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Procedural Memory System Supports Single Cue Trace Eyeblink Conditioning in Medial Temporal Lobe Amnesia

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Abstract

A number of studies investigating trace eyeblink conditioning have found impaired, but not eliminated, acquisition of conditioned responses (CRs) in both animals and humans with hippocampal removal or damage. The underlying mechanism of this residual learning is unclear. The present study investigated whether the impaired level of learning is the product of residual hippocampal function or whether it is mediated by another memory system that has been shown to function normally in delay eyeblink conditioning. Performance of bilateral medial temporal lobe amnesic patients who had a prior history of participating in eyeblink conditioning studies was compared to a control group with a similar training history and to an untrained control group in a series of single cue trace conditioning tasks with 500 ms, 250 ms, and 0 ms trace intervals. Overall, patients acquired CRs to a level similar to the untrained controls, but were significantly impaired compared to the trained controls. The pattern of acquisition suggests that amnesic patients may be relying on the expression of previously acquired, likely cerebellar based, procedural memory representations in trace conditioning.

Keywords

associative learning; Pavlovian conditioning; bitemporal amnesia

It is generally well accepted that normal acquisition of trace eyeblink conditioning requires not only an essential contribution from the cerebellum (R. F. Thompson, 1986, 1988) but also from forebrain areas including the hippocampus. In the animal model, for example, Solomon and colleagues (Solomon, Vander Schaaf, Norbe, Weisz, & Thompson, 1986) reported that hippocampal lesions disrupt acquisition of conditioned responses (CRs) during trace conditioning. This finding was later confirmed by Moyer, Deyo and Disterhoft (1990) who demonstrated that acquisition was eliminated using a 500 ms trace interval (but not a 300 ms trace interval) with rabbits who had relatively complete hippocampectomies. Similarly, our

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laboratory has demonstrated impaired trace conditioning in humans using a 500 ms silent trace period in amnesic patients with bilateral medial temporal lobe damage, and little if any acquisition as the trace interval was extended to 1000 ms (McGlinchey-Berroth, Carrillo, Gabrieli, Brawn, & Disterhoft, 1997). Similarly, Woodruff-Pak (1993) demonstrated impaired acquisition in two amnesic patients (one being the patient H.M.), although both eventually achieved criterion performance with additional training. In the case of H.M., however, it is difficult to ascribe his impaired performance to the medial temporal lobe damage alone, as he also suffered from cerebellar degeneration of the vermis and hemispheres that likely contributed to his impaired delay conditioning. A nagging question in the human studies, and one that could also be asked of the animal studies is: what is mediating residual trace eyeblink learning in these cases of hippocampal system lesions?

In our original study, we speculated two possible sources of the impaired learning observed in amnesic patients: residual hippocampal function leading to some preserved declarative memory or intact cerebellar function that, when given enough practice, can acquire some level of trace conditioning via procedural learning (McGlinchey-Berroth, Carrillo, Gabrieli, Brawn, & Disterhoft, 1997). The current study is an attempt to distinguish between these two possibilities.

Five severely amnesic patients with bilateral hippocampal system damage were tested across three trace intervals: 500 ms, 250 ms, and 0 ms. Given the repeated measures design, an important factor to consider was the possibility of carry-over effects from one interval to the next. This consideration was heightened by the fact that the five patients had participated in other eyeblink conditioning studies as well; three were tested in four prior studies, one in two studies, and one in one prior study. Thus, two control groups were needed, one to control for prior history of eyeblink conditioning training upon entering the study and three additional subgroups of controls to evaluate normal performance at each interval without potential carry-over effects from any other interval within the current study. Our rationale in teasing apart the two explanations of residual learning was as follows. If amnesic patients' conditioning impairment (compared to controls with a similar history of eyeblink conditioning) lessened as the interval became shorter (i.e., going from trace-500 to trace-250 to trace-0), the source of preserved learning would likely be declarative memory mediated by residual hippocampal system function and they would show little, if any, deficit with a 0-trace interval. This is because a contiguous, but not overlapping, unconditioned stimulus (US) paired with a conditioned stimulus (CS) is essentially a delay paradigm that does not require an essential contribution from the hippocampal memory system (as demonstrated by (Gabrieli et al., 1995)). On the other hand, if we observed that amnesic patients' conditioning impairment remained stable across the trace intervals, and did not remit as the essential hippocampal contribution lessened, we would conclude that the source of preserved learning would likely be procedural memory and mediated primarily by their intact cerebellar-based learning system.

Method

Participants

Amnesic participants—Patients with bilateral damage to medial temporal lobe structures (MT amnesics) were recruited from area hospitals and referred by a neurologist to the Memory Disorders Research Center at the VA Boston Healthcare System. Five amnesic patients were tested, within subject, in the 500, 250, and 0 ms trace intervals. Four of the patients became amnesic as the result of an anoxic episode, and the remaining one from encephalitis. Bilateral damage to the hippocampal formation was confirmed by CT or MRI in all cases except one (see Table 1; PD had at CT a number of years ago but it is no longer available). All of the patients had participated in at least one previous eyeblink conditioning study (Carrillo et al.,

2001; Gabrieli et al., 1995; McGlinchey-Berroth, Brawn, & Disterhoft, 1999; McGlinchey-Berroth, et al., 1997).

The demographic and neuropsychological characteristics of the amnesic patients are presented in Table 1. All of the patients were severely impaired on delayed memory testing, as indicated by their poor recall performance on the Wechsler Memory Scale, Third Edition (WMS-III) and their poor recognition performance for verbal and nonverbal material on the Warrington Recognition Test (Warrington, 1984). However, the amnesic participants had preserved intellectual and attentional function, as indicated by their performance on the Wechsler Adult Intelligence Scale, Third Edition (WAIS-III).

Normal control participants—A total of 26 control participants were recruited with the help of the Harvard Cooperative Program on Aging, Boston, MA. All were screened to be free of any neurological disease or illness. Five of the control participants formed the Trained Normal Control (T-NC) group. This group served as the matched sample to the amnesic patients, was trained, within subject, on each of the trace intervals (i.e., 500, 250, 0), and had a similar history of prior eyeblink conditioning (Carrillo et al., 2001; Gabrieli et al., 1995; McGlinchey-Berroth, Brawn, & Disterhoft, 1999; McGlinchey-Berroth et al., 1997). They were further matched to the patients with regard to age ($M = 56.00$, $SD = 16.42$), education ($M = 14.71$, $SD = 2.36$), and verbal intelligence as measured by the WAIS-III ($M = 115.43$, $SD = 11.74$). T tests indicated that the MT amnesics and T-NC were equivalent on each of these measures (p 's > 0.14).

Twenty-one control participants formed the Untrained Normal Control (U-NC) group. This group was randomly assigned to one of three subgroups of seven participants each (U-NC 500, U-NC 250, U-NC 0). These subgroups served to assess normal trace eyeblink conditioning performance at each of the trace intervals without possible training or carryover effects. Each subgroup was matched to the amnesic patients with regard to age (U-NC 500: $M = 59.71$, $SD = 6.26$; U-NC 250: $M = 56.71$, $SD = 19.93$; U-NC 0: $M = 58.00$, $SD = 12.99$), education (U-NC 500: $M = 15.14$, $SD = 2.80$; U-NC 250: $M = 15.00$, $SD = 2.65$; U-NC 0: $M = 14.57$, $SD = 2.44$), and VIQ (U-NC 500: $M = 108.43$, $SD = 10.95$; U-NC 250: $M = 108.14$, $SD = 7.03$; U-NC 0: $M = 109.71$, $SD = 15.13$). T -Tests confirmed that the subgroups of U-NC's did not differ from the MT amnesic patients on any of these characteristics (p 's > 0.49).

Apparatus

The apparatus used was a modified version of that used for eyeblink conditioning in the rabbit (Akase, Thompson, & Disterhoft, 1994; L. T. Thompson, Moyer, Akase, & Disterhoft, 1994). The CS was an 85 dB, 1 kHz tone that was delivered binaurally over earphones for a period of 400 ms. The US was a 100 ms, corneal airpuff delivered to the right eye. The magnitude of the airpuff averaged 3 psi. Eyeblink responses were measured via surface electromyography (EMG) electrodes (Nicolet, NY) placed over the orbicularis oculi muscle of the right eye. These responses were subsequently filtered, integrated and rectified. An adjustable headband was worn to support the airpuff delivery nozzle.

Procedure

After providing informed consent, participants were seated in an upright chair and fitted with the eyeblink apparatus. The experimenter then read the following:

"Please listen carefully to the following instructions. Remain seated comfortably and look at the TV screen in front of you. Please try not to touch the headband or earphones at anytime during the experiment, however, if you feel uncomfortable or feel that you need to adjust anything please let me know and I will stop the experiment to make the adjustments.

You will hear and feel a series of stimuli as you watch the movie during the experimental session. These stimuli will consist of a tone and a puff of air. The puff of air will most likely make you blink your eyes. All you are asked to do for this experiment is to relax and watch the movie, if you feel like blinking please do so. Let your natural reactions take over.”

The experimenter was seated in the same room, but out of the direct view of the participant, and answered any questions as they arose. Each participant watched a Charlie Chaplin film during the testing sessions.

The MT and T-NC participants were tested in three conditioning sessions that were separated by at least two weeks. Trace interval was varied across sessions and was either 500, 250, or 0 ms in duration. All participants were tested in the 500 ms trace interval condition, followed by the 250 ms condition, and then the 0 ms condition. We felt that this was the most conservative order and understood that any carry-over effects would be beneficial during the shorter intervals. These specific intervals were chosen because they are relatively short intervals for humans and we have demonstrated that these intervals could be mediated by amnesics using nonhippocampal circuits (McGlinchey-Berroth et al., 1997), if in fact that is the underlying mechanism. The U-NC's were randomly assigned to one interval in order of their recruitment (to 500, 250, 0, respectively) and were tested throughout the conduct of the study.

Each conditioning session consisted of 60 conditioning trials and 30 extinction trials. The conditioning trials were composed of a 750 ms baseline recording period, followed by the tone CS for 400 ms. A silent trace interval then preceded a 100 ms corneal airpuff. In total, eye movements were monitored for 3000 ms. During extinction trials, the corneal airpuff was withheld. As in our previous studies, the intertrial interval during conditioning and extinction averaged 18 seconds, but varied randomly from 16 to 20 seconds (e.g., McGlinchey-Berroth et al., 1997).

Operational Definitions

In defining a CR in the current study, it was necessary to consider the fact that longer trace intervals increase the possibility that random blinks may occur during the CS-US interval and be included as CRs. This is a potential source of error in computing short vs. long intervals. Recent eyeblink conditioning studies have used a method reported by Spence and Ross (1959), who suggest that most true CRs occur within the final 300–400 ms before US onset (Finkbiner & Woodruff-Pak, 1991; McGlinchey-Berroth et al., 1997; Solomon, Blanchard, Levine, Velazquez, & Groccia-Ellison, 1991). We adopted this method and only recorded blinks that occurred in the 300 ms prior to US onset as CRs. This method corrected for both voluntary and random blinks that could occur as the result of the longer 500 and 250 ms trace intervals. Additionally, an eyeblink was only scored as a CR if it was four standard deviations greater than the mean baseline response amplitude. Finally, eyeblinks with a latency less than 100 ms following CS onset were recorded as alpha responses and not considered CRs (Gormezano, 1966) for all trace intervals.

Results

Group differences were explored in a series of one-way ANOVAs for mean percentage of CRs acquired (late onset), CR peak latency, and UR peak amplitude. One of the MT amnesics (PD) produced voluntary blinks (characterized by large square eye closures) on each of the first 15 trials during Trace 0, thus his data was not included in the analysis for that interval.

ANOVA for the mean percentage of CRs acquired revealed significant Group effects for all intervals and these were further analyzed using Fisher's PLSD. In Trace 500, the mean percentage of CRs for MT = 39.80 ($SE = 6.85$), T-NC = 74.80 ($SE = 6.85$), and U-NC = 29.76

($SE = 5.79$), $F(2, 17) = 13.18$, $p = .001$; power = .989. Post hoc tests revealed significant group differences between the MT and T-NC ($p = .003$) and the T-NC and U-NC 500 ($p < .001$). Similarly, in Trace 250, the mean percentage of CRs for MT = 41.67 ($SE = 9.38$), T-NC = 76.80 ($SE = 9.38$), and U-NC = 38.05 ($SE = 7.93$), $F(2, 17) = 5.61$, $p = .02$; power = .770. Post hoc tests again revealed significant group differences between the MT and T-NC ($p = .02$) and the T-NC and U-NC ($p = .007$). Lastly in Trace 0, the mean percentage of CRs for MT = 49.60 ($SE = 9.13$), T-NC = 75.20 ($SE = 9.13$), and U-NC = 39.14 ($SE = 7.72$), $F(2, 16) = 7.15$, $p = .008$; power = .86. Post hoc tests revealed significant group differences between the MT and T-NC ($p = .01$) and the T-NC and U-NC ($p = .004$). These data indicate that the MT amnesics and the U-NC performed equivalently, on average, across the three trace intervals, while the T-NC's reached a higher level of acquisition than the other two groups at each of the three intervals. This finding is shown clearly in Figure 1, which displays the mean percentage of CRs across the three trace intervals for each group.

The data were further broken out into six blocks of 10 trials each. MT amnesics and T-NC did not evidence learning curves as a function of blocks; their acquisition was rapid and relatively stable across blocks and trace intervals. For example, the MT amnesics actually exceeded their mean acquisition in the first block of Trace 500 (producing a CR on 48% of the trials), with performance levels then dropping back over the course of trials. Similarly, the T-NC produced a CR on 69% of the trials in the first block of Trace 500. In contrast, the U-NC showed a pattern of acquisition across blocks at each interval. In the trace 500 condition, the acquisition rate of this group rose from a mean percentage of 25 CRs in the first block to 34 in the second block, and 48 in the third block. In the trace 250 condition, the acquisition rate of this group rose from a mean percentage of 36 CRs in the first two blocks to 54 in the third block. Lastly in the trace 0 condition, the acquisition rate of this group rose from 31 in the first block to 37 in the second block, and 40 in the third block.

ANOVA for the mean CR peak latency did not reveal any significant differences as a function of group or block (p 's $> .49$). For Trace 500 the mean CR peak latency for MT = 848.06 (5.30), T-NC = 847.97 (4.58), and U-NC = 848.60 (4.24). For Trace 250 the mean CR peak latency for MT = 592.55 (4.76), T-NC = 602.41 (2.52), and U-NC = 597.07 (3.01). For Trace 0 the mean CR peak latency for MT = 347.76 (8.68), T-NC = 355.30 (3.29), and U-NC = 348.56 (3.74). UR amplitude did not produce any significant effects (p 's $> .12$). For Trace 500 the mean UR amplitude for MT = 42.63 (4.42), T-NC = 42.27 (16.92), and U-NC = 35.31 (7.82). For Trace 250 the mean UR amplitude for MT = 38.57 (3.32), T-NC = 39.91 (8.24), and U-NC = 23.93 (14.17). For Trace 0 the mean UR amplitude for MT = 29.89 (3.47), T-NC = 45.58 (11.63), and U-NC = 43.05 (9.73).

Discussion

In this study, patients with bilateral medial temporal lobe damage with dense amnesia acquired conditioned responses in a single cue task consistently across three different silent, brief trace intervals. Further, the acquisition of responses did not follow a learning curve, but rather occurred rapidly at each interval, consistent with the expression of *previously* learned responses, as opposed to relatively slow acquisition of newly learned memories. Precisely, the same pattern was observed in a group of control participants who had a similar history of previous training as the amnesics. However, the control group's performance was superior to the patients' at each trace interval. Compare these data to the control participants who were being conditioned for the first time. The acquisition level of the untrained control participants was similar to the MT amnesics (not significantly different at any of the trace intervals) but was significantly less than the trained control participants at each interval. Additionally, the untrained groups did evidence an acquisition curve characteristic of new learning, and while not significant, mean CR acquisition increased with shorter trace intervals.

When considered in total, this study clearly demonstrates that although MT amnesics can acquire CRs during single-cue trace conditioning at brief intervals, there is a ceiling to their optimal level of performance that keeps them from attaining truly normal performance. We interpret these data as indicating that the learning expressed by the MT patients is supported primarily by a procedural memory system that is cerebellar based and can form resilient memory traces that are long lasting. Importantly, this system would appear to be limited to relatively short trace intervals, as medial temporal amnesic patients cannot acquire trace eyeblink conditioning at intervals approaching 1000 ms (McGlinchey-Berroth et al., 1997). While normal individuals can use this same system to express previously learned associations (accounting for flat learning curves of the T-NC), we suggest that they also engage the intact declarative system that is hippocampally based and acts to enhance learning and provide a boost in performance.

This interpretation is consistent with the findings of Woodruff-Pak (1993) who reported that H.M. attained criterial learning two years after his first eyeblink conditioning sessions in only a fraction of the trials it took to acquire the learning initially. A recent study by Pakaprot, Kim, and Thompson (2006) found that the expression of previously acquired trace conditioned responses was reversibly abolished in rabbits when the interpositus nucleus of the cerebellum was rendered inactivated by muscimol. This finding suggested that the interpositus nucleus is essential for the long-term expression of trace conditioned responses and might similarly be responsible for the expression of preserved trace conditioned responses observed in this study.

It is suggested that the CRs generated by the amnesic patients were most likely expressions of procedural representations. However, it is important to keep in mind that the cerebellum is thought to be part of a more extensive integrated cerebellar-thalamic-prefrontal system that controls complex forms of eyeblink conditioning such as trace (e.g., Weiss & Disterhoft, 1996). We do not have any indication, either structurally, from imaging scans, or functionally from neuropsychological assessment, that other structures within this system such as the thalamus and medial prefrontal cortex were impacted in the amnesic patients. Therefore, we conclude that these additional forebrain structures may have contributed to the residual learning observed.

While we feel that this is the most parsimonious interpretation of the data, we cannot provide empirical evidence to rule out the possibility that some of the learning may have been mediated by declarative memory that is hippocampally based. It could be argued that the contribution of the hippocampal system could be exhibited in an all-or-none manner, whereby if the trace interval is short enough, performance will be normal and, if the interval is longer than some critical duration, performance will be impaired. However, we do not feel that this is likely because in this study and in the previous trace interval study (McGlinchey-Berroth et al., 1997), medial temporal amnesics have never attained normal performance relative to control groups with similar training histories.

These data underscore the complexities involved in conducting and interpreting behavioral experiments with neurological populations, even in the case of the most basic learning paradigm. The current data would suggest that patients with medial temporal lobe damage do not acquire CRs during trace conditioning in the same manner as neurologically normal individuals. Paradoxically, even though patients achieved a level of acquisition similar to untrained normal individuals, it appears that they did so through compensatory mechanisms, thus further supporting the idea that normal trace conditioning is hippocampally dependent. This helps in understanding the possible mechanism of residual learning that has been observed in previous human and animal studies showing impaired, but not eliminated, acquisition of trace eyeblink conditioning.

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Figure 1. Mean percent conditioned responses across trace intervals for Medial Temporal Amnesics (MT), Trained-Normal Controls (T-NC), and Untrained-Normal Controls (U-NC). Error bars represent standard error of the mean.

Table 1

Patient Demographic and Neuropsychological Characteristics

Patient	Age	Ed	Etiology	WAIS-III		WMS-III			Warrington	
				Verbal IQ	General Memory	Visual Delay	Auditory Delay	Working Memory	Words	Faces
PD	65	20	anoxia	111	52	56	64	83	26	26
RL	73	18	anoxia	113	75	72	80	102	35	33
JM	52	12	anoxia	83	52	56	55	91	31	33
PS	43	14	anoxia	90	45	53	52	93	33	29
SS	74	18	encephalitis	135	45	53	58	141	35	32
Means	60	16		106	54	58	62	102	32	31
SD	14.0	3.3		20.6	12.4	10.0	11.1	22.8	3.7	3.1

Note. The WAIS-III and the WMS-III scaled scores yield a normalized, age adjusted mean of 100. On the Warrington Recognition test, one point is scored for each of 50 items. Age and Ed (Education) are expressed in years. Means and standard deviations (SD) are reported at the bottom of each column.