Research Article

IMPLICIT MEMORY IN AMNESIC PATIENTS: Impairment of Voice-Specific Priming

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Abstract—*Amnesic patients generally exhibit spared priming effects on implicit memory tasks despite poor explicit memory. In a previous study, we demonstrated normal auditory priming in amnesic patients on an identification-in-noise test in which the magnitude of priming is independent of whether the speaker's voice is the same or different at study and test. In the present experiment, we examined auditory priming on a filter identification test in which the magnitude of priming in control subjects is higher when the speaker's voice is the same at study and test than when it is different. Amnesic patients, by contrast, failed to exhibit more priming in a same-voice condition than in a different-voice condition. Voice-specific priming may depend on a memory system that is impaired in amnesia.*

It is well known that damage to limbic and diencephalic brain structures often produces an amnesic syndrome in which patients exhibit a general impairment of explicit memory for recently encountered information (cf. Squire, 1994; Weiskrantz, 1985). Despite their difficulties remembering previous experiences, however, it has been well established that amnesics can exhibit robust and frequently normal implicit memory for various kinds of information. The most extensively studied form of implicit memory in amnesia is the phenomenon of repetition or direct *priming:* facilitated identification of degraded words or objects as a consequence of prior exposure to them (e.g., Tulving & Schacter, 1990). Numerous studies have demonstrated that priming effects can be fully preserved in amnesic patients across a wide variety of materials and tests (for reviews, see Bowers & Schacter, 1993; Shimamura, 1986). This finding has been taken as evidence that priming is mediated by a memory system that does not depend on the limbic and diencephalic structures that are damaged in amnesia (e.g., Schacter, 1994; Squire, 1994).

Although virtually all demonstrations of preserved priming in amnesic patients have used visual materials and tests, we have recently developed paradigms and procedures for examining auditory priming. In a study of college students, we (Schacter & Church, 1992) documented priming effects on a task in which subjects attempt to identify words that are masked in white noise. The observed auditory priming was largely unaffected by depth-of-encoding manipulations or by study-to-test changes in speaker's voice (cf. Jackson & Morton, 1984). Another study (Schacter, Church, & Treadwell, 1994) examined whether such priming is preserved in amnesia. Amnesic patients and control subjects heard a series of spoken words and judged either the category to which each word belongs (semantic encoding task) or the pitch of the speaker's voice (nonsemantic encoding task); half of the speakers were males and half were females. Priming was assessed with an auditory identification test in which studied and nonstudied words were presented in white noise and subjects reported what they heard. Half of the studied words were spoken in the same voice as during the encoding task, and half were spoken in a different voice; voice change always involved a change in the speaker's gender (i.e., male–female or female–male). The experiment revealed that (a) amnesic patients showed just as much priming as control subjects, (b) the semantic encoding task and the nonsemantic encoding task yielded similar levels of priming in both subject groups, and (c) priming effects were nearly identical in the same-voice and different-voice conditions for both amnesics and control subjects. In addition, amnesics were impaired on an explicit recognition test that followed the identification test.

These data provide clear evidence that auditory priming, like visual priming, can be preserved in amnesic patients. One important finding was that priming in both amnesic patients and control subjects was unaffected by study-to-test changes in speaker's voice. Other experiments with college students, however, have revealed that study-to-test changes in speaker's voice can influence the magnitude of priming on auditory completion and identification tests that do not make use of white noise (Church & Schacter, 1994; Schacter & Church, 1992). On these tests, we have observed evidence for voice-specific priming as a consequence of study-to-test changes in speaker's gender, linguistic or emotional intonation within a single voice, and the fundamental frequency of a single speaker's voice. Thus, auditory priming appears to include a component that is specific to speaker's voice, which depends on acoustic or prosodic information, as well as a nonspecific component that depends on abstract phonological information (see Schacter, 1994).

The finding that amnesics exhibit normal implicit memory on the identification-in-noise test indicates preservation of voice-nonspecific priming, but leaves open the question of whether the voice-specific component of auditory priming is also intact in amnesics. The purpose of the present experiment was to investigate whether amnesic patients exhibit normal levels of voice-specific priming on a test that is known to yield such effects in nonamnesic subjects.

To investigate voice-specific priming, we used our low-pass filter identification test (described in Church & Schacter, 1994). Words presented on this test are degraded by reducing the decibel level of a distribution of higher frequencies, with the result that the words sound somewhat muffled, as if spoken from the other side of a wall. The general experimental paradigm was identical in most respects to Experiment 1 of our 1994 study (Church & Schacter, 1994): Subjects were exposed to a series of 24 words spoken by male and female speakers, and after a delay of several minutes were given the filter identification test for

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studied and nonstudied words; half of the studied words were spoken in the same voice, and half were spoken in a different voice. Subjects were then given an explicit yes/no recognition test for all words.

METHOD

Subjects

Twelve amnesic patients and 12 control subjects participated in the experiment. The amnesic patients had all been screened at the Memory Disorders Research Center of the Boston Veterans Administration Medical Center. Four of the patients became amnesic as a consequence of alcoholic Korsakoff's syndrome, and 8 of them became amnesic as a consequence of other, nonalcoholic etiologies (encephalitis, anoxia, thalamic infarct, ruptured aneurysm of the anterior communicating artery). The amnesic patients' mean age was 51.1 years, and they had on average 13.4 years of education. The amnesics' overall level of intellectual function was in the normal range, as indicated by their mean Verbal IQ of 100.9 on the Wechsler Adult Intelligence Scale-Revised (WAIS-R). They also exhibited normal attentional abilities, as indicated by a mean score of 101.7 on the Attention index of the Wechsler Memory Scale-Revised (WMS-R). By contrast, they consistently exhibited severe deficits on a variety of explicit memory tests. Table 1 displays the test scores of each amnesic patient on three indices of the WMS-R (General Memory, Delayed Memory, and Attention), together with age, verbal IQ, and results from the present experiment.

A group of 12 control subjects was also tested. Six of the control subjects had a history of alcoholism, and 6 had no history of alcoholism. The mean age of these subjects was 53.8 years, they had on average 14.1 years of education, and their mean Verbal IQ on the WAIS-R was 108.9.

Materials

The target materials consisted of 48 familiar words (see Schacter & Church, 1992, Experiment 3, for details) that were divided into two subsets of 24 words each. The two subsets were matched for frequency, first letter, number of syllables, and length (Graf & Williams, 1987; Kucera & Francis, 1967). We recorded words on a Macintosh computer with a Macrecorder (sampling rate = 22k), and then passed each word three times through the low-pass filter function that is part of the SoundEdit program. On each pass through the filter, the intensity of a distribution of frequencies above 2 kHz is reduced by 20 dB, and the intensity of a distribution of frequencies between 1 kHz and 2 kHz is reduced between 5 dB and 20 dB, with the highest frequencies being reduced the most in a sloping function.

Three male and three female speakers were recorded to yield two versions of each of the two study lists, the filter identification test, and the recognition test. Any word that was spoken by a male on one version of a tape was spoken by a female on the other, and vice versa. The four study list tapes each contained

Table 1. *Characteristics of the amnesic patients and the number of responses to studied and nonstudied words on the identification and recognition tests as a function of speaker's voice*

			WMS-R			ID			RN		
Etiology	Age	VIQ	GM	ATN	DLY	S	D	NS	S	D	NS
Korsakoff's	65	93	76	109	62	4	7	2	9	9	12
Korsakoff's	58	87	84	93	65	2	2	2	4	1	1
Korsakoff's	50	96	65	83	51	5	6	8	1	0	0
Korsakoff's	71	119	85	110	62	2	5	7	9	10	10
ACAA	66	125	95	99	53	3	4	12	6	5	11
ACAA	50	87	50	95	50	2	2	5	8	8	14
Anoxia	55	100	65	87	61	3	5	5	4	5	3
Anoxia	34	104	78	121	< 50	5	11	11	10	8	11
Anoxia	29	87	62	118	< 50	8	5	11	8	6	10
Encephalitis	42	111	81	107	69	7	8	8	6	8	6
Encephalitis	65	126	102	114	< 50	6	3	9	5	2	5
Thalamic infarct	51	84	79	89	76	4	4	6	3	4	2
Mean	51.1	100.9	76.9	101.7	58.3	4.3	5.3	7.2	6.1	5.5	7.1

Note. ACAA = ruptured aneurysm of the anterior communicating artery. VIQ = Verbal IQ from the Wechsler Adult Intelligence Scale-Revised. WMS-R = Wechsler Memory Scale-Revised; scores are presented separately for the indices of General Memory (GM), Attention (ATN), and Delayed Memory (DLY). The WMS-R does not provide scores below 50, and 50 was the lowest score used for computing means. ID = filter identification test; S = same-voice condition; D = different-voice condition; NS = nonstudied items. The number of correct responses in each condition is shown for this test. For S and D, the maximum number of correct responses was 12 for each condition; for NS, the maximum number of correct responses was 24. RN = yes/no recognition test; S = same-voice condition; D = different-voice condition; NS = nonstudied items. The number of "yes" responses in each condition is shown for this test. For S and D, the maximum number of "yes" responses (hits) was 12 for each condition; for NS, the maximum number of "yes" responses (false alarms) was 24.

24 words spoken clearly. The two filter identification tapes each included 48 degraded words, 24 that had been studied previously and 24 that had not been studied; the two recognition tapes each contained 48 words spoken clearly, 24 that had been studied and 24 that had not been studied (all of which had been presented on the filter identification test). On both the identification and the recognition tasks, half of the words were presented in the same voice as on the study task, and half of the words were presented in a different voice. Words that were presented in the same-voice condition on the filter test were presented in the different-voice condition on the recognition test, and vice versa. All words were presented using a cassette deck and headphones.

Design and Procedure

The experiment used a mixed-factorial design. The between-subjects variable was subject group (amnesic vs. control), and the within-subjects variables were item type (studied vs. nonstudied), speaker's voice (same vs. different), and type of test (low-pass filter vs. yes/no recognition). The same words were used on both the filter test and the recognition test. Half of the words had been studied previously, and half had not been studied previously; among the studied words, half were spoken in the same voice as during the study task, and half were spoken in a different voice. Words were counterbalanced across studied and nonstudied conditions, and same- and different-voice conditions.

All subjects were tested individually. During the encoding task, 24 words were presented auditorily, and subjects were asked to rate how clearly each speaker enunciated each word on a 4-point numeric scale (4 = well enunciated; 1 = poorly enunciated). There were 5 s between items for subjects to make their ratings. Subjects then performed a distractor task during which they generated the names of 15 cities beginning with the letters given in their booklets. The task required approximately 3 to 4 min to complete. After the distractor task, subjects were given the filter identification test. Subjects were told that they would hear a series of muffled words, that we were interested in their subjective perceptions of the words, and that they should respond by providing the first word that came to mind in response to each stimulus. Upon completion of the filter task, subjects were given the explicit recognition test. On this test, studied and nonstudied words were spoken clearly, and subjects were instructed to respond "yes" when they remembered the word from the study phase, and "no" when they did not remember the word from the study phase. Upon completion of the experiment, all subjects were debriefed.

RESULTS

Recognition Memory

The mean proportions of hits and false alarms for amnesics and control subjects are displayed in Table 2, and the raw data from individual amnesic patients are shown in Table 1. An analysis of variance was performed on corrected recognition scores that were computed by subtracting the false alarms from hits for each subject. In line with previous research, amnesic patients exhibited much lower levels of recognition accuracy than did control subjects, as indicated by a highly significant main effect of subject group, $F(1, 22) = 53.57$, $MS_e = .030$, $p < .0001$. There were trends for voice effects in recognition, but the main effect of speaker's voice was not significant, $F(1, 22) = 3.52$, $MS_e = .009$, and separate analyses of amnesic and control subjects revealed that voice effects were not significant in either group, $ts(11) = 1.44$ and 1.23 for control subjects and amnesics, respectively (see Church & Schacter, 1994, and Schacter & Church, 1992, for similar results). The Subject Group × Speaker's Voice interaction failed to approach significance, $F(1, 22) < 1$, $MS_e = .009$.

Table 2. *Proportion of studied and nonstudied words identified correctly on the filter identification test and proportion of hits and false alarms on the recognition memory test as a function of speaker's voice*

	Type of test							
	Identification				Recognition			
Subject group	S	D	*M*	NS	S	D	*M*	NS
Amnesic patients	.35 (.05)	.43 (.13)	.39 (.09)	.30	.51 (.21)	.46 (.16)	.49 (.19)	.30
Control subjects	.56 (.20)	.40 (.04)	.48 (.12)	.36	.89 (.58)	.83 (.52)	.86 (.55)	.31

Note. S = same voice; D = different voice; NS = nonstudied words. Values in parentheses for the identification test are priming scores computed by subtracting the proportion of nonstudied words reported from the proportion of studied words reported for a particular condition. For the recognition test, the displayed proportions of "yes" responses are hit rates for studied words (same and different voice) and false alarm rates for nonstudied words. Values in parentheses for the recognition test are corrected recognition scores computed by subtracting the proportion of false alarms for nonstudied words from the proportion of hits for studied words.

Filter Identification

Table 2 presents the proportion of studied and nonstudied words identified correctly by amnesic patients and control subjects, and Table 1 presents the raw identification data for each amnesic. Identification accuracy for nonstudied words was somewhat higher in the control group (.36) than in the amnesic patients (.30), but the difference was not significant, $t < 1$. Moreover, this nonsignificant trend was entirely attributable to the low baseline performance of the 4 Korsakoff's patients (cf. Hamman, Squire, & Schacter, in press; Schacter et al., 1994).

Both amnesic patients and control subjects identified more studied words than nonstudied words, thus indicating that priming occurred, $t(11) = 2.13, p < .05$, and $t(11) = 4.03, p < .001$, for amnesic and control subjects, respectively. However, the pattern of priming as a function of the voice manipulation differed sharply for the two groups: Control subjects showed considerably more priming in the same-voice condition than in the different-voice condition, whereas amnesic patients exhibited a trend in the opposite direction. Analysis of variance was performed on priming scores that were computed by subtracting each subject's baseline identification rate from the proportion of studied items identified correctly in same- and different-voice conditions, respectively. The analysis revealed a significant Speaker's Voice × Subject Group interaction, $F(1, 22) = 5.81$, $MS_e = .027$, $p < .03$, and no other significant effects. Priming scores in same- and different-voice conditions differed significantly for control subjects, $t(11) = 2.35, p < .05$, but not for amnesic patients, $t(11) = 1.27$.

Note that amnesic patients' overall priming score (.09) was only slightly less than that of control subjects (.12), even though the pattern of priming differed significantly. This effect is attributable to the fact that amnesics exhibited significant priming in the different-voice condition, $t(11) = 2.13, p < .05$, whereas control subjects did not, $t(11) < 1$. However, the difference between amnesic and control subjects in this condition was not significant, $t(22) < 1$, in part because the lack of different-voice priming by control subjects is largely attributable to a single aberrant subject who identified many fewer words in the different-voice condition than in the baseline condition. When the data from this single subject are excluded, the remaining control subjects exhibit marginally significant priming in the different-voice condition, $t(10) = 1.71, p < .07$.

DISCUSSION

The key result of the present experiment is that amnesic patients failed to exhibit more priming in the same-voice condition than in the different-voice condition. Control subjects, by contrast, showed significantly more priming in the same- than in the different-voice condition, thus replicating and extending previous findings of voice-specific priming with college students (Church & Schacter, 1994; Schacter & Church, 1992).

Our results contrast with the frequently reported observation that perceptual priming is normal in amnesic patients (for review, see Bowers & Schacter, 1993; Shimamura, 1986). Thus, it is important to consider whether the result may be an artifact of our procedures. One possibility that must always be confronted when amnesics show abnormal priming is that the effect is attributable to the use of intentional retrieval strategies in control subjects. Thus, voice-specific priming in our control group might be "contaminated" by the use of intentional retrieval strategies. However, this possibility seems unlikely because the control subjects did not exhibit significant voice-change effects on the recognition test, when they were asked to intentionally remember the target words. Moreover, previous research has shown that in college students, voice-specific priming can be observed under conditions in which possible contamination from intentional retrieval strategies can be ruled out (Church & Schacter, 1994; Schacter & Church, 1992).

Another possible source of artifact is hearing impairment. We excluded patients with hearing problems, but a few of our patients are elderly, and aging often produces loss of sensitivity to high frequencies. There are several reasons why hearing impairment is an unlikely explanation for our results, however. First, our control subjects were age-matched to the amnesic patients, and they exhibited robust voice-specific priming. Second, baseline identification performance did not differ significantly between amnesic and control subjects; hearing problems should have produced depressed levels of baseline identification performance. Third, in separate studies, we have failed to observe voice-specific priming in elderly adults who possess normal hearing and exhibit high levels of baseline identification performance (Schacter, Church, & Osowiecki, 1994).

If the absence of voice-specific priming in amnesics is not attributable to the foregoing factors, how are we to think about the result? We have argued previously that auditory priming on completion and identification tests depends largely on a presemantic perceptual representation system (PRS). The PRS can be viewed as a collection of cortically based domain-specific subsystems that represent information about the form and structure, but not the meaning and associative properties, of words and objects (e.g., Schacter, 1990, 1994; Tulving & Schacter, 1990). Because the PRS does not depend on limbic and diencephalic structures, we have argued that preserved visual priming effects in amnesic patients are attributable to preserved PRS subsystems. With respect to auditory priming, we have argued on various grounds that two PRS subsystems may be involved: an auditory word form subsystem that represents abstract information about the phonological structure of words and an acoustic subsystem that represents prosodic features of speaker's voice (see Schacter, 1994, for discussion).

Our previous finding (Schacter et al., 1994) that amnesics exhibit normal auditory priming on the identification-in-noise test, for which no voice-change effects are observed even in college students, suggests that the phonological word form subsystem of PRS is preserved in amnesia. The absence of voice-specific priming in amnesics leads us to suggest that in order to exhibit voice-specific priming on the filter identification test, it may be necessary to bind together phonological information concerning a spoken word form and acoustic information concerning the voice of the speaker who enunciates the word. Further, such binding may require the participation of limbic and diencephalic structures that are damaged in amnesic patients. A number of investigators have argued that a major function of the limbic-diencephalic system is to bind together the outputs of various different systems and subsystems (cf. Johnson & Chalfonte, 1994; Moscovitch, 1994; Schacter, 1994; Squire, 1994).

Although such bound traces normally provide the basis for explicit recall and recognition, we are suggesting that they can also influence priming. Thus, voice-specific priming may not depend on the PRS alone.

We acknowledge that our account is preliminary; we offer it as a suggestive hypothesis that merits systematic evaluation in future research. It should be noted, however, that this basic idea is consistent with two other sets of studies in which amnesic patients were characterized by priming impairments. Kinoshita and Wayland (1993) recently reported that control subjects exhibit more visual priming on a fragment completion test when typography is the same at study and test than when it differs. Korsakoff's amnesics, however, show similar levels of priming in same-typography and different-typography conditions. This visual specificity effect may require binding between visual features of words and abstract orthographic word forms. Schacter and Graf (1986) found that control subjects and patients with mild memory disorders exhibit more stem completion priming when the associative context of a studied word is the same at study and test than when associative context is different at study and test. However, they also reported that severely amnesic patients do not exhibit context-specific priming of new associations, and Shimamura and Squire (1989) likewise failed to observe context-specific priming in amnesic patients. There are reasons to believe that these contextual priming effects require binding between semantic and visual properties of words (for discussion, see Schacter, 1994).

These points of convergence from different lines of research suggest that it may be useful to distinguish between two forms of priming, which we refer to descriptively as Type A and Type B. Type A priming is supported by the PRS, is preserved in amnesic patients, and depends on relatively abstract perceptual information. Type B priming results from an interaction between the PRS and the limbic-diencephalic structures that ordinarily support explicit memory, is impaired in amnesic patients, and depends on highly specific perceptual or contextual features that have been bound together with abstract phonological, orthographic, or semantic representations.

In light of this distinction, it may be questioned whether voice-specific priming and other Type B effects should be viewed as implicit memory phenomena. It is unlikely for reasons noted earlier that voice-specific effects are the result of intentional, voluntary retrieval processes, and it is also unlikely that context-specific priming of new associations is attributable to intentional retrieval (see, e.g., Schacter & Graf, 1986; Schacter, Bowers, & Booker, 1989). Another possibility, however, is that such effects are based on unintentional, involuntary retrieval processes, but also involve some conscious awareness of the study episode; that is, voice-specific and other Type B priming effects (e.g., typefont-specific and context-specific effects) might be better described as instances of involuntary conscious memory (see Richardson-Klavehn, Gardiner, & Java, 1994; Schacter 1987) than as instances of implicit memory (for further discussion, see Schacter, 1994). Available data do not allow us to address this point conclusively, but it is clearly a key issue for future investigations of priming.

One further feature of our data merits consideration. As noted earlier, even though amnesics failed to exhibit voice-specific priming, their overall priming score was comparable to that of control subjects. Schacter and Graf (1986) found that severely amnesic patients, who did not exhibit context-specific priming, nevertheless showed the same overall level of priming as mildly amnesic patients, who did exhibit context-specific priming; the severely amnesic patients showed more priming in the different-context condition than did the mildly amnesic patients. Similarly, Shimamura and Squire (1989) found that amnesic patients, who failed to exhibit significantly more priming in a same- than a different-context condition, showed slightly higher levels of priming in the different-context condition than did control subjects. These findings might provide clues concerning the relations between the two kinds of priming that we have distinguished. For example, the processes underlying Type A and Type B priming may not be additive, and might even be mutually inhibitory. When one type of priming process is impaired, nonimpaired processes may "take over," so that overall levels of priming are not changed even though the qualitative pattern of priming is altered. Future studies that examine the relations between spared and impaired components of priming in amnesic patients are likely to illuminate this intriguing but little-understood phenomenon.

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