-through latencies in the passive avoidance paradigm used in this experiment.

EXPERIMENT 3

According to a contextual-cues hypothesis, any experience with an amnesic treatment that a subject can remember should be sufficient to differentiate training and amnesic treatments (Hinderliter et al., 1975). The purpose of Experiment 3 was to determine if eight prior hypothermia treatments would attenuate amnesia. Whether prior experience with any aspect of the hypothermia treatment will attenuate amnesia has not been examined. However, numerous types of manipulations, such as experience with similar training tasks, have been shown to alter the amnesic effects of hypothermia (Jensen, Riccio, & Gehres, 1975). Thus, an additional aim of this experiment was to determine if restraining rats in the tubes used for hypothermia treatment also would attenuate amnesia.

Method

Ten rats similar to those described in Experiments 1 and 2 received daily hypothermia treatments for 8 days. An additional group of 10 rats were restrained in hardware cloth cylinders and suspended in air. The time in tubes for the air-suspended group was approximately equal to the time of rats immersed in ice water.

On Day 9, each rat received a train-hypothermia treatment identical to that described in Experiment 1. Testing occurred 24 h after train-hypothermia. If a rat had not stepped into the black chamber within 15 min, it was assigned a test latency of 900.0 sec.

Results

Median train and test latencies for the prior-restraint rats were 18.0 and 434.4 sec, respectively. For the prior-hypothermia group, the respective train and test latencies were 18.0 and 632.0 sec. The short training latencies indicated that the prior experience manipulation had not generally produced an inactive rat.

The only significant differences found were between train and test latencies of each group, Ts(7) = 0, p = .02, indicating that amnesia was attenuated. An amnesia control group, i.e., rats which would receive only train-hypothermia, was not used in this experiment. Typically however, latencies of such "amnesic rats" from this laboratory have ranged between 9 and 130 sec (Hinderliter & Riccio, 1977; Riccio & Stikes, 1969). Furthermore, rats which had received hypothermia prior to train-hypothermia treatment in Experiment 1 had median test scores of 7.0 sec. These results of Experiment 3 indicate that amnesia was attenuated by both prior experience manipulations, where test latencies were greater than 400.0 sec.

As in Experiment 2, a weight loss was noted for rats receiving repeated hypothermia. Given that restraint rats failed to show a significant weight loss, it seems unlikely that performance artifacts correlated with weight loss were responsible for preventing amnesia.

Discussion

If the numerous types of prior experience manipulations are all different ways of inducing the same internal process(es) with respect to preventing amnesia, then sufficient exposure with the amnesic treatment should prevent amnesia, as does, for example, repeated exposure to the training apparatus prior to train-ECS (Lewis et al., 1968) or repeated train-ECS (Nachman & Meinecke, 1969). Typically, training and the amnesic treatments are unique events which subjects have experienced only once, and then in close association with each other. Both Hinderliter et al. (1975) and Thompson and Neely

(1970) have argued that, under such circumstances, the "cues" produced by the amnesic treatment are the most important in obtaining evidence of good retention. According to a contextual-cues interpretation, prior experience manipulations prevent amnesia because the train-amnesic treatment has been differentiated in that the "cues" produced by an amnesic treatment are not uniquely associated with the trained event. Thus, the amnesic-treatment-produced cues are not necessary for retrieval. The cues associated with the training event per se are sufficient for evidence of good retention to be obtained when training and the amnesic treatment have been differentiated. Considering the variety of prior experience manipulations shown to be effective in preventing amnesia, e.g., the prior restraint treatment used in this experiment, it does not seem likely that this protection from amnesia is due to prevention of the state-altering effects of amnesic treatments, as would have to be argued by Thompson and Neely (1970).

GENERAL DISCUSSION

The present experiments, in conjunction with several studies reviewed by Miller and Springer (1973), emphasize the role of retrieval mechanisms in producing retrograde amnesia. They also indicate that amnesic treatments may modify the way information is encoded for storage (and hence its retrievability) rather than disrupting storage or retrieval mechanisms per se. Hinderliter et al. (1975) have offered a contextual-cues hypothesis emphasizing the importance of retrieval cues in explaining retrograde amnesia.

According to this contextual-cues interpretation, the physiological insult produced by amnesic treatment does not prevent the process(es) necessary for an event to be stored or retrieved. Rather, the changes produced by the amnesic treatment are assimilated along with all other physiological events produced during the train-amnesic treatment manipulation. Because, in most amnesia studies, the training and the amnesic treatment are experienced only once, and then in close association with each other, the train-amnesic treatments are relatively undifferentiated experiences. Due to the extent of the physiological changes produced by the amnesic treatment, these changes become the most important cues necessary for retrieval. Also, it is assumed that these retrieval cues associated with the amnesic treatment dissipate relatively quickly. Thus, amnesia occurs at testing, usually 24 h after train-amnesic treatment, because the amnesic treatment cues are no longer present. Furthermore, it follows from a contextual-cues interpretation that any procedure that breaks the association of the train-amnesic treatment should prevent amnesia. For example, increasing the interval between training and the amnesic treatment should decrease the likelihood that these two events are assimilated together and thus amnesia is less likely to be produced. This temporal gradient effect has received extensive examination and is quite a robust phenomenon (Mah & Albert, 1973).

Results of Experiments 2 and 3, when considered along with numerous other reports, indicate that prior experience with almost any aspect of the train and/or amnesic treatment, when given a sufficient number of times, can attenuate amnesia. Possibly, these prior experience manipulations may break up or differentiate the train and amnesic events. According to a contextual-cues hypothesis, events similar to training or the amnesic treatment which can be remembered should be more than sufficient to differentiate train-amnesic treatments and thus prevent amnesia. Prior experience with hypothermia that is sufficient to produce evidence of learning can prevent amnesia, as was shown in Experiment 3.

An additional assumption of a contextual-cues hypothesis is that a critical number of cues associated with the train-amnesic treatment are necessary to produce good retention. DeVietti and Hopfer (1974) have shown that reexposing subjects to a sufficient number of training stimuli produced recovery from amnesia. These results are important in suggesting that exposure to training cues alone can increase retention performance of subjects which have received a train-amnesic treatment. Thus, the amnesic treatment cues are not the only contextual cues capable of restoring good retention performance. If sufficient cues of almost any aspect of the train-amnesic treatment are present during testing, evidence for amnesia may not be obtained. As the results of the present studies indicate, prior experience conditions sufficient to attenuate amnesia have been found. If an understanding of retrograde amnesia is to be obtained, it would be important to determine the necessary, as well as sufficient, conditions to attenuate retrograde amnesia. Such research is presently under way.

To summarize, the present findings indicate that experiential events, as well as physiological processes, are important in producing retrograde amnesia. This conclusion has important heurestic value. Much research has been undertaken to describe the physiological cause(s) of amnesia, e.g., brain seizure activity (Gehres, Randall, Riccio, & Vardaris, 1973). If the physiological processes initiated by amnesic agents are to be correlated with memorial processes, it is important to determine if these physiological events are altered by prior experience manipulations. If so, many questions logically follow—for example, to what extent are the physiological events altered by prior experience manipulations. If the prior

experience manipulations do not alter the physiological consequences of amnesic agents, yet amnesia is attenuated, the physiological events under investigation may be nothing more than epiphenomena. If such were the case, other types of changes also need to be monitored if a "physiological cause" of amnesia is to be described.

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