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Perceptual Learning, Awareness, and the Hippocampus

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ABSTRACT: Declarative memory depends on the hippocampus and related medial temporal lobe and diencephalic structures. Declarative memory has usually been found to be available to conscious recollection. A recent study (Chun and Phelps, Nat Neurosci 1999;2:844–847) found that damage to the medial temporal lobe (including the hippocampus) impaired performance on a perceptual learning task, yet the learning was accomplished in the absence of memory for the stimuli. This finding raised the possibility that some hippocampus-dependent tasks may be inaccessible to awareness and may be performed without evoking conscious memory processes. Using the same task, we show that when damage is confined largely to the hippocampal formation, perceptual learning is intact. Thus, the available data suggest that damage limited to the hippocampal formation does not impair nonconscious (nondeclarative) memory. Further, the data do not contradict the idea that hippocampus-dependent memory is accessible to conscious recollection. Finally, perceptual learning was impaired in patients, with extensive damage to the medial temporal lobe and with additional variable damage to lateral temporal cortex. *Hippocampus 2001;11:776–782.*

KEY WORDS: memory; declarative; nondeclarative; amnesia; medial temporal lobe

INTRODUCTION

Memory is not a single faculty but is composed of several different abilities that depend on different brain systems (Schacter and Tulving, 1994; Squire and Zola, 1997; Gabrieli, 1998). Declarative memory supports the ability to acquire new facts and events and depends on the integrity of the hippocampus and anatomically related structures in the medial temporal lobe and diencephalon. Nondeclarative memory supports skill and habit learning, simple forms of conditioning, and other forms of learning that are expressed through performance rather than recollection. Nondeclarative memory is independent of the medial temporal lobe and diencephalic structures important for declarative memory.

A fundamental issue about these memory systems is what criteria, aside from anatomy, might usefully distinguish them. One criterion that has been useful is that declarative memory supports the flexible use of acquired knowledge, whereas nondeclarative memory is more closely tied to the original learning situation and is less accessible to other response systems (Cohen, 1984; Glisky et al., 1986a,b; Saunders and Weiskrantz, 1989; Eichenbaum et al., 1989, 1990; Reber et al., 1996).

A second criterion is that declarative memory typically includes knowledge, or awareness, about what has been learned. In contrast, nondeclarative memory does not require awareness of any conscious memory content, and when awareness is present it appears to be epiphenomenal to task performance (Reber and Squire, 1994; Clark and Squire, 1998). The availability of material to conscious recollection has been considered a key feature of declarative memory and has been supposed to be fundamental to the distinction between declarative (hippocampus-dependent) and nondeclarative (hippocampus-independent) memory systems (Tulving and Schacter, 1990; Tulving, 1991; Schacter, 1992; Squire, 1992; Eichenbaum, 1997; Gabrieli, 1998).

The link between awareness and hippocampus-dependent learning was questioned in a recent study of perceptual learning (Chun and Phelps, 1999). On each trial, healthy controls searched for a 90°-rotated, colored T among colored right angles, and indicated the direction in which its base was pointing. After practice, they searched repeated displays faster than new displays. Amnesic patients with damage in the medial temporal lobe (including the hippocampus) did not show this advantage for repeated displays. Interestingly, despite the fact that the task appeared sensitive to amnesia, task knowledge was not accessible to awareness. Neither controls nor amnesic patients could recognize which displays were repeated. These results suggest that learning can be dependent on the hippocampus yet inaccessible to awareness.

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TABLE 1.

Performance on Standard Memory Tests[a]

Patient	Group	Diagram recall	Paired associates			Word recall (%)	Word recognition (%)
			Trial 1	Trial 2	Trial 3		
A.B.	H+	4	1	1	1	33	83
H.C.	H+	7	0	4	5	37	85
L.J.	H+	3	0	0	0	40	93
M.J.	H+	7	3	5	6	44	81
P.H.	H+	3	0	0	1	31	81
E.P.	MTL+	0	0	0	0	24	65
G.P.	MTL+	2	0	0	0	35	54
G.T.	MTL+	0	0	0	0	20	70
Control M (n = 8)		20.6	6.0	7.6	8.9	71.0	97.4

[a]The diagram recall score is based on the delayed (12-min) reproduction of the Rey-Osterrieth figure (Osterrieth, 1944; maximum score = 36). The paired-associates score is the number of word pairs recalled on three successive trials (maximum score = 10 per trial). The word recall score is the mean percentage of 15 words recalled across five successive study-test trials (Rey, 1964). The word recognition score is the mean percentage of words identified correctly across five successive study and test trials (yes-no recognition of 15 new words and 15 old words). The mean scores for controls are from Squire and Shimamura (1986). H+, damage to the hippocampal region and, in H.C. and P.H., to the parahippocampal gyrus. MTL+, large medial temporal lobe lesions and variable damage to anterolateral temporal cortex.

A consideration of the neuropathology in the four amnesic patients who were tested suggests a different way to understand the findings. Two of the 4 amnesic patients sustained damage to the medial temporal lobe as a result of encephalitis and "showed some additional atrophy of the surrounding temporal lobe consistent with this etiology." (Chun and Phelps, 1999). Thus, for these 2 patients, the inability to benefit from the repeated displays may have been due, not to hippocampal damage, but to damage to the entorhinal, perirhinal, and parahippocampal cortices, or to the lateral temporal cortex. Further, radiological information (from a relatively insensitive CAT scan) was available for only 1 of the other 2 patients. Thus, it is unclear what damage was responsible for the impairment that these patients exhibited.

In another study (Ryan et al., 2000), participants viewed a scene and then viewed it again after a change had sometimes been made in one region of the scene. Controls, but not amnesic patients, tended to look more at the region of the scene in which the change had occurred. By one measure (number of transitions into and out of the critical region), the effect appeared to occur only for those controls who were unable to specify how the scene had changed. By another measure (proportion of fixations in the critical region), aware and unaware subjects performed similarly. It was suggested that awareness might not always be needed for the expression of declarative memory. No anatomical information was provided for the amnesic patients, so that it is unclear what particular damage might have been important.

We tested 8 amnesic patients on the same test of perceptual learning that was used by Chun and Phelps (1999). Five of the amnesic patients (H+) have damage that appears to be restricted either to the hippocampal region (CA fields, dentate gyrus, and subicular complex) or to involve some additional damage to the parahippocampal gyrus. The other 3 patients (MTL+) sustained extensive damage to the medial temporal lobe as a result of encephalitis and also have variable damage to the lateral temporal cortex.

METHODS

Five amnesic patients (4 men and 1 woman) with damage either limited to the hippocampal region (CA fields, dentate gyrus, and subicular complex) or with some additional damage to the parahippocampal gyrus were tested on the same task used by Chun and Phelps (1999). Twenty-three controls (8 men and 15 women) were also tested, 15 on the standard version of the task and 8 on a more difficult version (see below). The amnesic patients averaged 60.0 ± 6.4 years of age (mean ± SEM; controls = 66.9 ± 1.1) and 17.8 ± 1.7 years of education (controls = 15.3 ± .5), and obtained an average Wechsler Adult Intelligence Scale-III (WAIS-III) score of 102.8 (subscale scores of 19.3 for Information and 55.5 for Vocabulary; controls = 22.4 and 57.4, respectively).

The amnesic patients have moderately severe memory impairment. All require supervisory care and are unable to live independently. Table 1 shows their performance on standard memory tasks. Immediate and delayed (12-min) recall of a short prose passage (Gilbert et al., 1968) averaged 5.8 and 0.0 segments, respectively (maximum number of segments = 21). The 23 controls recalled 7.8 and 6.4 segments, respectively. The patients averaged 106.0, 71.4, 72.2, 64.6, and 51.6 on the five indices of the Wechsler Memory Scale-Revised (attention/concentration, verbal memory, nonverbal memory, general memory, and delayed memory,

respectively; each index yields a mean score of 100 in the normal population, with a standard deviation of 15). The memory impairment occurs in the context of otherwise intact cognition. For example, the patients performed well on the Dementia Rating Scale (mean = 132.0/144), losing points primarily on the memory subportion (mean = 10.4 points). They also performed normally on the Boston Naming Test (Kaplan et al., 1983), achieving a mean score of 57.6 (maximum possible = 60, range = 55–60).

For 4 of the 5 patients, their bilateral hippocampal damage was quantified by magnetic resonance imaging (MRI) (Fig. 1). The average area of the full anterior-posterior length of the hippocampus, as well as the average area of the parahippocampal gyrus and the temporal lobe, was measured in 1-mm-thick coronal sections (for the areas measured, see Squire et al., 1990). The parahippocampal gyrus and temporal lobe measurements began at the level of the head of the hippocampus and extended posteriorly 35 mm (for L.J., the measurements were based on 5-mm-thick sections and covered 30 mm of the temporal lobe). For each patient, the hippocampal and parahippocampal gyrus areas were divided by the area of the temporal lobe to equate for overall brain size. Relative to 10 age and gender-matched healthy controls ($n = 4$ for M.J. and P.H., $n = 3$ for L.J., and $n = 3$ for H.C.), H.C., L.J., P.H., and M.J. have a reduction in average hippocampal area of 27%, 46%, 30%, and 22%, respectively. In addition, the area of the parahippocampal gyrus for H.C. and P.H. was reduced by 25% and 30%, respectively (L.J. = 6%; M.J. = −5%). The fifth patient (A.B.), who is unable to participate in magnetic resonance imaging studies, became amnesic in 1976 after an anoxic episode following cardiopulmonary arrest and is presumed to have hippocampal damage on the basis of this etiology and a neurologic examination indicating well-circumscribed amnesia.

We also tested 3 additional male patients who developed profound amnesia after herpes simplex encephalitis (patients E.P., G.P., and G.T.). Table 1 shows their performance on standard memory tests. They averaged 64.7 years of age (range = 53–77), with an average of 13.3 years of education (range = 12–16). They averaged 93.3 on the WAIS-III (mean subscale scores = 10.3 for Information and 30.3 for Vocabulary). They averaged 105.3, 65.0, 68.0, 61.3, and 52.0 on the five indices of the Wechsler Memory Scale-Revised. Immediate and delayed (12-min) recall of a short prose passage (Gilbert et al., 1968) averaged 1.7 and 0.0 segments, respectively (maximum number of segments = 21).

For these 3 patients, MRI studies revealed extensive and nearly complete damage to the medial temporal lobe, including the hippocampal region, the amygdaloid complex, and the entorhinal, perirhinal, and parahippocampal cortices (Fig. 1). Variable damage is also present lateral to these structures (Schmolck et al., 2000b; Stefanacci et al., 2000). E.P.'s damage is primarily medial temporal but also involves the anterior aspect of the fusiform gyrus. The lateral temporal cortex and the insula are also somewhat reduced in size bilaterally (lateral temporal lobe = 15% reduced in

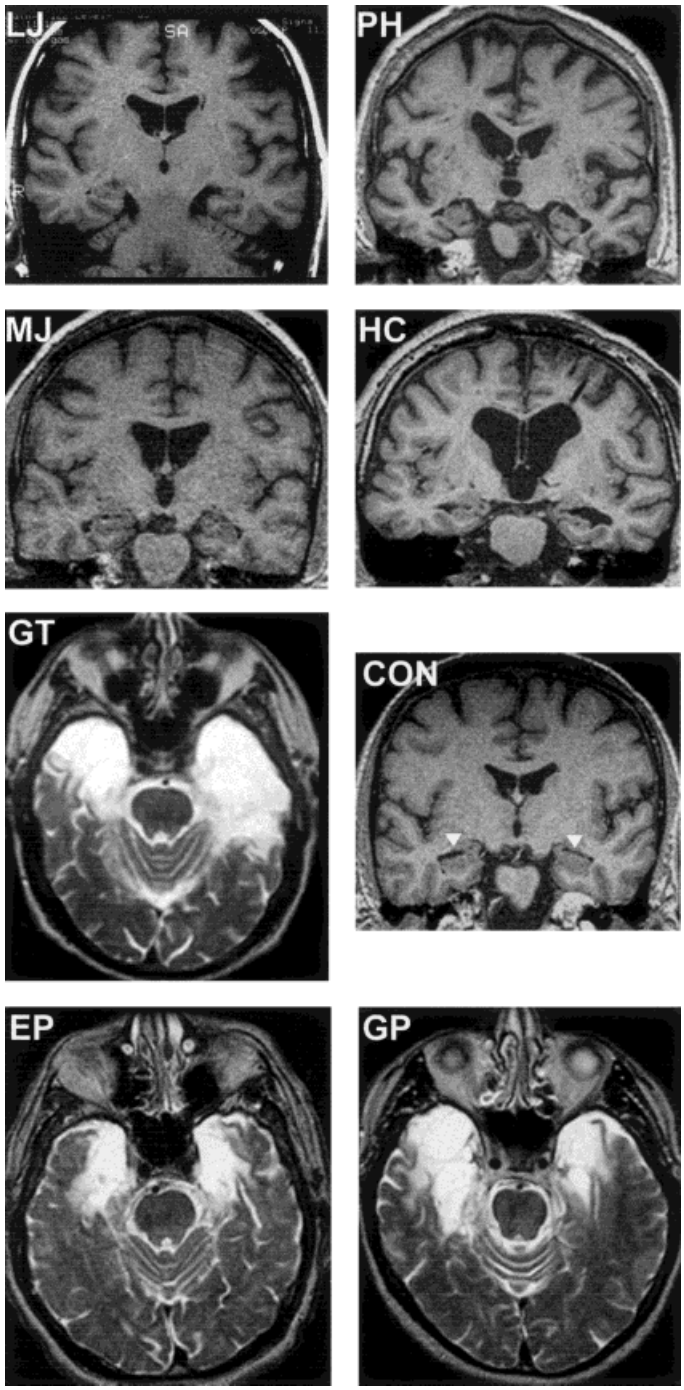

FIGURE 1. Magnetic resonance images for 4 H+ patients (LJ, PH, MJ, and HC), 3 MTL+ patients (GT, EP, and GP), and one healthy volunteer (CON). For the H+ patients and the volunteer, the images are T1-weighted coronal sections at the level of the mammillary nuclei (PH, MJ, HC, and CON) or approximately 1 cm posterior to the mammillary nuclei (LJ). The left side of the brain is on the right side of the image. In comparison to age- and gender-matched controls, LJ, PH, MJ, and HC exhibited a reduction in area for the hippocampal region of 46%, 30%, 22%, and 27%, respectively (hippocampal area/temporal lobe area; see text). White triangles on the image for the volunteer indicate the hippocampal region. For the MTL+ patients, the images are T2-weighted axial sections at the level of the temporal lobe. The left side of the brain is on the left side of the image. Damaged tissue is indicated by bright signal. For the MTL+ patients, damage includes virtually the entire medial temporal and variable damage to anterolateral temporal cortex (see text).

volume; insula = 12% reduced in area; Stefanacci et al., 2000). G.P. and G.T.'s damage extends further laterally. G.P.'s damage includes the fusiform gyrus as well as the inferior, medial, and superior temporal gyri. More caudally, the damage is limited to the fusiform gyrus and the inferior temporal gyrus. G.T. has the most severe damage, which extends laterally to involve the anterior 5.0 cm of his right temporal lobe and the anterior 7.0 cm of his left temporal lobe.

The task was as described by Chun and Phelps (1999) and was approved by the Human Subjects Committee at the University of California, San Diego. After giving informed consent, participants were seated in front of a computer screen at a viewing distance of approximately 60 cm. On each trial, participants saw 11 colored, L-shaped distractors and one colored, 90°-rotated T target. When participants pressed the space bar to initiate each trial, a small dot appeared in the middle of the computer screen, which was replaced after 500 ms by the stimulus array. Participants located the T target as quickly as possible and pressed one of two labeled keys to indicate the direction (left or right) in which its base was pointing. Pressing a key cleared the screen, and feedback was given to signal whether the response was correct or incorrect. Reaction times for targets correctly identified in less than 6 s were used in statistical analyses. The test session was preceded by 24 practice trials.

Trials occurred in sets of 24, with a 10-s pause after each set. Each set contained 12 new and 12 old displays, randomly intermixed within each set. Forty sets of 24 trials were given (960 trials), such that the same 12 old displays were repeated 40 times each (480 trials), and a total of 480 new displays was also presented. The background was gray, and each display contained an equal number of red, green, blue, and yellow items. Each color was assigned to an equal number of targets in the new displays and in the old displays. Targets and distractors were arranged in a grid of 6 by 4 possible locations. The target appeared equally often in one of these 24 possible locations: 12 locations were used for the 12 old displays, and the other 12 were used for the new displays. For new displays, the base of the T-shape pointed either to the left or to the right, and the distractors were randomly rotated at 90° intervals. For each repetition of the 12 old displays, the direction of the target was randomly assigned, but in all other respects these 12 displays preserved their appearance across the 40 times they were presented.

Midway through the session (after 480 trials) and again at the end of the session, participants were given a yes-no recognition memory test about the repeated displays. Participants were first asked, "Did you notice whether certain configurations (spatial layouts) of the stimuli were being repeated from block to block?" Participants pressed one of four keys labeled "pretty sure no," "maybe no," "maybe yes," or "pretty sure yes." Participants were then presented with the 12 old displays and 12 brand new displays, which were intermixed and presented one at a time. For each display, participants were asked to indicate whether the display was old or new by responding "pretty sure new," "maybe new," "maybe old," and "pretty sure old."

The procedure for the more difficult version of the task, given to 8 controls, was the same as for the standard version, except that the distractors were made to appear more similar to the targets (Chun and Phelps, 1999). This modification had the effect of making the target more difficult to locate, thus increasing reaction times.

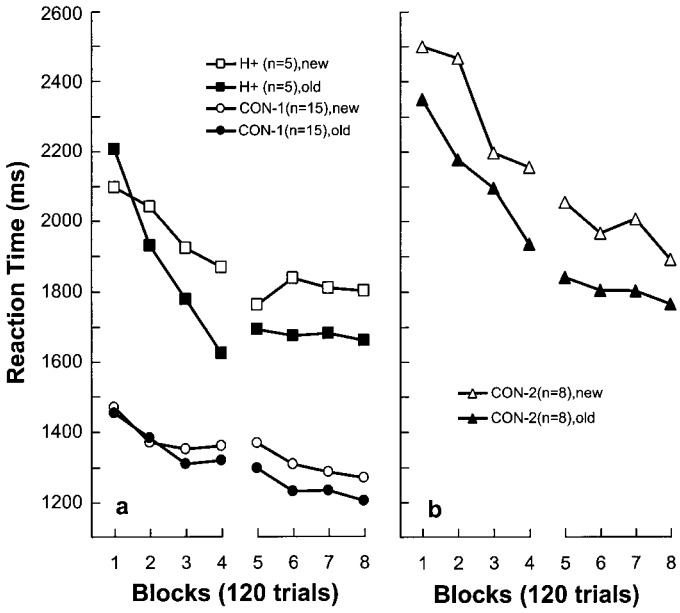

FIGURE 2. Reaction time (for correct trials) required to report the orientation of a T target among right-angle (L-shaped) distractors. All groups exhibited perceptual skill learning, as evidenced by overall improvement in reaction time across the session. a: Amnesic patients with hippocampal lesions (H+) and controls (CON-1) also benefited from repetition of displays, and searched repeated (old) displays faster than new displays. b: A more difficult version of the task slowed search speed for controls (CON-2) to about the level of amnesic patients. In this condition, the difference in reaction times between repeated (old) and new displays was similar for controls and amnesic patients.

RESULTS

Participants were very accurate at determining the direction of the rotated T target (controls, mean = 99.0% correct; H+, 98.5%; and MTL+, 98.9%). Figure 2 shows the time required to indicate the direction of the T target across the eight blocks (960 trials) of the testing session. The 5 amnesic patients with damage to the hippocampal formation (H+) exhibited the same pattern of reaction times as controls (Fig. 2a). First, amnesic patients and controls exhibited an overall improvement across the session (main effect of block, $F[7,4] = 2.58$ and $F[7,14] = 7.44$, for patients and controls, respectively; both $P < 0.05$). Second, both groups searched repeated displays more quickly than new displays (main effect of display type, $F[1,4] = 22.67$ and $F[1,14] = 4.23$, for patients and controls, respectively; both $P < 0.05$).

Chun and Phelps (1999) gave four blocks of trials (480 trials) to their participants and focused on the reaction times for new and old displays in blocks 3 and 4 (trials 241–480). Their controls searched repeated displays faster than old displays on these trials, but the 4 amnesic patients they tested did not. In addition, in blocks 1–4, their controls exhibited an interaction of block × trial type (new vs. old), but their amnesic patients did not. In contrast, our amnesic patients resembled the controls tested by Chun and Phelps (1999) (and the controls tested in the present study). First, for blocks 3 and 4 (240 trials), each of the five H+ patients tested

here was at least 51 ms faster for old displays than for new displays (mean new-old difference ± SEM = 193 ± 56 ms; $t[4] = 3.43$, $P < 0.05$). Second, in blocks 1–4, the H+ patients exhibited an interaction of block × trial type (new vs. old) ($F[3,12] = 3.45$, $P = 0.05$). We also found that during blocks 3–8 (the final 720 trials), each of the 5 amnesic patients averaged at least 60 ms faster on repeated displays than on new displays (mean new-old difference ± SEM = 149 ± 43 ms; $t[4] = 3.52$, $P < 0.05$).

The 5 H+ patients were slower on average than controls, and also displayed a larger difference in reaction times between the new and old displays (blocks 3–8, $t[18] = 2.11$, $P < 0.05$). Accordingly, the larger new-old effect exhibited by the amnesic patients might be related to their slower overall reaction times. To test this possibility, we gave 8 new controls a more difficult version of the search task, also used by Chun and Phelps (1999), in which the L-shaped distractors were more similar to the T target (Fig. 2b). The average difference in reaction times between the new and old displays on this task was similar to that of the amnesic patients (mean new-old difference for blocks 3–8 ± SEM = 149 ± 42 ms and 171 ± 49 ms, for patients and controls, respectively). Thus, it appears that the relatively large new-old difference displayed by the amnesic patients was commensurate with their overall reaction time.

The amnesic patients and the controls (CON-1) were very poor at recognizing the repeated displays. Both groups (H+ and CON-1) performed at chance overall on the yes-no recognition test given after block 4 as well as on the test given at the end of the session (mean percent correct ± SEM = 51.7 ± 3% and 51.7 ± 2% for CON-1; and 50.8 ± 2% and 51.7 ± 2% for the amnesic patients; one-sample *t*-tests, all $P > 0.1$). Moreover, neither group gave any sign of recognition when the score was based only on the items rated "pretty sure old" and "pretty sure new" (51.7 ± 2% and 44.8 ± 7% for controls and patients, respectively). However, the controls tested on the more difficult version of the task (CON-2) did perform above chance on the recognition task after block 4 (59.5 ± 3%, $t[7] = 3.21$, $P < 0.05$), though not at the end of the session (54.3 ± 4%, $t[7] = 1.07$, $P > 0.1$). The 16 controls (from CON-1 and CON-2) who believed displays had been repeated did not score significantly better on the two recognition tests than the 7 controls who did not believe displays had been repeated (54.3 ± 2% vs. 51.5 ± 3% correct, respectively; $t[21] = 0.90$, $P > 0.1$). Finally, 2 patients with H+ damage believed that displays had been repeated ("pretty sure yes") but did not perform differently than the other 3 patients.

We next tested 3 patients (MTL+) with profound amnesia following viral encephalitis, who have extensive damage to the medial temporal lobe as well as damage that extends variably into the lateral temporal lobes. Like the patients with damage limited to the hippocampal formation, these patients showed an overall improvement across the session ($F[7,2] = 4.79$, $P < 0.01$; Fig. 3). However, unlike the other patients (but consistent with the findings of Chun and Phelps, 1999), the MTL+ group did not benefit from the repetition of displays ($F[1,2] = 7.78$, $P > 0.1$). Indeed, for blocks 3–8, all 3 patients were nonsignificantly slower for old displays than for new displays (mean new-old difference ± SEM = −35 ± 21 ms). Finally, these 3 patients were unable to recognize the repeated displays on the two recognition tests (mean percent correct ± SEM = 45.0 ± 6%; $t[2] = 0.97$, $P > 0.1$).

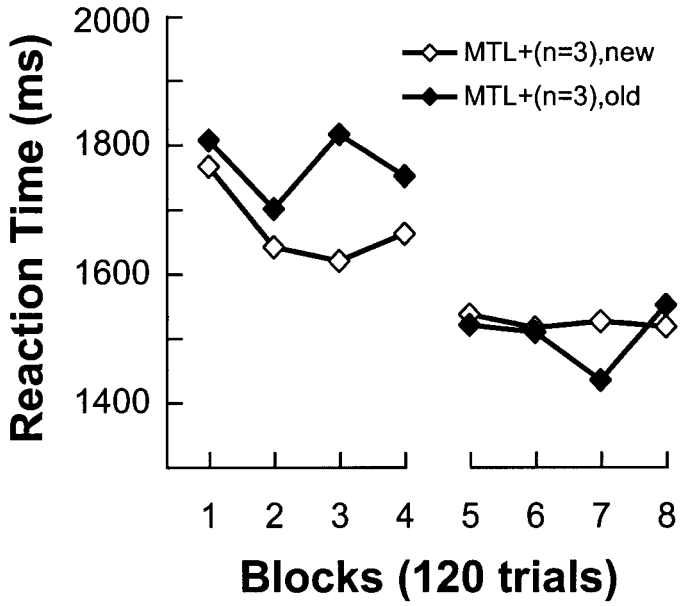

FIGURE 3. Reaction time (for correct trials) required to report the orientation of a T target among right-angle (L-shaped) distractors for amnesic patients who sustained large medial temporal lobe lesions together with variable damage to lateral temporal cortex (MTL+). These patients exhibited overall improvement in search speed across the session, but did not search repeated displays faster than new displays.

DISCUSSION

All participants exhibited perceptual learning across the test session, and none were able to recognize consistently which displays had been repeated and which were new. Thus, as described by Chun and Phelps (1999), the task appears to depend on implicit (or nondeclarative) memory. Importantly, amnesic patients with damage thought to be largely restricted to the hippocampal region (H+) benefited from the repetition of displays. Like the controls, these patients were faster at locating the target in repeated displays than in novel displays (Fig. 2a). Thus, contextually specific perceptual learning is not dependent on the hippocampus.

In contrast, the MTL+ patients, who have extensive damage to the medial temporal lobe together with variable damage to the lateral temporal cortex, did not benefit from the repetition of displays. There are several possibilities as to why the MTL+ patients were impaired on this task while the H+ patients were not. First, the MTL+ patients may have been impaired as a result of the extensive damage to the entorhinal, perirhinal, and parahippocampal cortices. Second, the impairment might have resulted from lateral cortical damage. Third, given that the MTL+ patients had

virtually complete hippocampal damage, whereas the H+ patients had partial hippocampal damage, it is difficult to exclude entirely the possibility that the task is in fact dependent on the hippocampus and that spared hippocampal tissue in the H+ patients allowed them to perform the task in a normal way.

In the absence of patients with extensive damage restricted entirely to medial temporal lobe structures and patients with damage restricted to the lateral temporal cortex, we cannot determine which of these regions is critical for normal performance (or if both regions are critical). However, we believe it unlikely that the task depends on the integrity of the hippocampus. First, the new-old difference exhibited by the H+ patients was *greater* than the new-old difference exhibited by controls given the same version of the task (Fig. 2a). Further, the performance of H+ patients was very similar to the performance of controls given the more difficult version of the task (Fig. 2b). Thus, there was no indication in the behavioral data that the hippocampal lesions in these patients impaired their ability to benefit from the repetition of displays. Second, the percent reduction in the hippocampal region (average = 31%) for the 4 H+ patients with MRI-confirmed hippocampal damage likely reflects widespread neuronal loss in the hippocampus. For 2 previous cases where similar reductions in hippocampal area had been documented by structural imaging (L.M. and W.H.), postmortem histological analysis revealed neuronal loss in all hippocampal cell fields (Rempel-Clower et al., 1996). Thus, a 50% reduction in hippocampal area should not be taken to mean that 50% of hippocampal neurons are intact. At the same time, only postmortem analysis can indicate conclusively how much damage has occurred.

We also considered what difference might exist between the MTL+ patients who were impaired and the H+ patients who performed normally. Do the patients differ only with respect to the severity of their memory impairment, or are there other differences as well? We recently described impairments in the 3 MTL+ patients that are not exhibited by patients with more limited damage to the hippocampal region. In particular, the 3 MTL+ patients have difficulty detecting and explaining ambiguity in sentences (Schmolck et al., 2000b), they have difficulty in perceiving emotion in faces (Schmolck and Squire, 2001), and they have mild impairments in tasks of semantic knowledge about living and nonliving things (Schmolck et al., 2000a). Thus, the 3 MTL+ patients do exhibit more than severely impaired memory. Either severe memory impairment or these other cognitive defects (or both) could have contributed to the impairment reported here.

It is interesting to note that one of the anoxic patients tested by Chun and Phelps (1999) was subsequently described as having damage restricted to the "hippocampus proper" (patient P.S. in Verfaellie et al., 2000). Unlike our 5 H+ patients, this patient did not search repeated displays faster than novel displays. However, 3 of the 15 controls tested in the present study (Fig. 2a) also did not search repeated displays faster than novel displays. Thus, the finding that 1 out of 5 patients with MRI-confirmed damage restricted to the hippocampal region (P.S. and 4 of our H+ patients) did not search repeated displays faster than novel displays might be expected from the variability noted in the performance of healthy individuals. In contrast, 5 of 5 postencephalitic patients (2 from the previous study, who on the basis of etiology likely had extensive medial temporal lobe damage, plus the 3 MTL+ patients from the present study) did not search repeated displays faster than novel displays. Thus the impairment observed in the postencephalitic patients is unlikely to be attributable to variability.

In summary, in agreement with previous work (Chun and Phelps, 1999), the present results show that the memory that allows individuals to find targets embedded in repeated displays faster than targets embedded in novel displays is nondeclarative and unavailable to conscious recollection. The present findings also suggest that this form of memory is independent of the hippocampus. Accordingly, the findings suggest that damage to the hippocampus does not impair nonconscious (nondeclarative) memory. In this sense, the data support the link between the hippocampus and conscious (declarative) memory. Further studies are needed to identify the relative importance of the medial temporal cortex adjacent to the hippocampus and lateral temporal cortex in this task of perceptual learning. Studies in the monkey, where the locus and extent of damage can be relatively well-controlled, may be especially helpful.

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