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INTACT PRIMING OF PATTERNS DESPITE IMPAIRED MEMORY*

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Abstract—The priming of patterns was examined in normal control subjects (NCS) and in the amnesic patient H.M., whose anterograde amnesia followed bilateral medial temporal-lobe excision 33 years earlier. Despite H.M.'s impaired recognition of the patterns, he demonstrated a pattern priming effect equivalent in magnitude to that of the NCS. The results demonstrated that intact priming with novel, nonverbal material can occur despite severe amnesia. Pattern priming may reflect adaptive visual processes involved in learning novel perceptual organizations of known visual codes, such as letters, words, shapes, and objects.

INTRODUCTION

PRIMING refers to the influence of prior processing of stimuli upon later performance with those stimuli. For example, exposure to a word in a study list increases the likelihood that normal and amnesic subjects will complete a 3-letter stem to that word [10, 11, 20, 45], will produce that word when listing members of a category [15, 19, 22], and will identify that word when it is presented very briefly [2, 4, 13] or when it emerges from visual noise [11, 45]. Remarkably, the magnitude of these priming effects is normal (intact) in amnesic patients despite their impaired recall or recognition of the study list words. The presence of intact priming in amnesia indicates that priming can be mediated by memory systems that are separable from those that subserve recall and recognition: a memory system is defined as the minimal neural network required to record, retain, and retrieve a form of knowledge.

Priming can be demonstrated on a broad range of tasks and is probably not a unitary phenomenon. Nevertheless, most demonstrations of intact priming in global amnesia have used verbal tasks, such as reading, spelling, or completing letters into words. There are, however, a few examples of priming in amnesia that do not involve words as stimuli. After amnesic patients identified incomplete line drawings of animals and common objects, they demonstrated considerable improvement when reidentifying the incomplete drawings an hour or more later [26, 44]. A similar finding was reported in eight amnesic patients (seven of

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whom had closed-head injuries) who reduced greatly their times to detect hidden figures in picture puzzles that they had studied a day earlier [9]. Three amnesic patients also improved their ability to find incongruous features in complex cartoon-type drawings taken from the McGill anomalies task [46]; the improvements were maintained for as long as 4 days.

These demonstrations of nonverbal priming in amnesia are intriguing, but their meaning remains elusive for several reasons. First, in no case was the priming demonstrated as being intact in the amnesic patients (i.e. as being equal to that of control subjects). Priming in the amnesic patients was either less than that seen in the control subjects [26, 44] or not compared to that of control subjects [9, 46]. Second, no tests of explicit recall and recognition of the nonverbal stimuli were given in those studies, precluding a rigorous dissociation between nonverbal priming and recall/recognition memory. It is possible that given a multiple-choice or yes/no recognition test, the amnesic subjects in the earlier studies would have shown a recognition impairment comparable to their priming impairment. MILNER *et al.* [26] hypothesized that the small deficit in priming shown by amnesic patients was due to their being restricted to using perceptual memory while normal subjects could consult perceptual memory and explicit verbal memory of the prior testing session. Although Milner's hypothesis is plausible, it remains unknown whether nonverbal priming can be fully intact in amnesia and can be dissociated rigorously from recall and recognition memory.

The status of nonverbal priming in amnesia is important for theories about the nature of preserved priming capacities. In attempting to elucidate the processes that support verbal priming, investigators have explored the boundary conditions for intact priming effects in amnesia [33, 34, 39]. Evidence exists that intact priming can occur across modalities and without physical similarities between the primed word and the stimulus that evokes access of the primed word [19, 41]. Amnesic patients, however, have failed to show normal priming for nonwords on some tasks [4, 10, but see 2, 11, 13]. One explanation for this finding is that the nonwords cannot be known premorbidly by the patients, and that their anterograde memory impairments prevent them from gaining a lexical or semantic representation of newly seen nonwords. If priming depends upon the activation of an established representation (e.g. [27]), it may be hypothesized that amnesic patients can show intact priming only when they have premorbid knowledge of the primed response, i.e. when they possess a normal initial representation of that stimulus. It is easy to identify knowledge of words as being the basis of verbal priming, but is less clear what could constitute a knowledge base for nonverbal priming.

The present study sought to discover whether an amnesic patient could show normal priming with stimuli that were patterns and that lacked premorbidly established mental representations of the sort that appear necessary for verbal priming. Dot-pattern stimuli were used that could be organized or parsed perceptually to suggest a variety of figures (Fig. 1). We postulated that the perceptual interpretation of a dot pattern depended upon flexible visual processes that could be influenced by prior experience with a particular figural organization of the dot pattern. If such priming effects were to occur in normal control subjects (NCS), it would be possible to compare their priming effects to those of an amnesic patient who, unlike the NCS, would be unable to remember the figures that were biasing the later perceptual organization of the dot patterns.

Priming Study 1 examined whether experience with a figure primed (above a baseline or control condition) the perceptual organization of a dot pattern in NCS and in the amnesic patient H.M. Priming Study 2 was an extension of Study 1 that employed a different method of establishing a baseline. Also, a recognition memory study was conducted in order to establish whether H.M. could remember the figures (i.e. the source of the priming) at the time that the status of the priming effect was measured.

METHOD

Subjects

The amnesic patient H.M. (aged 61 or 62 years in the several testing sessions, with 12 years of education) and 15 NCS participated in this study. H.M. underwent bilateral medial temporal-lobe resection in 1953, at age 27, in an attempt to alleviate his otherwise intractable epilepsy [37]. The surgical removal was estimated to extend 8 cm back from the tips of the temporal lobes, including the prepyriform gyrus, uncus, amygdala, hippocampus, and parahippocampal gyrus. Temporal neocortex was reported to have been spared. Since the resection, H.M. has had a severe and pervasive anterograde amnesia for verbal and nonverbal materials in all modalities [8, 38]. H.M. took part in 4 testing series that were at least 2 months apart: Priming Study 1 in April, 1986; the recognition-memory tests in February, 1987; a replication of Priming Study 1 in April, 1987; and Priming Study 2 in November, 1987.

In Priming Study 1, five men and six women constituted the NCS group, which had a mean age of 56.8 years (range 49–65) and a mean educational level of 13.9 years (range 11–18). In Priming Study 2, two men and one woman constituted the NCS group; they had a mean age of 59.7 years (range 50–74) and a mean educational level of 15.7 (range 14–17). For the test of recognition memory, two women and one man constituted the NCS group; they had a mean age 66.3 years (range 57–82) and a mean educational level of 13.7 (range 12–17).

Materials

The stimuli, taken from GARNER [16], were derived from six dot patterns, each consisting of five dots from the nine possible dots in a 3×3 square matrix (Fig. 1). For each dot pattern, three target figures were constructed by connecting the five dots with straight lines. Three 6-item priming test forms were made by randomly selecting one target figure for each dot pattern. Explicit recognition memory for the figures was measured using 4-choice recognition tests. There were three recognition memory tests, each of which was paired to one of the priming tests in that the same target figures were used for the corresponding priming and recognition tests. For each recognition item, a dot pattern was shown at the top of the page and four figures that connected the dots were shown at the bottom. One figure was the target and three were foils.

Procedure

For Priming Study 1, there were four test sessions. In Session 1, subjects were asked to draw any figure onto each dot pattern that connected all the dots with straight lines. The figures that subjects drew constituted their baseline figures. In Session 2, which always occurred at least 6 hr later, subjects were asked to copy six target figures from one form onto corresponding dot patterns (each figure was above the corresponding pattern on an $8\frac{1}{2} \times 11$ in. piece of paper). Then, the materials were removed, and subjects were asked to write down the names of as many famous entertainers from the 20th-century as they could; this interference task was done for 3 min. Next, subjects were given the dot patterns (without any lines) and asked to draw any figure that connected the dots in each pattern with straight lines. In Session 3, the same procedure was followed except that a second, different set of target figures were copied onto the same dot patterns, and subjects were asked to write down the names of as many famous political figures from the 20th-century as they could for the interference task. In Session 4, the third set of target figures were copied onto the dot patterns, and subjects were asked to write down the names of as many athletes from the 20th-century as they could for the 3-min interference task. Unlike Sessions 2 and 3, subjects were then asked to draw the same figures they had just copied onto the dot patterns. Thus, Session 4 provided a measure of cued recall (the dot patterns being the cues) of incidentally encoded figures. The three test forms were administered in the same order for all subjects, with a day between Sessions 2, 3 and 4. H.M. participated in all four sessions on each of two separate occasions, a year apart.

The protocol was modified for Priming Study 2, which was conducted 6 months after Study 1. As before, subjects drew the first figure that came to mind on each of the dot patterns. These figures served as baseline figures. Then, at least 6 hr later, subjects copied target figures (from the same set used in Priming Study 1) that had been selected on the basis of their not being drawn in the baseline session. This procedure ensured that none of the target figures were an individual subject's primary, or spontaneous, perceptual organization of the dot patterns. The rest of the test (copy figures, the interference task, and drawing figures onto dot patterns) was administered as described above.

For the separate test of recognition memory, subjects copied one set of target figures onto dot patterns. Following the same 3-min delay task used in the priming test, subjects were asked to indicate which one of the four figures on the bottom of each page they had copied earlier onto the dot pattern displayed at the top of each page. Each of the NCS did this task once with six target figures; H.M. did this task with 18 target figures, 6 per session in each of 3 sessions.

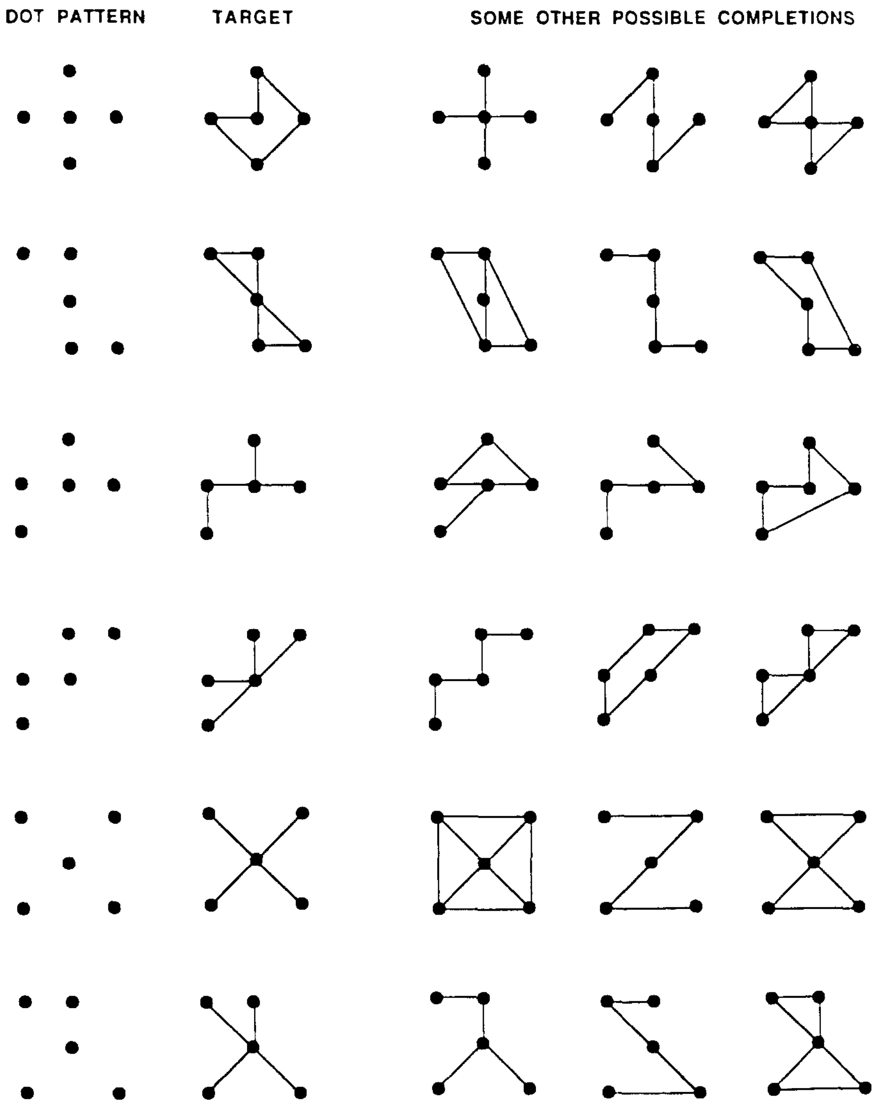

Fig. 1. Dot patterns are shown in column one. Examples of target figures copied onto dot patterns (i.e. primed figures) are shown in column two. Some other figures that could be drawn onto dot patterns are shown in the remaining columns.

RESULTS

Priming

In order to determine whether the control subjects demonstrated priming in Study 1, paired comparisons were made between the number of target figures drawn in the baseline condition and the number drawn in the primed (post-copying) condition. In baseline Session 1, the control subjects drew randomly a mean of 0.27 (SD, 0.47) target figures used in Session 2 compared to mean of 1.27 (SD, 0.64) target figures following the copying in Session 2. This difference was significant ($t = 5.24$, d.f. $= 10$, $P < 0.01$, one-tailed), indicating that copying a target figure onto a dot pattern increased the probability of its subsequently being drawn freely onto the same dot pattern. In baseline Session 1, the control subjects drew randomly a mean of 1.18 (SD, 0.75) target figures used in Session 3 compared to a mean of 1.91 (SD, 1.04) target figures following the copying in Session 3. This difference also was significant ($t = 1.90$, d.f. $= 10$, $P < 0.04$, one-tailed). Thus, control subjects showed significant priming in both Sessions.

In order to compare the status of priming in H.M. to that of the control subjects, a strict scoring method provided individual priming scores for Sessions 2 and 3. If a target figure was drawn by a subject in the baseline condition, that figure was discounted for that subject. Thus, each subject served as his or her own control. The priming score was the percentage of remaining target figures drawn identically to the copied figures in Sessions 2 and 3. The control group had a mean score of 20.8% (SD, 15.9). H.M. had, over four separate sessions, a mean priming score of 28% (Fig. 2). Other scoring methods, including scoring by line segments, produced the same finding of intact priming in H.M. In Priming Study 2, subjects again served as their own controls because the target figures were selected to be different from those drawn in the baseline condition. The priming score was the percentage of target figures drawn identically to the copied figures. The control groups's mean score was 27%; H.M. scored 33%.

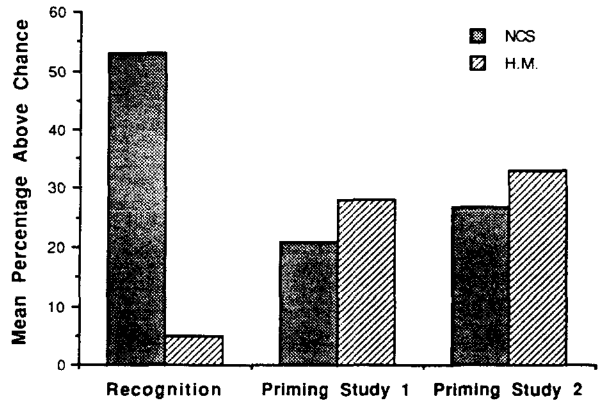

Fig. 2. Results of the recognition memory test, Priming Study 1, and Priming Study 2, showing mean percentage correct above chance for normal control subjects (NCS) and H.M.

Inspection of H.M.'s priming performance across the different sets of target figures and the two different protocols (Priming Study 1 and Study 2) provides further evidence in favor of his showing a reliable priming effect. H.M.'s priming scores reflected 10 instances of his primed drawing of seven different target figures. He primed once on five of the figures, two times on another figure, and three times on another figure. For two of the dot patterns, he drew different primed figures on the same dot pattern depending on the particular target figure that he had copied in that session. He drew only one of the seven primed target figures (a figure he primed on once) in the 18 baseline trials (3 trials per pattern) that were given at the start of the three experimental series (Study 1 twice and Study 2). Restated in terms of percentages, H.M. randomly drew 6% of the target figures that he subsequently primed on, vs priming scores of 28 and 33% in Studies 1 and 2, respectively. He demonstrated 90% of his priming on figures that he never drew spontaneously. Finally, H.M. showed priming in all five separate testing sessions.

Cued recall and recognition memory

The cued recall scores of incidentally encoded target figures from Session 4 (Study 1) were calculated in the same way as the priming scores, i.e. a target figure was counted as being recalled correctly only if it was completely correct. The control group had a mean score of 23.5% (SD, 21.1). H.M., over two sessions, had a mean score of 33.3%. Because different figures were used in Session 4 than in Sessions 2 and 3, caution must be taken in comparing the cued recall scores to the priming scores. With this caveat in mind, the cued recall scores of the control subjects were surprisingly low. Their scores were not significantly higher than their priming scores (20.8%) and were lower than H.M.'s cued recall score. A month or more later, we retested five of the control subjects with the same materials used in Session 4 but with different instructions. The subjects were told, as they copied the figures, that their memory for the figures would be tested; otherwise the procedure was the same. These five subjects, who originally recalled a mean of 15.6% of the figures following incidental encoding, recalled 47.0% following intentional encoding.

The recognition score was the percentage of correct answers; chance performance was 25%. The control group had a mean recognition score of 78% (range 67–100). H.M.'s score was 33% (Fig. 2). It remained possible, however, that H.M. could have recognized those particular figures that had evoked priming in him. Therefore, his recognition score was recalculated separately for the seven figures that he had primed on. H.M. recognized only one of those figures corrrectly, a 14% recognition performance that was below chance (25%) and below his overall recognition performance of 33%.

DISCUSSION

Copying the target figures influenced H.M.'s subsequent free drawing onto the dot patterns as much as copying influenced the free drawing of the NCS. H.M. had a normal magnitude of priming on three different test forms, on three separate testing occasions, and with two different protocols. The intact priming of patterns occurred despite H.M.'s impaired recognition of the target figures. The results demonstrate a novel form of pattern priming in normal subjects, provide the first report of intact nonverbal priming in H.M., and show that intact priming of nonverbal material can be dissociated from impaired recognition memory of the same material. Because the patterns did not relate directly to premorbidly formed verbal representations, their similar influence on normal and amnesic subjects extends the boundary conditions for the occurrence of intact priming in global amnesia.

The cued recall results are less straightforward. In accordance with prior analyses of verbal cued performance by amnesic patients [20, 41], we interpret H.M.'s apparently good cued recall performance as another example of his intact priming capacity. That is, when asked to recall and draw the previously copied target figures, H.M. simply drew the first figures that came to mind as he had in the priming sessions. Thus, his cued recall score of 33% was similar to his priming score of 28%. The same reliance on the first figure that came to mind, however, did not help on the multiple-choice recognition test. H.M. scored identically (33%) on the cued recall and multiple-choice recognition tests, whereas control subjects did far better on the recognition test (78%) than on the cued recall test (23.5%). H.M.'s pattern of performance on the two tests of explicit memory for the target figures is unlike that of the normal control subjects, and supports the idea that his cued recall responses were based upon priming mechanisms and not upon the mechanisms that support recall and recognition memory.

Unlike the results reported by GRAF *et al.* [20] and by SHIMAMURA and SQUIRE [41], the NCS in the present experiment did not have a better cued recall than priming score, suggesting that the control subjects did not have strong voluntary or explicit access to their memory of the copied target figures. Like H.M., the control subjects may have drawn the first figures that they could imagine. It may be that H.M. achieved normal levels of nonverbal priming in this study because normal subjects could not consult explicit memory of any target figures without the entire target figure being shown, as in the multiple-choice recognition test. Additional support for the notion that pattern priming may depend upon perceptual learning mechanisms that are dissociable from recognition memory comes from two recent studies with normal subjects [29, 35]. One study [29] involved stimuli that were quite similar to the ones used in the present study, and also used a four-alternative, forced-choice measure of recognition memory. Unlike the present study, the measure of priming was performance on a speeded perceptual task. Recognition memory and priming in normal subjects were dissociable by the criteria of differential declines in memory performance over time and of stochastic independence between recognition and priming performance. The other study with normal subjects found a dissociation between priming and recognition memory for line drawings of three-dimensional objects [35]. The results of those two studies and the failure of the control subjects in the present experiment to score higher on cued recall than on priming measures (unless subjects were forewarned of a memory test) indicate that the preservation of pattern priming in H.M. reflects the separability of perceptual learning and recognition processes in the course of normal cognition.

The preservation of pattern priming in H.M. is consistent with dichotomies that have been drawn between the different kinds of learning that are impaired and intact in amnesia. COHEN and SQUIRE [6] proposed a distinction between declarative and procedural kinds of memory and GRAF and SCHACTER [18] a distinction between explicit and implicit memory. In accordance with many prior studies with H.M., he was impaired by the multiple-choice recognition measure of declarative, explicit memory. He was unimpaired, however, by the procedural, implicit priming measure. It should also be pointed out that the prior dichotomies did not focus exclusively upon measures of verbal memory. There are examples of skill learning that are preserved in amnesia, that are consistent with the declarative/procedural and explicit/implicit distinctions, and that do not have an apparent verbal basis. Mirror tracing [25], rotary pursuit [7], and serial reaction time [30] are three such skills. It is difficult, however, to compare skill learning and priming studies directly, because the former have always been given in protocols that involve many trials of the same kind in a single session, while priming studies have tended to use a single presentation of a stimulus and a single subsequent response.

The pattern priming results address theories about the basis of intact priming in amnesia. SCHACTER and GRAF [36] proposed that there are two kinds of priming, activational and episodic. Activational priming is viewed as being dependent upon the activation of long-term memory representations and being intact in severely amnesic patients who established premorbidly the required memory representations. Episodic priming is seen as being dependent upon the formation of new memory representations, or new associations, that are mediated, at least in part, by the same episodic memory processes that support recall and recognition. Therefore, episodic priming may be preserved only in patients with relatively mild memory impairments (e.g. [36, 42, but see 2]). It is difficult to describe pattern priming as only the activation of long-term memory representations, because it seems unlikely that either normal subjects or H.M. had pre-existing memory representations associating the particular dot patterns with the particular target figures that were employed in the present experiment. Pattern priming required the formation of new associations between dot patterns and target figures that were established in the course of copying target figures onto dot patterns. The new associations influenced subsequent processes of perceptual parsing and organization that assigned a structure to the dot pattern and guided the NCS' and H.M.'s primed drawings. The measure of new associations was the presence and degree of priming. H.M.'s severe amnesia, however, precludes the possibility that his intact priming pattern reflects the operation of recall and recognition mechanisms [8, 38]. Thus, H.M.'s intact pattern priming cannot be classified satisfactorily as an example of either activational or episodic priming.

We propose a different interpretation of the basis of pattern priming, one that emphasizes the visual information-processing demands of perceiving a figural organization emerging from a pattern of unconnected dots. Through vision, we may see an infinite variety of stimuli as we encounter novel stimuli or all the changes in appearance of known stimuli due to variation in size, position, shading, and occlusion. Because there is an infinite number of potential visual stimuli, a perceptual learning system cannot establish a limited vocabulary of potential stimuli, as a system designed for lexical access may do. An adaptive perceptual system must have the capacity to establish a new association between any visual stimulus perceived in a specific instance (e.g. a dot pattern) and the outcome of visual processes that represent the stimulus in meaningful visual codes, such as letters, words, objects, or, in the present case, target figures. Such an adaptive perceptual system may also be activational in part in if it uses a limited long-term vocabulary of shapes (e.g. 1) or parsing routines (e.g. 21) to analyze the variety of percepts that may be encountered in vision. Thus, it seems that perceptually based priming must involve the formation of novel associations between percepts and structural descriptions of percepts. As shown by H.M.'s intact pattern priming and by prior reports of robust perceptual priming in amnesic patients [9, 26, 44, 46], new associations supporting perceptual priming may be established despite severe deficits in episodic memory.

The idea that novel perceptual associations may be established without declarative or explicit memory is consistent with a number of studies that found intact perceptual priming of nonwords in severely amnesic patients. Activational theories of priming predict that amnesic patients cannot show priming for nonwords because amnesic patients do not have pre-existing memory representations of nonwords and, unlike normal subjects, cannot acquire such novel representations postmorbidly. Yet H.M. has shown intact priming for nonwords on two perceptual tasks (11, 13; and see 2 for a similar finding with S.S.). Moreover, the activational account predicts that severely amnesic patients will not prime for new associations between premorbidly known, but premorbidly unassociated, words. MOSCOVITCH [28], however, found priming of new associations in amnesic patients on two tasks involving perceptually degraded verbal stimuli (the patients in that study were a mixed group in terms of etiology and severity). In addition, COHEN et al. [5] reported that amnesic patients showed substantial priming on a symmetry-detection task involving novel checkerboard patterns. The activational/episodic distinction, therefore, may not apply to perceptual tasks in which even severely amnesic patients seem to learn new associations. That distinction, however, may still apply to priming tasks that do not seem to be primarily perceptual in nature, such as word completion, fragment completion, category exemplar production, lexical decision, and sentence puzzles. Performance on these tasks is well documented to be intact in severly amnesic patients when activation of pre-existing memory representations can account for priming (e.g. 2, 3, 10, 11, 12, 13–15, 17–20, 23, 39–41) but impaired when new associations are needed to support priming (e.g. 3, 10, 13, 22, 24, 36, 42, but see 2). SCHACTER [34], in a review of priming studies with normal subjects and of neuropsychological studies with agnosic and dyslexic patients, has come to a similar conclusion about "perceptual representations systems" that may mediate priming on tasks in which word form or structural descriptions of objects play an important role.

We have suggested [13, 14, 23] that extrastriate visual areas may be especially important in perceptual priming. Extrastriate areas show selective activation in positron emission tomography (PET) studies when normal subjects perform a task thought to be sensitive to word form [32]. Furthermore, single-cell recordings in monkeys have shown that neurons in extrastriate visual cortex (V2), but not primary striate visual cortex (V1), respond selectively to stimuli that humans perceive as contours, or subjective continuities, despite no actual line being present [31, 43]. In the pattern priming task, one could think of the lines that subjects draw connecting the dot patterns as expressions of subjective continuities, and the priming of normal subjects and of H.M. as changes in the probability of perceiving one subjective pattern of line continuities over other plausible subjective patterns. In that case, pattern priming could reflect, at least in part, changes in extrastriate perceptual mechanisms that occured when subjects copied target figures onto dot patterns.

Pattern priming and the other sorts of perceptually based priming discussed above demonstrate the influence of prior perceptual analysis upon later perceptual organization of flexibly interpretable stimuli. H.M.'s intact pattern priming indicates that such perceptual learning does not depend upon recall and recognition memory capacities. The poor cued recall performance of the NCS demonstrated that even people with unimpaired recall and recognition have poor conscious access to the newly acquired perceptual information. Perhaps the perceptual associations that mediate pattern priming are established in early stages of visual processing that are relatively inaccessible to awareness. It may be that visual areas of the brain learn from perceptual experience by changing or redistributing the probability of deriving a particular perceptual analysis of a given stimulus. In the case of stimuli that have a correct interpretation, such as incomplete or hidden figures, experience-based change results in enhanced visual skill in item-specific object recognition. In the present experiment, there were no objectively correct figural organizations of the dot patterns, but the priming may be interpreted as an experience-based change in deriving one particular figural analysis of a dot pattern. The vocabulary of figure knowledge is less well specified than that of verbal or object knowledge, but some proposals have been made (e.g. 1, 21). Pattern priming, then, may reflect important adaptive properties of early perceptual processes that play a useful and ubiquitous role in vision.

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REFERENCES

1. BEIDERMAN, I. Human image understanding: recent research and a theory. *Computer Vision, Graphics, and Image Processing* **32,** 29 73, 1985.

2. CERMAK, L. S., BLACKFORD, S. P., O'CONNOR, M. and BLEICH, R. B. The implicit memory ability of a patient with amnesia due to encephalitis. *Brain Cognit.* **7,** 145–156, 1988.
3. CERMAK, L. S., BLEICH, R. P. and BLACKFORD, S. P. Deficits in the implicit retention of new associations by alcoholic Korsakoff patients. *Brain Cognit.* **7,** 312–323, 1988.
4. CERMAK, L. S., TALBOT, N., CHANDLER, K. and WOLBARST, L. R. The perceptual priming phenomenon in amnesia. *Neuropsychologia* **23,** 615–622, 1985.
5. COHEN, N. J., ABRAMS, I., HARLEY, W. S., TABOR, L., GORDON, B. and SEJNOWSKI, T. J. Perceptual skill learning and repetition priming for novel materials in amnesic patients, normal subjects, and neuron-like network models. *Soc. Neurosci. Abstr.* **12,** 1162, 1986.
6. COHEN, N. J. and SQUIRE, L. R. Preserved learning and retention of pattern-analyzing skill in amnesia: dissociation of knowing how and knowing that. *Science* **210,** 207–210, 1980.
7. CORKIN, S. Acquistion of motor skill after bilateral medical temporal-lobe excision. *Neuropsychologia* **6,** 255–265, 1968.
8. CORKIN, S. Lasting consequences of bilateral medial temporal lobectomy: clinical course and experimental findings in H.M. *Seminars in Neurology* **4,** 252–262, 1984.
9. CROVITZ, H. F., HARVEY, M. T. and MCCLANAHAN, S. Hidden memory: a rapid method for the study of amnesia using perceptual learning. *Cortex* **17,** 273–278, 1981.
10. DIAMOND, R. and ROZIN, P. Activation of existing memories in the amnesic syndromes. *J. Abnorm. Psychol.* **93,** 98–105, 1984.
11. GABRIELI, J. D. E. Memory systems of the human brain: dissociations among learning capacities in amnesia. Unpublished doctoral dissertation, Massachusetts Institute of Technology, Cambridge, MA, 1986.
12. GABRIELI, J. D. E., COHEN, N. J. and CORKIN, S. The impaired learning of semantic knowledge following bilateral medial temporal-lobe resection. *Brain Cognit.* **7,** 157–177, 1988.
13. GABRIELI, J. D. E. and KEANE, M. M. Priming in the amnesic patient H.M.: new findings and a theory of intact and impaired priming in patients with memory disorders. *Soc. Neurosci. Abstr.* **14,** 1290, 1988.
14. GABRIELI, J. D. E., KEANE, M. M., STANGER, B. Z., KJELGAARD, M. M., BANKS, K. S., CORKIN, S. and GROWDON, J. H. Dissociations among perceptual-structural, lexical-semantic and event-fact memory subsystems in global amnesia, Alzheimer's disease, and normal memory (submitted).
15. GARDNER, H., BOLLER, F., MOREINES, J. and BUTTERS, N. Retrieving information from Korsakoff patients: effects of categorical cues and reference to the task. *Cortex* **9,** 165–175, 1973.
16. GARNER, W. R. and CLEMENT, D. E. Goodness of pattern and pattern uncertainty. *J. Verb. Learn. Verb. Behav.* **2,** 446–452, 1963.
17. GORDON, B. Preserved learning of novel information in amnesia: evidence for multiple memory systems. *Brain Cognit.* **7,** 257–282, 1988.
18. GRAF, P. and Schacter, D. L. Implicit and explicit memory for new associations in normal and amnesic subjects. *J. Exp. Psychol.: Learn. Mem. Cognit.* **11,** 501–518, 1985.
19. GRAF, P., SHIMAMURA, A. P. and SQUIRE, L. R. Priming across modalities and priming across category levels: Extending the domain of preserved function in amnesia. *J. Exp. Psychol.: Learn. Mem. Cognit.* **11,** 386–396, 1985.
20. GRAF, P., SQUIRE, L. R. and MANDLER, G. The information that amnesic patients do not forget. *J. Exp. Psychol.: Learn. Mem. Cognit.* **10,** 164–178, 1984.
21. HOFFMAN, D. D. and RICHARDS, W. A. Parts of recognition. *Cognit.* **18,** 65–96, 1984.
22. KEANE, M. M., GABRIELI, J. D. E. and CORKIN, S. Multiple relations between fact-learning and priming in global amnesia. *Soc. Neurosci. Abstr.* **13,** 1454, 1987.
23. KEANE, M. M., GABRIELI, J. D. E., KJELGAARD, M. M., GROWDON, J. H. and CORKIN, S. Dissociation between two kinds of priming in global amnesia and Alzheimer's disease. *Soc. Neurosci. Abstr.* **14,** 1290, 1988.
24. MCANDREWS, M. P., GLISKY, E. L. and SCHACTER, D. L. When priming persists: long-lasting implicit memory for a single study episode in amnesic patients. *Neuropsychologia* **25,** 487–506, 1987.
25. MILNER, B. Les troubles de la mémoire accompagnant des lésions hippocampiques bilaterales. In *Physiologie de l'hippocampe.* Paris: Centre National de la Récherche Scientifique, 1962.
26. MILNER, B., CORKIN, S. and TEUBER, H.-L. Further analysis of the hippocampal amnesia syndrome: 14-year follow-up study of H.M. *Neuropsychologia* **6,** 215–234, 1968.
27. MORTON, J. The interaction of information in word recognition. *Psychol. Rev.* **76,** 165–178, 1969.
28. MOSCOVITCH, M., WINOCUR, G. and MCLACHLAN, D. Memory as assessed by recognition and reading time in normal and memory-impaired people with Alzheimer's disease and other neurological disorders. *J. Exp. Psychol.: Gen.* **115,** 331–347, 1986.
29. MUSEN, G. and TREISMAN, A. Implicit and explicit memory for visual patterns. *J. Exp. Psychol.: Learn. Mem. Cognit.* **16,** 127–137, 1990.
30. NISSEN, M. J. and BULLEMER, P. Attentional requirements of learning: evidence from performance measures. *Cognit. Psychol.* **19,** 1–32, 1987.
31. PETERHANS, E. and VON DER HEYDT, R. Mechanisms of contour perception in monkey visual cortex. II. Contours bridging gaps. *J. Neurosci.* **9,** 1749–1763, 1989.

32. POSNER, M. I., PETERSEN, S. E., FOX, P. T. and RAICHLE, M. E. Localization of cognitive operations in the human brain. *Science* **240,** 1627–1631, 1988.
33. SCHACTER, D. L. Implicit memory: history and current status. *J. Exp. Psychol.: Learn. Mem. Cognit.* **13,** 501–518, 1987.
34. SCHACTER, D. L. Perceptual representation systems and implict memory: toward a resolution of the multiple memory systems debate. In *Development and Neural Bases of Higher Cognitive Function, Annals of the New York Academy of Sciences*, A. DIAMOND (Editor), in press.
35. SCHACTER, D. L., COOPER, L. A. and DELANEY, S. M. Implicit memory for unfamiliar objects depends on access to structural descriptions. *J. exp. Psychol.: Gen.*, in press.
36. SCHACTER, D. L. and GRAF, P. Preserved learning in amnesic patients: perspectives from research on direct priming. *J. clin. exp. Neuropsychol.* **8,** 727–743, 1986.
37. SCOVILLE, W. B., DUNSMORE, R. H., LIBERSON, W. T., HENRY, C. E. and PEPE, A. Observations on medial temporal lobotomy and uncotomy in the treatment of psychotic states. In *Proceedings of the Association for Research in Nervous and Mental Disease*, pp. 347–369. Williams and Wilkins: Baltimore, Maryland, 1953.
38. SCOVILLE, W. B. and MILNER, B. Loss of recent memory after bilateral hippocampal lesions. *J. Neurol. Neurosurg. Psychiat.* **20,** 11–21, 1957.
39. SHIMAMURA, A. P. Priming effects in amnesia: evidence for a dissociable memory function. *Q. J. exp. Psychol.* **38A,** 619–644, 1986.
40. SHIMAMURA, A. P., SALMON, D. P., SQUIRE, L. R. and BUTTERS, N. Memory dysfunction and word priming in dementia and amnesia. *Behav. Neurosci.* **101,** 347–351, 1987.
41. SHIMAMURA, A. P. and SQUIRE, L. R. Paired-associate learning and priming effects in amnesia: a neuropsychological study. *J. exp. Psychol.: Gen.* **113,** 556–570, 1984.
42. SHIMAMURA, A. P. and SQUIRE, L. R. Impaired priming of new associations in amnesia. *J. exp. Psychol.: Learn. Mem. Cognit.* **15,** 721–728, 1989.
43. VON DER HEYDT, R. and PETERHANS, E. Mechanism of contour perception in monkey visual cortex. I. Lines of pattern discontinuity. *J. Neurosci.* **9,** 1731–1748, 1989.
44. WARRINGTON, E. K. and WEISKRANTZ, L. A new method of testing long-term retention with special reference to amnesic patients. *Nature, Lond.* **217,** 972–974, 1968.
45. WARRINGTON, E. K. and WEISKRANTZ, L. The amnesic syndrome: consolidation or retrieval? *Nature, Lond.* **228,** 628–663, 1970.
46. WARRINGTON, E. and WEISKRANTZ, L. An analysis of short-term and long-term memory defects in man. In *The Physiological Basis of Memory*, J. A. DEUTSCH, (Editor), pp. 365–396. Academic Press, New York, 1973.