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Perceptual Thresholds and Priming in Amnesia

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The widely accepted idea that perceptual priming is intact in amnesia was challenged recently by the suggestion that perceptual identification (PID) thresholds are elevated in amnesia and that this impairment could mask a priming deficit by artificially inflating priming scores. The authors examined the PID thresholds of amnesic patients across a wide range of stimulus conditions and accuracy levels. Baseline thresholds and priming effects were fully intact for all amnesic patients except in a condition using small stimuli (1.1° × 0.25° of visual angle). In that condition, only the patients with Korsakoff's syndrome were impaired. Accordingly, elevated perceptual thresholds are not a necessary consequence of amnesia, and normal priming in amnesia is not an artifact of threshold differences. The results support the conclusion that priming is independent of the brain structures important for declarative memory that are damaged in amnesia.

Considerable evidence has accumulated in recent years for distinguishing between different kinds of memory that depend on multiple separate brain systems (Richardson-Klavehn & Bjork, 1988; Schacter, 1987; Squire, 1982; Tulving, 1985; Weiskrantz, 1990). The major distinction is between declarative (explicit) memory, which affords conscious recollection of past facts or episodes, and nondeclarative (implicit) memory, which is assessed indirectly through performance (cf. Schacter, 1994; Squire, Knowlton, & Musen, 1993). Although dissociations can be demonstrated in normal subjects between these two kinds of memory, amnesic patients provide a particularly compelling source of evidence for distinguishing between kinds of memory. Amnesic patients are severely impaired on tests of declarative memory, but they perform normally on a variety of nondeclarative memory tasks including skill learning (Brooks & Baddeley, 1976; Cohen & Squire, 1980; Musen, Shimamura, & Squire, 1990; Nissen & Bullemer, 1987; Squire & Frambach, 1990), adaptation level effects (Benzing & Squire, 1989), artificial grammar learning and prototype learning (Knowlton & Squire, 1993; Knowlton, Ramus, & Squire, 1992), and priming (Shimamura, 1986; Tulving & Schacter, 1990). Declarative memory depends on the integrity of the medial temporal lobe and diencephalic structures that are damaged in amnesia (Squire & Zola-Morgan, 1991; Zola-Morgan & Squire, 1993). Nondeclarative memory is independent of these brain structures.

The conclusion that nondeclarative memory is spared in amnesia is based on a considerable body of evidence comparing normal and memory-impaired subjects. Perhaps the most extensively studied form of nondeclarative memory is priming, the improvement in detecting or identifying a stimulus based on its recent exposure (Schacter, Chiu, & Ochsner, 1993; Tulving & Schacter, 1990). In both normal subjects and amnesic patients, priming has been demonstrated for familiar and novel stimuli, including words, nonwords, simple line patterns, and figures. These results can be summarized by the statement that amnesic patients and control subjects exhibit equivalent priming across many different tasks and types of materials.

The notion that priming is intact in amnesic patients was challenged recently, based on a study of correlations among measures of priming, measures of recognition memory, and quantitative magnetic resonance imaging (MRI) measures of brain damage in 3 groups of neurological patients (Jernigan & Ostergaard, 1993). The subject groups were patients with Alzheimer's disease, patients with Huntington's disease, amnesic patients, and control subjects. Priming was assessed with a perceptual identification task (Cermak, Talbot, Chandler, & Wolbarst, 1985) in which previously studied words and new words were initially presented briefly and then for increasing durations until subjects could identify each word.

Contrary to what might have been expected from previous work, a multiple regression analysis found an association between priming and damage to "temporolimbic" structures (this region was reported to include the hippocampus, amygdala, and adjacent gray matter in the medial temporal lobe). The association between priming and temporolimbic damage was not apparent in a simple correlational measure but

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became significant in the multiple regression analysis, when volume loss in the caudate nucleus was also taken into account. The finding across subject groups was that loss of caudate nucleus volume was associated with improved priming scores, and temporolimbic damage was associated with poorer priming scores. These two influences (caudate nucleus damage and temporolimbic damage) exerted opposing effects of similar magnitude on priming. Accordingly, the two effects canceled each other and would have gone undetected unless they had been considered together in a multiple regression analysis.

On the basis of these results, Jernigan and Ostergaard (1993) proposed that previous findings of intact priming in amnesic patients may have been complicated by the presence of undetected striatal damage. Specifically, it was proposed that striatal damage should impair baseline performance scores in priming studies. As a result, priming scores would be spuriously inflated, and any deficit in priming would thereby be masked.

It is well known that baseline performance differences can pose problems when comparing subject groups (Cermak et al., 1985; Cermak, Verfaellie, Milberg, & Letourneau, 1991; Chapman, Chapman, Curran, & Miller, 1994; Ellis & Young, 1988, p. 284; Loftus, 1985; Snodgrass, 1989). If one group initially performs more poorly than another, it may be easier for the poorer group to improve than the group that initially performs well. For example, in one report (Heindel, Salmon, & Butters, 1990), patients with Huntington's disease (HD) were impaired relative to control subjects on a task of identifying fragmented pictures. Following a study phase, the HD patients exhibited greater priming than control subjects. This result was explained by noting that the HD patients were initially slower and less efficient than control subjects at the baseline task of identifying the fragmented pictures. Accordingly, it was easier for their performance to improve. Although this example and the study by Jernigan and Ostergaard (1993) focus on the importance of striatal pathology in causing baseline differences, it should be noted that any baseline difference in stimulus detection or identification, regardless of its origin, could potentially influence priming and complicate the interpretation of the results.

Jernigan and Ostergaard (1993) concluded that it is questionable whether evidence from amnesic patients can provide support for the existence of separate memory systems, at least where such evidence is based on studies of priming. Such evidence, they argued, may be complicated by the fact that amnesic patients process stimuli more slowly and less efficiently than control subjects, that is, amnesic patients perform more poorly than control subjects at baseline word identification. This is an important claim, because if it were true, an important source of support for the existence of multiple separate memory systems would be invalidated.

The proposal developed by Jernigan and Ostergaard (1993) was based on the combined findings from 3 different patient groups and was not demonstrated within the amnesic patient group itself. With respect to amnesic patients, their proposal depends on the crucial assumption that amnesic patients and control subjects typically exhibit systematic differences in baseline performance levels. Although there is reason to doubt the correctness of this assumption (see General Discussion), comprehensive studies have not been conducted to investigate baseline performance levels of amnesic patients in priming tasks.

The finding of intact baseline performance scores in amnesic patients in earlier studies, which were not designed specifically to assess baseline performance, is not sufficient to decide the issue. These studies commonly assessed baseline performance under only one experimental condition, not across a wide range of performance. For example, in a recent study of perceptual priming for words and nonwords (Haist, Musen, & Squire, 1991), amnesic patients exhibited fully intact baseline word identification performance in a condition that yielded approximately 50% correct identification accuracy. Yet, it is possible that at some other difficulty level a deficit in baseline performance would have been detected in the amnesic patients.

In four experiments, we examined the possibility that amnesic patients have a deficit in baseline performance that could affect their scores on tests of priming. We assessed baseline and primed performance in amnesic patients and control subjects, using a perceptual identification task that sampled a wide range of difficulty levels from near 0% to near 100% performance accuracy. The logic behind this approach is simple. If normal baseline performance and normal priming can be demonstrated for amnesic patients across a wide range of difficulty levels, then preserved priming in amnesic patients cannot be attributed to a deficit in baseline performance.

In Experiment 1, we compared baseline perceptual identification ability in amnesic patients and control subjects, testing across a wide range of performance levels. We used an unusually small stimulus size to ensure that performance would reach very low levels with brief exposure durations. In Experiment 2, the procedure for assessing baseline performance was modified to conform to the procedure used in a previous study that had found normal perceptual identification ability in amnesic patients (Haist et al., 1991). Again, testing was carried out across a wide range of performance levels. Experiment 3 assessed baseline perceptual identification ability in amnesic patients and control subjects, while systematically varying the size of the stimuli from small (as in Experiment 1) to large (as in Experiment 2). Experiment 4 investigated priming. The procedure was the same as in Experiment 2, except that half of the stimuli were first presented to subjects in a study phase.

Experiment 1

The purpose of Experiment 1 was to compare the baseline perceptual identification performance of amnesic patients and control subjects. Based on the results of pilot studies, six exposure durations were selected for word presentation such that performance could be assessed across a wide range of accuracy levels.

Method

Amnesic Patients

Patients with Korsakoff's syndrome. Six patients with alcoholic Korsakoff's syndrome (4 men and 2 women) were tested (see Tables 1

Table 1
Characteristics of Amnesic Patients

Patient	Lesion	Age (in years)	WAIS–R IQ	WMS–R Attention	WMS–R Verbal	WMS–R Visual	WMS–R General	WMS–R Delay
			Korsakoff's syndrome					
NC	Dien (K)	50	90	62	80	60	69	<50
RC	Dien (K)	77	106	115	76	97	80	72
NF	Dien (K)	58	94	91	62	73	53	<50
VF	Dien (K)	74	103	93	77	65	67	64
PN	Dien (K)	66	99	81	77	73	67	53
JW	Dien (K)	57	98	104	65	70	57	57
M		64	98.3	91.0	72.8	73.0	65.5	57.6
			Other amnesic patients					
AB	HF[a]	56	104	87	62	72	54	<50
PH	HF	71	115	117	67	83	70	57
WH	HF	71	113	88	72	82	67	<50
MG	Dien	61	111	113	89	84	86	63
LJ	Unknown	56	98	105	83	60	69	<50
M		63	108.2	102.0	74.6	76.2	69.2	54.0

Note. The Wechsler Adult Intelligence Scale—Revised (WAIS–R) and the Wechsler Memory Scale—Revised (WMS–R) indices yield a mean score of 100 in the normal population with a standard deviation of 15. The WMS–R does not provide scores for subjects who score below 50. Therefore, the five scores below 50 were scored as 50 for calculating a group mean. HF = hippocampal formation; Dien = diencephalon; K = Korsakoff's syndrome.
[a]The lesion site has not been confirmed radiologically but is strongly supported by the etiology of amnesia (see text).

and 2). All had participated in either an MR study (Squire et al., 1990; for NF, unpublished observations) or a quantitative CT study (Shimamura et al., 1989). These studies demonstrated marked reductions in the volume of the mammillary nuclei, reduced thalamic tissue density, and frontal lobe atrophy.

The 6 patients averaged 64 years of age and 11.3 years of education. Individual IQ scores (Wechsler Adult Intelligence Scale—Revised [WAIS–R]; Wechsler, 1981) and scores on the Wechsler Memory Scale—Revised (WMS–R; Wechsler, 1987) appear in Table 1. Immediate and delayed (12 min) recall of a short prose passage averaged 4.5 and 0 segments, respectively (21 total segments; Gilbert, Levee, & Catalano, 1968). Scores for other memory tests appear in Table 2. The mean score on the Dementia Rating Scale (Mattis, 1976) was 129.3 (maximum = 144, range = 125–132), with most of the points

Table 2
Memory Test Performance

Patient	Diagram recall	Paired associates	Word recall (%)	Word recognition (%)	50 words	50 faces
		Korsakoff's syndrome				
NC	3	1–0–1	23	71	31	37
RC	3	0–0–3	19	85	37	30
NF	4	0–0–2	36	76	28	27
VF	8	0–0–0	27	91	27	31
PN	2	1–1–1	29	83	31	31
JW	4	0–0–2	28	96	29	34
M	4.0	0.3–0.2–1.5	27.0	83.7	30.5	31.6
		Other amnesic patients				
AB	4	1–1–2	33	83	32	33
PH	3	0–0–1	27	84	36	34
WH	1	0–0–0	40	84	29	24
MG	0	0–0–2	33	71	30	34
LJ	3	0–0–0	40	93	33	29
M	2.2	0.2–0.2–1.0	34.6	83.0	32.0	30.8
Healthy (n = 8)	20.6	6.0–7.6–8.9	71	98	41.1	38.1
Alcoholics (n = 8)	16.4	5.1–8.0–8.8	62	97	36.2	36.2

Note. The diagram recall score is based on delayed (12 min) reproduction of the Rey–Osterrieth figure (Osterrieth, 1944; maximum score = 36). The average score for copying the figure was 27.9, a normal score (Kritchevsky, Squire, & Zouzounis, 1988). The paired-associates score is the number of word pairs recalled on three successive trials (maximum score = 10/trial). The word recall score is the percentage of words identified correctly across five successive study–test trials (Rey, 1964). The word recognition score is the percentage of words identified correctly by yes/no recognition across five successive study–test trials. The score for words and faces is based on a 24-hr recognition test of 50 words or 50 faces (modified from Warrington, 1984; maximum score = 50, chance = 25). The mean scores for healthy control and alcoholic, nonamnesic subjects shown for these tests are from Squire and Shimamura (1986).

lost on the Memory subportion (mean points lost = 7.0) and the Initiation-Perseveration subportion (mean points lost = 3.3). The mean score on the Boston Naming Test (Kaplan, Goodglass, & Weintraub, 1983) was 54.5 (maximum = 60, range = 48–59). Scores for normal subjects on these tests can be found elsewhere (Janowsky, Shimamura, Kritchevsky, & Squire, 1989; Squire, Amaral, & Press, 1990).

Other amnesic patients. Five other amnesic patients (2 men and 3 women) with etiology other than Korsakoff's syndrome were also tested (see Tables 1 and 2). (Patient WH did not participate in Experiment 1 because he could not comfortably focus his eyes at the viewing distance used.) Patient MG (female) sustained a bilateral thalamic infarction. Of the other patients, 3 (PH, WH, and LJ) have bilateral hippocampal damage identified by MR imaging (for PH, Polich & Squire, 1993; for WH, Squire et al., 1990; for LJ, unpublished observations). Patient AB, who is unable to participate in MR studies, became amnesic in 1976 after an anoxic episode and is presumed to have hippocampal damage based on this etiology.

These 5 patients averaged 63 years of age and 16 years of education. Individual IQ scores (WAIS–R) and scores on the WMS–R appear in Table 1. Immediate and delayed (12 min) recall of a short prose passage averaged 5.0 and 0 segments, respectively. Scores on other memory tests appear in Table 2. The mean score on the Dementia Rating Scale (Mattis, 1976) was 134.6 (range = 130–143). Most of the points were lost on the Memory subportion of the test (mean points lost = 6.6). The mean score on the Boston Naming Test was 56.8 (range = 55–58).

Control Subjects

The control subjects (n = 11, 5 men and 6 women) were either employees or volunteers at the San Diego Veterans Affairs Medical Center or were members of the retirement community of the University of California, San Diego. They were selected to match the amnesic patients with respect to age (63.8 years, range = 51–76), education (13.1 years), and WAIS–R subtest scores for Information (20.4, amnesic patients = 20.5) and Vocabulary (53.3, amnesic patients = 54.2). Immediate recall and delayed recall of the short prose passage were 5.8 and 5.0 segments, respectively.

Visual Acuity Testing

Visual acuity testing was conducted to ensure that amnesic patients and control subjects did not differ in visual acuity. Two tests of visual acuity were used: (a) a standard test of visual acuity (reduced Snellen chart), and (b) a paragraph printed in the same format (e.g., visual angle, typefont, etc.) as target items in Experiment 1. The stimulus parameters from Experiment 1 were chosen for visual acuity testing because they created the most difficult viewing conditions encountered in any of the four experiments. All subjects (with the exception of patient JW and 1 control subject) could detect the orientation of characters that subtended as little as 0.17° of visual angle, and they could read a paragraph that was presented in the same format used for target items in Experiment 1. Patient JW and the 1 control subject could detect the orientation of characters subtending 0.24° of visual angle, and they both could read the paragraph, though less fluently than the other subjects.

Materials

A set of 252 six-letter words (mean frequency = 26 per million; range 1–98; Kucera & Francis, 1967) were used as target items on the perceptual identification test. About half the words used were nouns, and the remainder were adjectives, verbs, or adverbs. An additional set of 10 six-letter words was used as practice items and was administered immediately before the perceptual identification test.

Procedure

Three subject groups (patients with Korsakoff's syndrome, the other amnesic patients, and control subjects) were tested at six different exposure durations: 33, 50, 67, 83, 100, and 116 ms. The experiment consisted of a perceptual identification test preceded by a short practice phase intended to familiarize subjects with the experimental procedure. All stimuli were presented on a Macintosh SE computer (PsychLab Version .85). Words were presented at the center of the screen in 12-point lowercase letters in Times Roman font, subtending approximately 1.1° of horizontal and 0.25° of vertical visual angle. Viewing distance was 60 cm. The examiner sat next to the subject and recorded each response.

The perceptual identification test was administered in 12 blocks of 21 items each. Two blocks were assigned to each of the six exposure durations: 33, 50, 67, 83, 100, and 116 ms, so that a total of 42 items were presented at each duration. Materials were counterbalanced across exposure durations, and the order of presentation of the 12 blocks was counterbalanced across subjects. Subjects were instructed that they would be shown words very briefly and that they should try to read aloud each word as quickly and accurately as possible. Subjects were also informed that none of the words would be proper nouns. Guessing was encouraged. Short breaks were scheduled between blocks. Each trial began with a fixation cross presented at the center of the screen for 500 ms. The fixation cross was then replaced by a word, presented in lowercase letters at the designated duration for that block of words. A random pattern mask immediately replaced the stimulus, remaining on the screen for 250 ms. The next trial began 500 ms after the subject's response.

Results

A strict criterion was used in scoring responses. Responses were scored as correct only if they matched the target word exactly or its plural form. Figure 1 shows the main results.

The major finding was that baseline perceptual identification performance was severely impaired in the patients with Korsakoff's syndrome, but the performance of the other amnesic patients was similar to control subjects. All 6 patients with Korsakoff's syndrome exhibited a severe deficit in perceptual identification. A 2 (subject group: patients with Korsakoff's syndrome vs. control subjects) × 5 (exposure duration: 50, 67, 83, 100, and 116 ms) mixed-factorial analysis of variance (ANOVA) yielded a significant main effect of subject group, $F(1, 15) = 4.43$, $MSE = 0.24$, $p < .001$, exposure duration, $F(4, 60) = 23.90$, $MSE = 0.01$, $p < .001$, and a Group × Exposure Duration interaction, $F(4, 60) = 13.02$, $MSE = 0.01$, $p < .001$. (Because performance was at floor for all groups at the 33-ms exposure duration, data from this condition were excluded from this analysis.) The interaction between subject group and exposure duration was forced by a floor effect in the performance of the patients with Korsakoff's syndrome. Indeed, only 1 Korsakoff's syndrome patient (NF) consistently scored above 0 across exposure durations.

In contrast to the severe impairment exhibited by the patients with Korsakoff's syndrome, all of the other amnesic

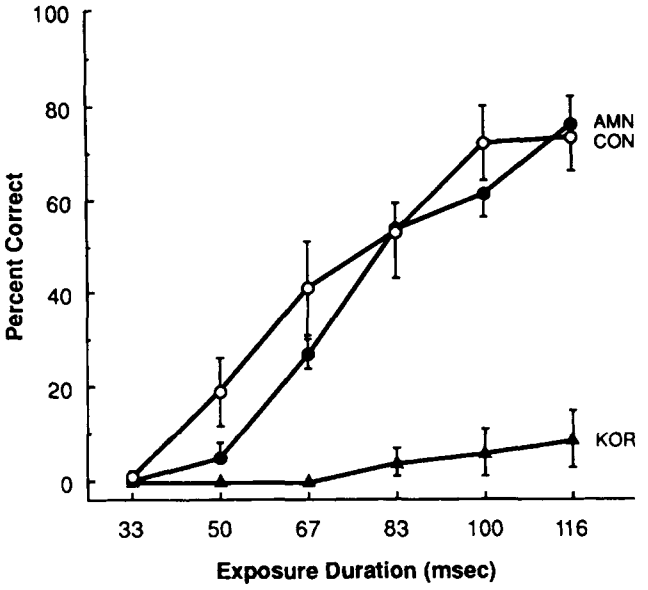

Figure 1. Accuracy of word identification (12-point, lowercase, approximately 1.1° [horizontal] × 0.25° [vertical] of visual angle) as a function of exposure duration for amnesic patients with Korsakoff's syndrome (KOR), other amnesic patients (AMN), and control subjects (CON). Error bars indicate standard error of the mean.

patients exhibited perceptual identification performance similar to that of the control subjects.[1] A 2 (subject group: other amnesic patients vs. control subjects) × 5 (exposure duration: 50, 67, 83, 100, and 116 ms) mixed-factorial ANOVA yielded no effect of subject group, $F(1, 13) = 0.25$, $MSE = 0.28$, $p > .63$, an effect of duration, $F(4, 52) = 64.38$, $MSE = 0.01$, $p < .001$, and no interaction, $F(4, 52) < 1 = 1.72$, $MSE = 0.01$, $p > .16$. Although the patients did perform numerically lower than the control subjects across exposure durations, planned comparisons at each exposure duration yielded no significant differences: for 50 ms, $t(13) = 1.06$; for 67 ms, $t(13) = 0.80$; for 83 ms, $t(13) = -0.02$; for 100 ms, $t(13) = 0.85$; for 116 ms, $t(13) = -0.30$; $p > 0.10$ for each comparison, two-tailed t test.

Discussion

Experiment 1 indicated that the performance of amnesic patients on perceptual identification depended critically on the etiology of amnesia. With the small stimulus (1.1° horizontal) used in Experiment 1, deficits in baseline perceptual identification performance were limited to patients with Korsakoff's syndrome. The other amnesic patients performed similarly to the control subjects. Indeed, at two exposure durations (83 and 116 ms), these amnesic patients performed numerically better than control subjects. This result shows that amnesic patients need not have significant baseline performance deficits. At the same time, it should be noted that, with the small number of available amnesic patients, it would have been difficult to detect small differences between groups.

A previous study suggested that baseline perceptual identification deficits in amnesia can be even more limited than was suggested by Experiment 1. Haist et al. (1991) tested many of the same amnesic patients that were tested in Experiment 1 (all but 4 patients: 2 with Korsakoff's syndrome, NF and JW, and 2 other amnesic patients, PH and WH). In a perceptual identification task, intact baseline performance as well as intact priming were observed, even for the patients with Korsakoff's syndrome. Specifically, the exposure duration needed to achieve 50% baseline identification accuracy for words was the same for control subjects, patients with Korsakoff's syndrome, and non-Korsakoff patients (47.7 ms, 54.0 ms, and 52.5 ms, respectively). There were some methodological differences between the two studies that might account for the difference in results between the earlier study and the current Experiment 1.

To explore this issue, Experiment 2 repeated the procedure of Experiment 1 but with the same stimulus parameters (i.e., the same stimulus format, stimulus size, and type of masking stimulus) that were used in the Haist et al. (1991) study. The question of interest was whether baseline performance was normal with these stimulus parameters for all the amnesic patients, including patients with Korsakoff's syndrome. A second important issue is that the earlier study tested baseline perceptual identification performance at only one accuracy level (i.e., under conditions in which subjects could identify at baseline testing approximately 50% of the stimulus words). Accordingly, we also asked whether baseline performance was normal or impaired when performance was sampled across a wide range of accuracy levels.

Experiment 2

Experiment 2 was identical to Experiment 1 except for three changes in the procedure: (a) the stimuli were approximately 10 times larger than in Experiment 1; (b) the stimuli were presented in uppercase letters; (c) the mask was a string of ampersands.

Method

Amnesic Patients

All 11 amnesic patients participated in Experiment 2.

[1] In Figure 1, Patient AB obtained scores of 0%, 14%, 31%, 62%, 76%, and 93% correct as exposure durations increased from 33 ms to 116 ms. Nineteen months earlier, he had obtained markedly poorer scores with the same material (0% correct through the 83 ms exposure duration, 19% correct at 100 ms, and 43% correct at 116 ms). Subsequent to this earlier testing, we discovered that AB had worn nonprescription reading glasses for testing and that his family was no longer obtaining prescription glasses for him because he regularly lost or broke them. The data in Figure 1 were obtained after we obtained new corrective lenses for him. His visual acuity, as assessed prior to this testing, also improved with the new lenses from below the mean of the other amnesic patients to as high as the level achieved by any participant in Experiments 1–4. In two other sessions that followed the same procedure, AB's performance with his new glasses continued to match that of the control participants shown in Figure 1. One of these sessions used all new 5- and 7-letter words with the same mean frequency as the words in Experiment 1, and the other used the words from Experiment 2. Because AB performed well, even when all new stimuli were used, his good performance cannot depend on the material in Experiment 1 having been presented in a testing session 19 months earlier.

Control Subjects

The control subjects (n = 10, 5 men and 5 women) were either employees or volunteers at the San Diego Veterans Affairs Medical Center or were members of the retirement community of the University of California, San Diego. They were selected to match the amnesic patients with respect to age (62.8 years, range = 49–71), education (14.5 years), and WAIS–R subtest scores for Information (21.7, amnesic patients = 20.5) and Vocabulary (53.5, amnesic patients = 54.2). Immediate recall and delayed recall of the short prose passage were 7.2 and 5.8 segments, respectively.

Materials

A set of 252 six-letter words (mean frequency = 22 per million; range 1–96; Kucera & Francis, 1967) were used as target items on the perceptual identification test. None of these words were used in Experiment 1. An additional set of 10 six-letter words were used as practice items and administered immediately before the perceptual identification test.

Procedure

Three subject groups (patients with Korsakoff's syndrome, the other amnesic patients, and control subjects) were tested at six exposure durations: 33, 50, 67, 83, 100, and 116 ms. The experiment consisted of a perceptual identification test preceded by a short practice phase. Words were presented at the center of the computer screen in 72-point uppercase letters in Helvetica font, subtending approximately 11° of horizontal and 1.7° of vertical visual angle. Viewing distance was 50 cm. The examiner sat next to the subject and recorded each response. The perceptual identification test and practice phase were administered exactly as in Experiment 1 with one exception. To conform to the procedure used by Haist et al. (1991), the masking stimulus in Experiment 2 was a row of six ampersands (&&&&&&) presented for 500 ms, whereas the masking stimulus used in Experiment 1 was a random pattern mask presented for 250 ms.

Results

The same strict criterion used in Experiment 1 was used to score responses. Figure 2 shows the main results.

Using the stimulus parameters of the Haist et al. (1991) study eliminated baseline perceptual identification deficits for all subjects who had exhibited such deficits in Experiment 1. A 3 (subject group: patients with Korsakoff's syndrome, other amnesic patients, and control subjects) × 5 (exposure duration: 50, 67, 83, 100, and 116 ms) mixed-factorial ANOVA yielded no significant main effect of subject group, $F(2, 17) < 1$, a significant effect of duration, $F(4, 68) = 51.10$, $MSE = 0.009$, $p < .001$, and no interaction, $F(8, 68) < 1$. The mean standard error across conditions was 0.07, range = 0.04–0.10. Again, because performance was at floor for all subject groups at the 33-ms exposure duration, the data from that condition were excluded from the analyses.

Discussion

The main finding was that the baseline performance of amnesic patients, including patients with Korsakoff's syndrome, was entirely normal in the perceptual identification task. Accordingly, it appears that the perceptual identification

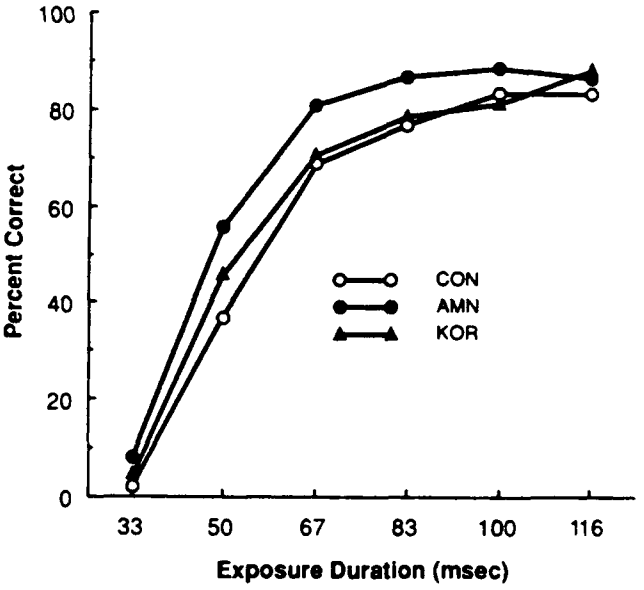

Figure 2. Accuracy of word identification (72-point, uppercase, approximately 11° [horizontal] × 1.75° [vertical] of visual angle) as a function of exposure duration for amnesic patients with Korsakoff's syndrome (KOR), other amnesic patients (AMN), and control subjects (CON).

performance of amnesic patients is not only determined by etiology (Experiment 1; see Figure 1). Baseline perceptual identification is also determined by the particular stimulus conditions that are used. Patients with Korsakoff's syndrome were impaired under the stimulus conditions of Experiment 1. With the stimuli used in Experiment 2, all amnesic patients exhibited intact baseline perceptual identification performance across the full range of the performance scale, from near 0% accuracy to near 100% accuracy. Apparently, one or more of the different stimulus parameters used in Experiment 1 was responsible for the deficit in the amnesic patients with Korsakoff's syndrome that was observed in Experiment 1. The crucial parameter could have been one or a combination of the three parameters that changed between Experiment 1 and Experiment 2: stimulus size, uppercase versus lowercase letters, or the type of masking stimulus used.

Experiment 3

There were three methodological differences between Experiments 1 and 2. First, in Experiment 2 the stimuli were unusually large (about 11° of visual angle), approximately 10 times larger than the stimuli used in Experiment 1. In addition, the stimuli in Experiment 2 were presented in Helvetica font in 72-point uppercase letters, whereas in Experiment 1 stimuli were presented in Times Roman font in 12-point lowercase letters. Finally, the masking stimulus used in Experiment 2 was a string of ampersands, whereas in Experiment 1 the mask was formed using randomly joined letter parts.

Of these differences, it seems likely that the size of the stimuli (visual angle subtended) was the crucial difference between the two studies that resulted in normal performance

by both patient groups in Experiment 2 and impaired performance by the patients with Korsakoff's syndrome in Experiment 1. In Experiment 3, we systematically varied the visual angle subtended by the stimuli to determine how small the stimuli must be to reveal a deficit (in the patients with Korsakoff's syndrome). Stimuli were presented at each of 10 different visual angles (1° to 10° in 1° increments). The stimulus parameters were the same as those used in Experiment 2 (with respect to type font, type case, and masking stimulus), and perceptual thresholds were assessed using the method of ascending limits employed by Jernigan and Ostergaard (1993). We expected that performance would be intact when the stimuli were presented at 10° of visual angle, because this set of stimulus parameters was close to the conditions of Experiment 2. The question of interest was, as the visual angle decreased, at what point would a deficit emerge for the patients with Korsakoff's syndrome.

We also included an additional condition that closely matched the stimulus parameters used in Experiment 1 (i.e., the same type font, type case, masking stimulus, and 1° of visual angle). In this way, it was possible to compare performance directly under the conditions of Experiment 1, under the conditions of Experiment 2, and under conditions that varied the visual angle of the stimuli from 1° to 10° (otherwise using the stimulus parameters of Experiment 2).

Method

Amnesic Patients

The 6 patients with Korsakoff's syndrome participated in Experiment 3.

Control Subjects

The control subjects (n = 6, 4 men and 2 women) had all participated in Experiment 2. They matched the amnesic patients with respect to age (65.6 years, range = 61–71), education (14.5 years), and WAIS–R subtest scores for Information (22.8, amnesic patients = 18.5) and Vocabulary (51.5, amnesic patients = 50.0). Immediate recall and delayed recall of the short prose passage were 6.8 and 5.1 segments, respectively.

Materials

A set of 72 six-letter words (mean frequency = 22 per million, range 1–102; Kucera & Francis, 1967) were taken from the materials used in Experiment 1 and were used as target items for the perceptual identification test. Words were selected that had not been correctly identified by any of the patients with Korsakoff's syndrome in the perceptual identification test of Experiment 1.

The 72 words were randomly divided into 12 lists of six items each, with the constraint that the mean word frequency for each list was between 17 and 31 occurrences per million. Eleven of the lists were used as target lists for the perceptual identification test; the remaining list was used for practice.

Design and Procedure

The experiment consisted of a perceptual identification test preceded by a short practice phase. Eleven six-item lists were presented during the perceptual identification test. Viewing distance was 60 cm. Ten of the lists were presented under the same stimulus conditions used in Experiment 2 (identical type font, type case, and masking stimulus), except that each list was presented at a different visual angle (1, 2, 3, 4, 5, 6, 7, 8, 9, or 10°). The 11th list was presented under the same stimulus conditions used in Experiment 1 (identical type font, type case, and masking stimulus); this list was presented at 1° of visual angle. The practice phase consisted of six items presented under the stimulus conditions of Experiment 2 and at 5° of visual angle. The 11 lists were presented in one of two random orders, counterbalanced across subjects. Within each list, the six items were always presented in the same order. Because the masking stimulus for the 11th list differed markedly from the masking stimulus used for the other 10 lists, a practice trial with the new masking stimulus preceded the first trial of the 11th list.

For each item in the perceptual identification test, a threshold was assessed by the method described by Jernigan and Ostergaard (1993), with the exception that the initial exposure duration used in Experiment 3 was 33 ms instead of 16 ms as in their study. Thus, each word was initially presented for 33 ms and was followed immediately by the masking stimulus. The subject's task was to identify the word presented. If the word was not identified, exposure duration was gradually increased in 17-ms steps, and the procedure was repeated until the subject was able to identify the word. This exposure duration was used as the identification threshold for the item. In a few cases when a word could not be identified even at an exposure duration of 200 ms, the exposure duration was then increased in 100-ms steps for that item until the subject could identify the word.

Results

The same strict criterion used in Experiments 1 and 2 was used to score responses. Figure 3 shows the main results for the patients with Korsakoff's syndrome and control subjects.

A 2 (group: patients with Korsakoff's syndrome vs. control subjects) × 11 (visual angle of presentation: 1° of visual angle with the stimulus parameters from Experiment 1, 1–10° of visual angle with the stimulus parameters from Experiment 2) mixed-factorial ANOVA was performed on the mean threshold scores for each subject. There was an effect of visual angle, $F(1, 10) = 13.57, MSE = 1733.95, p < .0001$, and an interaction between group and visual angle, $F(10, 100) = 5.82, MSE = 1733.95, p < .0001$, but no overall effect of group, $F(1, 10) = 3.03, MSE = 8348.00, p > .10$.

A priori orthogonal tests using a t statistic between groups at each visual angle condition indicated that thresholds were abnormally high for the amnesic patients when stimuli were presented at 1° and 2° of visual angle: for the 2° condition, $t = 2.48$; for the 1° condition, $t = 6.43$; for the 1° condition with Experiment 1 stimulus parameters, $t = 6.38$; critical t value for multiple comparisons, $t(.025, 110) = 1.98$.

Discussion

Patients with Korsakoff's syndrome exhibited impaired perceptual identification performance but only with stimulus displays presented at small visual angles (or, equivalently, at high spatial frequencies). When stimuli were presented at visual angles of 3–10°, all the patients performed as well as control subjects. So long as the visual angle was small, an impairment was observed whether we used the stimulus parameters of Experiment 1 or Experiment 2. Accordingly, the

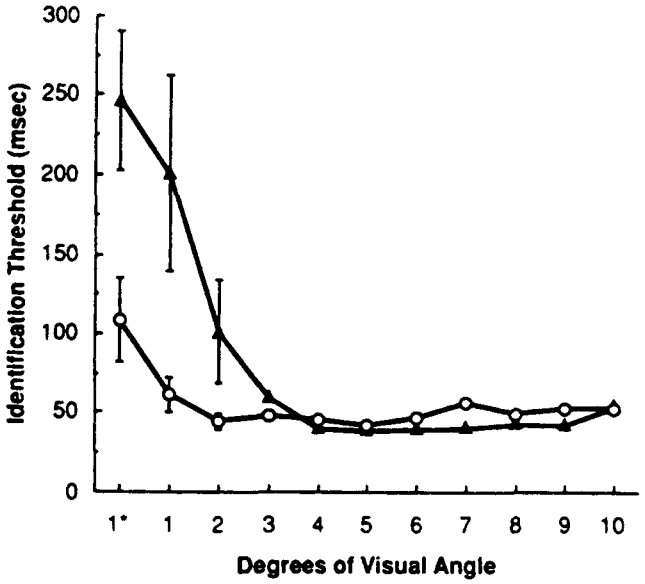

Figure 3. Word identification threshold as a function of the visual angle subtended by the words (horizontal visual angle varied from 1° to 10°). The other stimulus parameters were the same as in Figure 2 (Experiment 2). 1* = word identification threshold at 1° of visual angle, using the stimulus parameters from Figure 1 (Experiment 1). Error bars indicate standard error of the mean. For clarity, error bars are omitted for visual angle conditions greater than 2° (largest value = 62 ms). Triangles indicate patients with Korsakoff's syndrome; circles indicate control subjects.

impaired baseline performance observed in Experiment 1 cannot be attributed entirely to the particular stimulus parameters used in that experiment (e.g., the type case, type font, or masking stimulus). Nevertheless, it is true that when stimuli were presented at 1° of visual angle, the combination of stimulus parameters used in Experiment 1 made perceptual identification nonsignificantly more difficult than the combination used in Experiment 2 (compare condition 1* and 1 in Figure 3; post hoc test, Tukey's LSD = 55.24, difference between the 1° of visual angle condition with Experiment 1's stimulus parameters and the corresponding condition with Experiment 2's stimulus parameters for patients with Korsakoff's syndrome = 45.78, 46.88 for controls).

Experiment 4

Having demonstrated that baseline perceptual identification performance can be intact in amnesic patients across a wide range of accuracy levels (Experiment 2), we next assessed whether priming is also preserved across the same range of conditions. Experiment 4 was identical to Experiment 2, except that half of the target words were first presented to subjects in a study phase. In this way, for both amnesic patients and control subjects, primed perceptual identification performance was compared with baseline perceptual identification performance.

Method

Amnesic Patients

All 11 amnesic patients participated in Experiment 4.

Control Subjects

The control subjects (n = 10, 3 men and 7 women) were either employees or volunteers at the San Diego Veterans Affairs Medical Center or were members of the retirement community of the University of California, San Diego. They were selected to match the amnesic patients with respect to age (62.8 years, range = 52–80), education (14.5 years), and WAIS–R subtest scores for Information (21.0, amnesic patients = 20.5) and Vocabulary (55.1, amnesic patients = 54.2). Immediate recall and delayed recall of the short prose passage were 8 and 6.4 segments, respectively.

Materials

A set of 252 six-letter words (mean frequency = 22 per million; range 1–106; Kucera & Francis, 1967) were used as study items and target items for the perceptual identification test. None of the words had been used in Experiments 1 or 2. An additional set of 10 six-letter words were used as practice items and were administered immediately before the perceptual identification test.

Procedure

Three subject groups (patients with Korsakoff's syndrome, other amnesic patients, and control subjects) were administered a study phase in which they saw half of the words that would later appear in the perceptual identification test. Then, immediately after the study phase, subjects were given a perceptual identification test in which words were presented at four different exposure durations: 33, 50, 67, and 83 ms.

For the study phase, the assignment of words to studied versus nonstudied conditions was counterbalanced across subjects. The 126 words assigned to the study phase were presented one at a time in a random order on a computer screen, and subjects were asked to rate each item on a 1-to-5 scale according to how much they liked the word. Words remained on the screen until the subject responded by typing a number (1–5). A rating of 1 corresponded to *dislike very much,* and a rating of 5 corresponded to *like very much.* An index card with the rating scale printed on it remained in view during the presentation of all items.

The perceptual identification test was administered exactly as in Experiment 2. Half of the items presented in the perceptual identification test had been previously presented to each subject; the other half had not been presented but served as study items for a different subject. For each of the 12 blocks of 21 words, approximately half of the words in each block had been presented earlier in the study phase. Studied and nonstudied words were randomly intermixed within each test block.

Results

The same strict criterion used in Experiments 1 and 2 was used to score responses. Figure 4 shows the main results.

The 3 groups were equivalent in the baseline condition, in the primed condition, and in the magnitude of priming at each exposure duration. First, three separate 3 × 4 mixed-design ANOVAs were carried out: 3 (subject groups: patients with

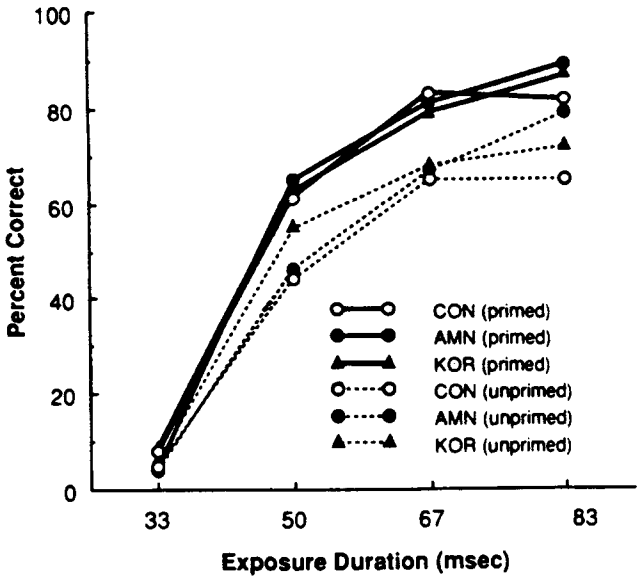

Figure 4. Accuracy of word identification as a function of exposure duration and primed versus unprimed status of the words. KOR = Korsakoff's syndrome patients; AMN = amnesic patients; CON = control subjects.

Korsakoff's syndrome, other amnesic patients, and control subjects) × 4 (exposure durations: 33, 50, 67, and 83 ms). The three analyses used the scores from the baseline condition, the scores from the primed condition, and the difference (priming) scores between the baseline and priming conditions. For baseline performance, there was no effect of subject group, $F(2, 18) < 1$, a significant effect of duration, $F(3, 54) = 117.08$, $MSE = 0.015, p < .001$, and no interaction, $F(6, 54) < 1$.

For performance in the primed condition, there was no effect of subject group, $F(2, 18) < 1$, a significant effect of duration, $F(3, 54) = 233.73$, $MSE = 0.01, p < .001$, and no interaction, $F(6, 54) < 1$.

Significant priming was observed for all subject groups. First, a 2 (priming: studied vs. nonstudied) × 3 (exposure duration: 50, 67, and 83 ms) within-subjects ANOVA on scores from the studied and nonstudied conditions across the subjects in all 3 groups found an effect of priming, $F(1, 20) = 72.65$, $MSE = 0.007, p < .0001$, exposure duration, $F(2, 40) = 44.83$, $MSE = 0.014, p < .0001$, and no interaction, $F(2, 40) < 1$.

Significant priming did not occur in the 33-ms duration condition because of a floor effect, and that condition was therefore excluded from this analysis and all subsequent analyses in this section. Next, to determine whether significant priming occurred within each subject group, a 2 (priming: studied vs. nonstudied) × 3 (exposure duration: 50, 67, and 83 ms) within-subjects ANOVA was performed separately for each subject group. For the patients with Korsakoff's syndrome, there was an effect of priming, $F(1, 5) = 12.03$, $MSE = 0.011, p < .02$, exposure duration, $F(2, 10) = 14.34$, $MSE = 0.010, p < .001$, and no interaction, $F(2, 10) < 1$. The results were the same for each group. For the other amnesic patients, there was an effect of priming, $F(1, 4) = 23.00$, $MSE = 0.007$, $p < .01$, exposure duration, $F(2, 8) = 13.71$, $MSE = 0.016, p < .003$, and no interaction, $F(2, 8) = 1.83$, $MSE = 0.004, p > .10$. Finally, for the control subjects, there was an effect of priming, $F(1, 9) = 38.47$, $MSE = 0.011, p < .0002$, exposure duration, $F(2, 18) = 18.13$, $MSE = 0.016, p < .0001$, and no interaction, $F(2, 18) < 1$.

Table 3 shows the priming results for Experiment 4. To compare the magnitude of priming between groups, we carried out a 3 (subject group) × 3 (exposure duration) mixed-design ANOVA based on the priming scores (difference between primed and unprimed conditions). There was no effect of subject group, $F(2, 18) < 1$, no effect of exposure duration, $F(2, 36) < 1$, and no interaction, $F(4, 36) < 1$. Next, this analysis was repeated, comparing each of the amnesic patient groups separately with the control group. The results of the 3-group comparison also held for the separate comparisons. For the comparison between patients with Korsakoff's syndrome and control subjects, there was no effect of subject group, $F(1, 14) = 1.14$, $MSE = 0.021, p > .10$, no effect of exposure duration, $F(2, 28) < 1$, and no interaction, $F(2, 28) < 1$. For the comparison between other amnesic patients and control subjects, there was also no effect of subject group, $F(1, 13) < 1$, no effect of exposure duration, $F(2, 26) < 1$, and no interaction, $F(2, 26) < 1$.

Discussion

The results from the baseline condition replicated the results of Experiment 2. Individual amnesic patients exhibited highly similar baseline performance in Experiments 2 and 4, indicating that the measure of baseline performance was reliable (correlation between performance averaged across the 50, 67, and 83-ms exposure duration conditions in Experiments 2 and 4 for the 11 amnesic patients was significant, $r = .82$, $p < .01$).

The results from the primed condition indicated in addition that the intact perceptual identification performance observed for amnesic patients in Experiment 2 also extends to priming and that priming is intact across the entire performance scale. Furthermore, the magnitude of priming was largely invariant across three exposure durations as baseline performance spanned 47% to 71% correct (overall priming was 15% for the 50-ms exposure duration, 15% for the 67-ms condition, and 14% for the 83-ms condition). Because preserved priming occurred in the absence of any baseline performance deficit, it cannot be the case that some deficit in priming was present but

Table 3
Experiment 4 Priming Scores

	Exposure duration (ms)							
	33		50		67		83	
Group	*M*	*SEM*	*M*	*SEM*	*M*	*SEM*	*M*	*SEM*
Korsakoff's syndrome patients	0.03	0.03	0.09	0.06	0.12	0.04	0.15	0.08
Other amnesic patients	0.00	0.01	0.20	0.03	0.15	0.03	0.09	0.06
Control subjects	0.04	0.03	0.17	0.04	0.17	0.05	0.16	0.04

Note. In each case, the scores are the difference between the studied and nonstudied conditions.

was masked by a deficit in baseline identification performance. Accordingly, these results support the conclusion that priming on the perceptual identification test is intact in amnesia and independent of the medial temporal lobe and diencephalic structures damaged in amnesia.

General Discussion

In four experiments, we consistently found that baseline thresholds for perceptual identification were normal for amnesic patients across a wide range of accuracy levels. The exception to this pattern was a consistent deficit limited to patients with Korsakoff's syndrome when the stimulus displayed subtended small visual angles. Specifically, deficits were observed only for words that subtended less than 3° of visual angle, with the severity of the deficit sharply increasing when the stimuli subtended only 1° of visual angle. With larger stimuli, both priming and baseline thresholds for perceptual identification were normal for all amnesic patients.

These findings do not support the proposal (Jernigan & Ostergaard, 1993) that baseline performance in perceptual identification is systematically worse in amnesic patients than in control subjects and that this poorer baseline performance masks a deficit in priming. The present findings (i.e., normal baseline performance and normal priming in perceptual identification) support a different conclusion; namely, there are no systematic differences in perceptual identification performance between amnesic patients and control subjects, and priming of perceptual identification is fully intact in amnesic patients.

It is worth noting that in many conditions the amnesic patients actually obtained numerically better baseline thresholds for perceptual identification than control subjects. Thus, in Experiment 1 the amnesic patients without Korsakoff's syndrome performed better than the control subjects at the 83 ms and 116 ms exposure durations (Figure 1). In Experiment 2, the 11 amnesic patients as a group performed better than the control subjects at all exposure durations from 50 ms to 116 ms (Figure 2). In Experiment 3, the patients performed better than the control subjects in the conditions in which stimuli subtended 4° to 9° of visual angle (Figure 3). In Experiment 4, the 11 amnesic patients as a group performed better than the control subjects on unprimed word identification at all exposure durations from 50 ms to 83 ms (Figure 4). Accordingly, it seems unlikely that there were small differences in baseline perceptual identification thresholds between the amnesic patients and the control subjects that were not detected because of insufficient power.

If baseline performance and priming in perceptual identification tasks are indeed intact in amnesic patients, how can the results of the correlational analysis carried out by Jernigan and Ostergaard (1993) be explained? Two findings from their multiple regression analysis formed the basis of the argument: (a) Amnesic patients exhibited a deficit in baseline performance relative to control subjects (see Figure 3F, p. 19, in Jernigan & Ostergaard, 1993), and this deficit appeared to be linked to striatal damage; and (b) damage to temporolimbic areas was associated with impaired priming scores.

The finding of a baseline performance deficit in their amnesic patient group is most likely due to the fact that the amnesic group in their study consisted primarily of patients with Korsakoff's syndrome (9 of the 11 amnesic patients). As demonstrated in our Experiments 1 and 3, this etiological group does have a deficit in the perceptual identification of high spatial frequency stimuli. The stimuli used by Jernigan and Ostergaard (1993) were within the critical range of visual angles in which a deficit in perceptual identification can be observed in patients with Korsakoff's syndrome. Specifically, their word stimuli had a modal length of six letters (range = four to seven letters), and they were presented on an Apple IIe screen in the default uppercase font, yielding a modal stimulus size of 3 cm. Subjects viewed the stimuli at distances ranging from approximately 60 to 90 cm (subjects were allowed to select the viewing distance most comfortable to them; A. L. Ostergaard, personal communication, December 1993). One can calculate that a modal (six letter) stimulus was presented at between 1.8° to 2.8° of visual angle. (It may be worth mentioning that one also cannot rule out a direct contribution of poor visual acuity in their amnesic patient group to perceptual identification performance as no screening was carried out to assess visual acuity.)

These considerations may also help to understand other available evidence concerning perceptual identification performance in amnesic patients. Cermak and colleagues (Cermak et al., 1985, 1991) reported that amnesic patients require longer exposure durations than do control subjects to identify nonstudied words. However, all of the amnesic patients who participated in these experiments were patients with Korsakoff's syndrome. In a more recent study, Schacter, Church, and Treadwell (1994) examined priming effects in patients with Korsakoff's syndrome and other amnesic patients on an auditory perceptual identification test in which previously studied and nonstudied words were masked by white noise. Schacter et al. (1994) reported normal priming effects in both subgroups of amnesic patients. Baseline identification performance of the entire amnesic patient group was nonsignificantly lower than baseline identification performance of the control subjects. More important, analysis of the 2 amnesic patient subgroups revealed that non-Korsakoff amnesic patients exhibited entirely normal baseline identification performance, whereas patients with Korsakoff's syndrome exhibited significantly impaired baseline identification performance.

A study by Musen and Squire (1992) yielded a virtually identical outcome. They reported intact priming in a mixed group of amnesic patients on a task that involved perceptual identification of novel line patterns. In addition, the proportion of nonstudied patterns identified correctly was similar for amnesic patients and control subjects (52.2% vs. 58.2%, $p > .10$). We computed separate baselines for the patients with Korsakoff's syndrome ($n = 4$) and the other amnesic patients ($n = 5$). The baseline identification rate for the other amnesic patients (61.2%) was slightly higher than that of control subjects (58.2%), whereas the identification rate for patients with Korsakoff's syndrome was substantially lower (41.6%), $t(7) = 3.85, p < .01$. Nevertheless, as in the Schacter et al. (1994) study, both patients with Korsakoff's syndrome and other amnesic patients exhibited virtually identical levels

of priming (8.3% and 7.3%) that did not differ significantly from the priming exhibited by control subjects (10.4%).

The foregoing studies, taken together with the present results, demonstrate clearly that (a) visual and auditory baseline perceptual identification performance is intact across a range of conditions in amnesic patients other than those with Korsakoff's syndrome, and (b) such patients exhibit normal priming effects together with intact baseline performance (cf. Haist et al., 1991; Schacter, Cooper, & Treadwell, 1993). These considerations are at odds with Jernigan and Ostergaard's (1993) proposal that normal priming effects in amnesic patients are an artifact of impaired baseline performance.

Based on the results of their multiple regression analysis, Jernigan and Ostergaard (1993) proposed that impaired baseline perceptual identification might result from slowed perceptual and lexical processing caused by striatal damage. However, no relationship between striatal damage and impaired baseline perceptual identification was demonstrated for the amnesic patient group alone. Instead, the multiple regression analysis was based on pooled data from all the subjects in the control group and 3 different patient groups, including the amnesic group. It is therefore possible that the relationship identified in their study between striatal damage and impaired baseline perceptual identification performance came not from the amnesic patients but from 1 or both of the other patient groups. For example, patients with HD have prominent pathology in the striatum, and this group did exhibit impaired baseline scores in their study (see Figure 3F, p. 19, in Jernigan & Ostergaard, 1993; Bruyn, Bots, & Dom, 1979; Vonsattel et al., 1985).

Striatal damage is neither typical nor necessary in the neuropathology of amnesia. The caudate nucleus was not damaged in the surgical patient HM (Scoville & Milner, 1957), and it is reportedly intact in the postencephalitic patient Boswell (Damasio, Eslinger, Damasio, Van Hoesen, & Cornell, 1985). In Patient RB, detailed histological examination revealed no bilateral damage in the basal ganglia (Zola-Morgan, Squire, & Amaral, 1986). Finally, in the study by Jernigan and Ostergaard (1993) itself, there was no detectable caudate damage in the amnesic group (see the amnesic and control scores in their Figure 3A, p. 19), $t(18)$ estimated at $-1.5, p > .1$.

The second important finding that emerged from the regression analysis of Jernigan and Ostergaard's (1993) study was a significant association between temporolimbic damage and priming. Again, the results of the multiple regression analysis were obtained with pooled data from 3 different patient groups and control subjects, and this relationship was not demonstrated for the amnesic patient group alone. Thus, it is possible that the reported relationship between temporolimbic damage and priming could have been absent in the amnesic patient group, and the association could have been carried by 1 or both of the other patient groups. For example, in patients with Alzheimer's disease, one might expect an association between temporolimbic loss and priming, because (a) temporolimbic pathology is a prominent early sign of the disease (Hyman, Van Hoesen, Damasio, & Barnes, 1984; Van Hoesen, Hyman, & Damasio, 1991); (b) priming deficits and deficits in baseline performance occur in Alzheimer's disease (Gabrieli, 1991; Keane, Gabrieli, Fennema, Growdon, & Corkin, 1991); and (c) the severity of temporolimbic damage might remain a marker for the severity of the disease, even as the disease progresses in neocortex, with the result that the severity of temporolimbic damage could correlate with many of the cognitive impairments.

Taking these arguments together, it is plausible that both the patients with HD and the patients with Alzheimer's disease are sources of the effect of striatal damage on baseline performance in the multiple regression analysis. Both these groups had more caudate damage and poorer baseline performance than amnesic patients (see Figure 3, A and F in Jernigan & Ostergaard, 1993). In addition, it is plausible that the patients with Alzheimer's disease are the source of the effect of temporolimbic damage. This interpretation of the multiple regression analysis is as consistent with the data as one that attributes both of these effects to the amnesic patient group (or all 3 groups), and it is also more consistent with existing knowledge about the primary lesion sites in these 3 patient groups.

As noted earlier, the present study is not the first to identify deficits in perceptual identification in patients with Korsakoff's syndrome (cf. Cermak et al., 1985, 1991; Musen & Squire, 1992; Schacter et al., 1994), but it does appear to be the first to show that such deficits are limited to stimuli in a particular range of spatial frequency. In an earlier study (Oscar-Berman, Goodglass, & Cherlow, 1973), substantial deficits were found in patients with Korsakoff's syndrome in a tachistoscopic perceptual identification task in which a word or figure was shown briefly without a masking stimulus. Patients with Korsakoff's syndrome required more viewing time than control subjects (both normal subjects and alcoholic patients without Korsakoff's syndrome) to identify each stimulus (approximately 85 ms for patients with Korsakoff's syndrome compared to approximately 25 ms for control subjects). In another experiment in the same study, stimuli were presented in a backwards-masking paradigm. Patients with Korsakoff's syndrome required an interstimulus interval nearly twice as long as normal subjects to escape the interfering effect of the mask and to identify correctly the stimulus (approximately 90 ms for patients with Korsakoff's syndrome compared to approximately 50 ms for normal subjects). These deficits were interpreted as indicating that patients with Korsakoff's syndrome do not process incoming visual information as efficiently as normal subjects, a conclusion entirely consistent with the findings of the current study. It is worth noting that, because no alcoholic control group was tested in the current study, the limited deficits in perceptual identification found for the amnesic patients with Korsakoff's syndrome could reflect the consequences of alcoholism rather than Korsakoff's syndrome per se.

Although no evidence of a systematic deficit in baseline perceptual identification ability was found for the amnesic patients in this study, other than those with Korsakoff's syndrome, it is certainly prudent to be concerned about the possible distorting effects of baseline differences on priming scores. In our Experiment 4, there was a strong negative correlation for individual control and amnesic subjects ($R = -.85$, R^2 [adjusted] = .71), $F(1, 19) = 50.08$, $MSE =$

0.002, $p < .001$, between baseline identification thresholds and the magnitude of priming. Individual subjects who had lower baseline thresholds (i.e., those who performed less efficiently) exhibited greater priming than subjects who had higher baseline thresholds and were already performing more efficiently. Note, however, that this relationship held for both control subjects and amnesic patients and that the baseline perceptual identification thresholds of the 2 groups were equivalent. Had the groups differed in baseline threshold performance, priming scores could have been systematically distorted. This empirical evidence supports Jernigan and Ostergaard's (1993) caveat regarding the possible distorting effects of baseline differences on priming (for general discussion of baseline-related issues, see Chapman et al., 1994).

In summary, no evidence was found in the current study to support Jernigan and Ostergaard's (1993) proposal that systematic differences in baseline thresholds between amnesic patients and control subjects may generally mask an actual deficit in priming for the amnesic patients. Both baseline thresholds and priming of perceptual identification were normal in amnesic patients across a wide range of the performance scale. Deficits were restricted to patients with Korsakoff's syndrome in a narrow range of conditions. Because both baseline performance and priming were normal in patients with confirmed damage to the hippocampal formation and related structures, the hypothesis that temporolimbic damage impairs priming was not supported. The results suggest that the priming effects that we have documented are subserved by separate brain systems from those important for declarative memory.

References

Benzing, W., & Squire, L. R. (1989). Preserved learning and memory in amnesia: Intact adaptation-level effects and learning of stereoscopic depth. *Behavioral Neuroscience, 103,* 548–560.

Brooks, D. N., & Baddeley, A. (1976). What can amnesic patients learn? *Neuropsychologia, 14,* 111–122.

Bruyn, G. W., Bots, G. T. A. M., & Dom, R. (1979). Huntington's chorea: Current neuropathological status. *Advanced Neurology, 23,* 83–93.

Cermak, L. S., Talbot, N., Chandler, K., & Wolbarst, L. R. (1985). The perceptual priming phenomenon in amnesia. *Neuropsychologia, 23,* 615–622.

Cermak, L., Verfaellie, M., Milberg, W., & Letourneau, L. (1991). A further analysis of perceptual identification priming in alcoholic Korsakoff patients. *Neuropsychologia, 29,* 725–736.

Chapman, L. J., Chapman, J. P., Curran, T. E., & Miller, M. G. (1994). Do children and the elderly show heightened semantic priming? How to answer the question. *Developmental Review. 14,* 159–185.

Cohen, N. J., & Squire, L. R. (1980). Preserved learning and retention of pattern analyzing skill in amnesia: Dissociation of knowing how and knowing that. *Science, 210,* 207–209.

Damasio, A. R., Eslinger, P. J., Damasio, H., Van Hoesen, G. W., & Cornell, S. (1985). Multiple amnesic syndrome following bilateral temporal and basal forebrain damage. *Archives of Neurology, 42,* 252–259.

Ellis, A. W., & Young, A. W. (1988). *Human cognitive neuropsychology.* Hillsdale, NJ: Erlbaum.

Gabrieli, J. D. E. (1991). Differential effects of aging and age-related neurological diseases on memory subsystems of the brain. In F. Bollar & J. Grafman (Eds.), *Handbook of neuropsychology* (Vol. 5, pp. 149–166). New York: Elsevier.

Gilbert, J., Levee, R., & Catalano, K. (1968). A preliminary report on a new memory scale. *Perceptual and Motor Skills, 27,* 277–278.

Haist, F., Musen, G., & Squire, L. R. (1991). Intact priming of words and nonwords in amnesia. *Psychobiology, 19,* 275–285.

Heindel, W. C., Salmon, D. P., & Butters, N. (1990). Pictorial priming and cued recall in Alzheimer's and Huntington's disease. *Brain and Cognition, 13,* 282–295.

Hyman, B. T., Van Hoesen, G. W., Damasio, A. R., & Barnes, C. L. (1984). Alzheimer's disease: Cell specific pathology isolates the hippocampal formation. *Science, 225,* 1168–1170.

Janowsky, J. S., Shimamura, A. P., Kritchevsky, M., & Squire, L. R. (1989). Cognitive impairment following frontal lobe damage and its relevance to human amnesia. *Behavioral Neuroscience, 103,* 548–560.

Jernigan, T. L., & Ostergaard, A. L. (1993). Word priming and recognition memory are both affected by medial temporal lobe damage. *Neuropsychology, 1,* 14–26.

Kaplan, E., Goodglass, H., & Weintraub, S. (1983). Boston Naming Test [manual]. Philadelphia: Lea & Febiger.

Keane, M. M., Gabrieli, J. D. E., Fennema, A. C., Growdon, J. H., & Corkin, S. (1991). Evidence for a dissociation between perceptual and conceptual priming in Alzheimer's disease. *Behavioral Neuroscience, 105,* 326–342.

Knowlton, B. J., Ramus, S. J., & Squire, L. R. (1992). Intact artificial grammar learning in amnesia: Dissociation of classification learning and explicit memory for specific instances. *Psychological Science, 3,* 172–179.

Knowlton, B., & Squire, L. R. (1993). The learning of categories: Parallel brain systems for item memory and category knowledge. *Science, 262,* 1747–1749.

Kritchevsky, M., Squire, L. R., Zouzounis, J. A. (1988). Transient global amnesia: Characterization of anterograde and retrograde amnesia. *Neurology, 38,* 213–219.

Kucera, H., & Francis, W. N. (1967). *Computational analysis of present-day American English.* Providence, RI: Brown University Press.

Loftus, G. R. (1985). Evaluating forgetting curves. *Journal of Experimental Psychology: Learning, Memory and Cognition, 11,* 397–404.

Mattis, S. (1976). Dementia Rating Scale. In R. Bellack & B. Keraso (Eds.), *Geriatric psychiatry* (pp. 77–121). New York: Grune & Stratton.

Musen, G., Shimamura, A. P., & Squire, L. R. (1990). Intact text-specific reading skill in amnesia. *Journal of Experimental Psychology: Learning, Memory and Cognition, 6,* 1068–1076.

Musen, G., & Squire, L. R. (1992). Nonverbal priming in amnesia. *Memory and Cognition, 20,* 441–448.

Nissen, M. J., & Bullemer, P. (1987). Attentional requirements of learning: Evidence from performance measures. *Cognitive Psychology, 19,* 1–32.

Oscar-Berman, M., Goodglass, H., & Cherlow, D. G. (1973). Perceptual laterality and iconic recognition of visual materials by Korsakoff patients and normal adults. *Journal of Comparative Physiology and Psychology, 82,* 316–321.

Osterrieth, P. A. (1944). Le test de copie d'une figure complexe [The test of copying a complex figure]. *Archives de Psychologie, 30,* 206–356.

Polich, J., & Squire, L. R. (1993). P300 from amnesic patients with bilateral hippocampal lesions. *EEG and Clinical Neurophysiology, 86,* 408–417.

Rey, A. (1964). *L'examen clinique psychologie* [The clinical exam in psychology]. Paris: Presses Universitaires de France.

Richardson-Klavehn, A., & Bjork, R. A. (1988). Measures of memory. *Annual Review of Psychology, 39,* 475–542.

Schacter, D. L. (1987). Implicit memory: History and current status.

Journal of Experimental Psychology: Learning, Memory and Cognition, 13, 501–518.

Schacter, D. L. (1994). Priming and multiple memory systems: Perceptual mechanisms of implicit memory. In D. L. Schacter & E. Tulving (Eds.), *Memory systems, 1994* (pp. 223–268). Cambridge, MA: MIT Press.

Schacter, D. L., Chiu, C. Y., & Ochsner, K. N. (1993). Implicit memory: A selective review. *Annual Review of Neuroscience, 16,* 159–182.

Schacter, D. L., Church, B. A., & Treadwell, J. (1994). Implicit memory in amnesic patients: Evidence for spared auditory priming. *Psychological Science, 4,* 331–335.

Schacter, D. L., Cooper, L. A., & Treadwell, J. (1993). Preserved priming of novel objects across size transformation in amnesic patients. *Psychological Science, 4,* 331–335.

Scoville, W. B., & Milner, B. (1957). Loss of recent memory after bilateral hippocampal lesions. *Journal of Neurology, Neurosurgery, and Psychiatry, 20,* 11–21.

Shimamura, A. P. (1986). Priming effects in amnesia: Evidence for a dissociable memory function. *Quarterly Journal of Experimental Psychology, 38A,* 619–644.

Shimamura, A. P., & Squire, L. R. (1989). Impaired priming of new associations in amnesia. *Journal of Experimental Psychology: Learning, Memory and Cognition, 15,* 721–728.

Snodgrass, J. G. (1989). Sources of learning in the picture fragment completion task. In S. Lewandowsky, J. C. Dunn, & K. Kirsner (Eds.), *Implicit memory theoretical issues* (pp. 259–282). Hillsdale, NJ: Erlbaum.

Squire, L. R. (1982). The neuropsychology of human memory. *Annual Review of Neuroscience, 5,* 241–273.

Squire, L. R., Amaral, D. G., & Press, G. A. (1990). Magnetic resonance measurements of hippocampal formation and mammillary nuclei distinguish medial temporal lobe and diencephalic amnesia. *Journal of Neuroscience, 10,* 3106–3117.

Squire, L. R., & Frambach, M. (1990). Cognitive skill learning in amnesia. *Psychobiology, 18,* 109–117.

Squire, L. R., Knowlton, B., & Musen, G. (1993). The structure and organization of memory. *Annual Review of Psychology, 44,* 453–495.

Squire, L. R., & Shimamura, A. P. (1986). Characterizing amnesic patients for neurobehavioral study. *Behavioral Neuroscience, 100,* 866–877.

Squire, L. R., & Zola-Morgan, M. (1991). The medial temporal lobe memory system. *Science, 253,* 1380–1386.

Tulving, E. (1985). Memory and consciousness. *Canadian Psychologist, 26,* 1–12.

Tulving, E., & Schacter, D. L. (1990). Priming and human memory systems. *Science, 247,* 301–306.

Van Hoesen, G. W., Hyman, B. T., & Damasio, A. R. (1991). Entorhinal cortex pathology in Alzheimer's disease. *Hippocampus, 1,* 1–8.

Vonsattel, J. P., Myers, R. H., Stevens, T. J., Ferrante, R. J., Bird, E. D., & Richardson, E. P., Jr. (1985). Neuropathological classification of Huntington's disease. *Journal of Neuropathology and Experimental Neurology, 44,* 559–577.

Warrington, E. K. (1984). *Recognition Memory Test.* Windsor, England: NFER-Nelson.

Wechsler, D. (1981). Wechsler Adult Intelligence Scale—Revised [manual]. New York: Psychological Corp.

Wechsler, D. (1987). Wechsler Memory Scale—Revised [manual]. New York: Psychological Corp.

Weiskrantz, L. (1990). Some contributions of neuropsychology of vision and memory to the problem of consciousness. In A. Marcel & E. Bisiach (Eds.), *Consciousness and contemporary science* (pp. 183–199). New York: Oxford University Press.

Zola-Morgan, S., & Squire, L. R. (1993). Neuroanatomy of memory. *Annual Review of Neuroscience, 16,* 547–563.

Zola-Morgan, S., Squire, L. R., & Amaral, D. G. (1986). Human amnesia and the medial temporal region: Enduring memory impairment following a bilateral lesion limited to field CA1 of the hippocampus. *Journal of Neuroscience, 6,* 2950–2967.

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