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Implicit memory in amnesic patients: When is auditory priming spared?

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Abstract

Amnesic patients often exhibit spared priming effects on implicit memory tests despite poor explicit memory. In previous research, we found normal auditory priming in amnesic patients on a task in which the magnitude of priming in control subjects was independent of whether speaker's voice was same or different at study and test, and found impaired voice-specific priming on a task in which priming in control subjects is higher when speaker's voice is the same at study and test than when it is different. The present experiments provide further evidence of spared auditory priming in amnesia, demonstrate that normal priming effects are not an artifact of low levels of baseline performance, and provide suggestive evidence that amnesic patients can exhibit voice-specific priming when experimental conditions do not require them to interactively bind together word and voice information. (*JINS*, 1995, *1*, 434-442.)

Keywords: Amnesia, Priming, Implicit memory

Introduction

It is well known that amnesic patients can exhibit robust implicit memory despite impaired explicit memory. The most extensively studied form of implicit memory in amnesic patients is known as direct or repetition priming, where exposure to a word or object influences subsequent identification of that item (for review, see Roediger & McDermott, 1993; Schacter et al., 1993). Many experiments have shown that priming effects can be preserved in amnesic patients across a wide variety of materials and tests (for review, see Shimamura, 1986; Bowers & Schacter, 1993). This finding has been taken as evidence that priming is mediated by a memory system that does not depend on the medial temporal lobe/diencephalic structures that are damaged in amnesia (e.g., Gabrieli et al., 1994; Schacter, 1994; Squire, 1994).

Although most demonstrations of preserved priming in amnesic patients have used visual materials and tests, we recently reported experiments that examine auditory priming in amnesia. In a study by Schacter and colleagues (Schacter et al., 1994), amnesic patients and control subjects heard a list 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. Subjects then performed 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. The experiment revealed equivalent amounts of priming in amnesic patients and control subjects in all experimental conditions. In contrast, amnesics exhibited a significant impairment on an explicit recognition test that followed the identification test.

These data indicate that auditory priming can be preserved in amnesic patients. Note, however, that priming in both amnesics and controls was unaffected by study-to-test changes in speaker's voice—that is, the magnitude of priming was nearly identical in the same-voice and different-voice conditions for both amnesics and controls. This outcome was not surprising, because previous research with college students had already shown that priming on the identification-in-noise test is not significantly influenced by voice change (Schacter & Church, 1992). But other studies of college students have revealed that

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study-to-test changes in speaker's voice does influence the magnitude of priming on auditory completion and identification tests that do not make use of white noise (Schacter & Church, 1992; Church & Schacter, 1994). Thus, auditory priming is comprised of a component that is specific to speaker's voice as well as a nonspecific component (cf. Kirsner et al., 1989). Schacter et al.'s (1994) data suggest that the nonspecific component of auditory priming is spared in amnesic patients.

To determine whether the voice-specific component of auditory priming is also preserved in amnesics, Schacter et al. (1995) used a low-pass filter identification test developed by Church and Schacter (1994). Words presented on this test are degraded by reducing the decibel level of a distribution of higher frequencies, so that words are muffled and hence difficult to identify. Schacter et al. (1995) found that control subjects exhibited more priming when speaker's voice was the same at study and test than when it was different. In contrast, amnesic patients exhibited similar amounts of priming in same- and different-voice conditions.

Why do amnesic patients exhibit apparently normal levels of nonspecific auditory priming together with impaired voice-specific priming? We have argued previously that auditory priming on completion and identification tests depends largely on a presemantic perceptual representation system (PRS) that is preserved in amnesic patients. The PRS is 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). With respect to auditory priming, we have suggested 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. The former subsystem, we contended, subserves the component of priming that is insensitive to changes in speaker's voice, while the latter subsystem subserves priming effects that are sensitive to changes in speaker's voice.

Our finding that amnesics exhibit normal auditory priming on the identification-in-noise test (Schacter et al., 1994), which failed to yield evidence of voice-specific priming even in our control subjects, suggests that the phonological word form subsystem of PRS is preserved in amnesia. In contrast, the absence of voice-specific priming in amnesics (Schacter et al., 1995) led us to suggest that in order to exhibit voice specific priming on the filter identification test under the conditions of our experiments, it may be necessary to bind together phonological information concerning a spoken word form and acoustic information concerning the voice of the speaker enunciating the word. Furthermore, such binding may require the participation of medial temporal lobe/diencephalic structures that are damaged in amnesic patients. Several investigators have argued that a major function of the medial temporal lobe/diencephalic system is to bind together the outputs of various different systems and subsystems (cf. Cohen & Eichenbaum, 1993; Johnson & Chalfonte, 1994; Moscovitch, 1994; Schacter, 1994; Squire, 1994). Although such bound traces normally provide the basis for explicit recall and recognition, Schacter et al. (1995) suggested that they can also influence implicit test performance.

Schacter et al. (1995) also noted that a similar idea could be applied to findings of impaired typefont-specific priming in Korsakoff amnesics (Kinoshita & Wayland, 1993) and impaired priming of novel associations in amnesic patients of mixed etiologies (e.g., Schacter & Graf, 1986; Shimamura & Squire, 1989). Typefont-specific priming may require binding between visual features of words and abstract orthographic word forms, and priming of new associations may require binding between semantic and visual features of words.

In summary, one interpretation of extant data on auditory priming in amnesic patients is that a phonological component of priming that depends on the PRS is preserved and a voice-specific component that requires episodic binding is impaired. However, alternative interpretations of these data are also possible. For example, Ostergaard and Jernigan (1993) have recently argued that priming is not preserved in amnesic patients. To support this claim, they have pointed to the existence of impaired priming in several studies, nonsignificant trends for priming impairments in other studies that may have failed to detect significant differences because of low statistical power, and differences in baseline levels of performance between amnesics and controls that can cloud interpretation of the priming data (but see also Hamman et al., 1995). Thus, Schacter et al.'s (1995) failure to observe voice-specific priming in amnesic patients may simply reflect the fact that auditory priming in general is not preserved in amnesic patients. There may be no need to postulate, as Schacter et al. (1995) did, that one kind of auditory priming is preserved in amnesic patients and another kind is impaired. The observation of normal priming by Schacter et al. (1994) may have limited generality, or might even be an unreplicable phenomenon.

To address these issues, and to provide more information about the voice-specific component of priming in amnesic patients, we describe two experiments that examine auditory priming in amnesics and controls. One purpose of these experiments is to determine whether amnesic patients can exhibit voice-specific priming under conditions in which voice-specific effects do not require binding between words and voices. In the Schacter et al. (1995) experiment, all of the voices that were used on the filter identification test had already appeared on the study list. Thus, all of the test voices were equally familiar to the subjects, and voice-specific priming could be exhibited only when subjects had retained a specific association between a particular target word and a speaker's voice. Consider, however, an experiment in which all of the target

words are spoken by a single voice during the study phase. On a subsequent auditory identification test, studied and nonstudied words are spoken either by the same (familiar) voice or by an entirely unfamiliar voice that had not appeared at all during the study phase. More priming in a same- than a different-voice condition could occur on such a test based solely on information about the familiar speaker's voice that was acquired during the study episode; it is not necessary to form a specific association between each target word and a particular speaker's voice. Indeed, Nygaard et al. (1994) have demonstrated such a voice familiarization effect in college students.

A voice familiarization effect—that is, more priming for words tested with a familiar voice than with an unfamiliar voice—does not require any word/voice binding. In terms of the PRS hypothesis noted earlier, a voice familiarization effect could be supported solely by an acoustic PRS subsystem; the outputs of the phonological and acoustic subsystems need not be linked together. Accordingly, if amnesic patients in the Schacter et al. (1995) experiment failed to exhibit voice-specific priming because this effect required binding between a word and a voice, then they ought to exhibit voice-specific effects, and normal priming more generally, when no word/voice binding is required. On the other hand, if auditory priming in general is not preserved in amnesic patients, then there should be evidence of impaired auditory priming even when no word/voice binding is necessary.

Experiment 1

In Experiment 1, we investigated the foregoing issues by exposing amnesic patients and control subjects to a series of words that were all spoken by a single speaker. We then administered an auditory identification test in which half of the words were spoken by the familiar voice and half were spoken by an unfamiliar voice.

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 eight of them became amnesic as a consequence of other, nonalcoholic etiologies (encephalitis, anoxia, thalamic infarct, ruptured anterior acommunicating artery aneurysm). The amnesic patients' mean age was 53.1 yr and they averaged 13.4 yr of education. The amnesics' overall level of intellectual function was in the normal range, as indicated by their mean Verbal IQ of 101.1 on the Wechsler Adult Intelligence Scale-Revised (WAIS-R). In contrast, they consistently exhibited severe deficits on a variety of explicit memory tests. For example, on the Wechsler Memory Scale-Revised (WMS-R), their score on the General Memory Index was 76.9 and their score on the Delayed Memory Index was 58.3, which indicate severe impairments (each index of the WMS-R, like the WAIS-R, produces a mean of 100 and standard deviation of 15 in the normal population). However, they achieved a mean score of 101.8 on the Attention Index of the WMS-R, which confirms that their impaired performance on the other WMS-R indices is attributable to memory deficit and not attentional deficit (see Schacter et al., 1995, for more details on individual patients).

A group of 12 control subjects was also tested. Four of the control subjects had a history of alcoholism, and 8 had no history of alcoholism. The mean age of the control subjects was 54.6 yr, they averaged 14.2 yr of education, and their mean verbal IQ on the WAIS-R was 104.6.

Materials

The target materials consisted of 48 familiar words that were divided into two subsets of 24 words each. The two subsets were matched for frequency, first syllable, number of syllables, and length (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 lowpass 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 was reduced by 20 dB and the intensity of a distribution of frequencies between 1 kHz and 2 kHz was reduced between 5 dB and 20 dB, with the highest frequencies being reduced the most in a steeply sloping function.

One male and one female speaker 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 the male on one version of a tape was spoken by the female on the other, and vice versa. The four study list tapes each contained 24 words spoken clearly by either the male or the female speaker. 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 recognition tasks, half of the words were presented in the same voice as on the study task (male/male or female/female) and half of the words were presented in the different voice (male/female or female/male); 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 the different voice. Words were counterbalanced across studied and nonstudied conditions and across 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 the speaker enunciated each word on a four point numeric scale, (4 = well enunciated; 1 = poorly enunciated). There were five seconds 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 three to four minutes 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 the 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

Filter identification

Table 1 presents the proportion of studied and nonstudied words identified correctly by amnesic patients and control subjects as a function of whether speaker's voice was familiar or unfamiliar. Four points about these data are worth noting. First, there was a trend for more accurate identification of words spoken in a familiar voice than in an unfamiliar voice for both studied and nonstudied items. Second, control subjects showed a higher baseline identification rate for nonstudied items than did amnesic patients. Third, there was a large effect of study for amnesics and controls in both the familiar and unfamiliar voice conditions. Fourth, the magnitude of this priming effect was virtually identical in amnesics and controls.

Table 1. Proportion of studied and nonstudied words identified correctly on the Filter Identification Test as a function of speaker's voice in Experiment 1

	Studied words			Nonstudied words		
Subject group	F	U	M	F	U	M
Amnesic patients	.493	.444	.469	.361	.312	.337
Control subjects	.611	.535	.573	.458	.416	.437

F = Familiar voice; U = Unfamiliar voice; M = Mean.

An analysis of variance (ANOVA) revealed a highly significant main effect of Item Type (studied *vs.* nonstudied), $F(1,22) = 20.72$, $MSE = .001$, $p < .001$, indicating that priming occurred. The Item Type × Subject Group interaction did not approach significance, $F(1,22) < 1$, $MSE = .021$, reflecting the fact that amnesic patients and control subjects exhibited identical amounts and patterns of priming. The main effect of Speaker's Voice failed to approach significance, $F(1,22) = 1.76$, $MSE = .039$, $p = .198$, but there was a marginally significant main effect of Subject Group, $F(1,22) = 3.09$, $MSE = .082$, $p = .093$. No other effects approached significance (Fs < 1).

To examine priming more specifically, we obtained priming scores by subtracting the proportion of nonstudied words identified correctly from the proportion of studied words identified correctly. There are two different ways in which such an analysis could be performed: (1) subtracting the mean baseline score (collapsed across familiar and unfamiliar voices) from the proportion of studied words identified in the familiar and unfamiliar voice conditions, respectively; or (2) subtracting separately the proportion of nonstudied words identified correctly in the familiar and unfamiliar voice conditions from the corresponding proportions for studied words. We analyzed the data both ways and obtained the same pattern of results. Because the proportion of nonstudied words identified correctly did not differ significantly in the familiar and unfamiliar voice conditions ($F < 1$), we report the results of the first method of analysis.

As in the overall ANOVA, an ANOVA performed on priming scores indicated that the effect of Speaker's Voice failed to approach significance, $F(1,22) = 1.11$, $MSE = .042$. There was no effect of Subject Group, $F(1,22) < 1$, $MSE = .042$ nor was there any hint of a Subject Group × Speaker's Voice interaction, $F(1,22) < 1$, $MSE = .042$, indicating that the marginally significant trend for an effect of Subject Group in the overall ANOVA is attributable to group differences in baseline performance. We therefore performed a separate ANOVA on identification performance for nonstudied words. The main effect of Subject Group was marginally significant, $F(1,22) = 3.39$, $MSE = .024$, $p = .072$.

In previous studies of auditory priming, we have found that Korsakoff patients, as opposed to non-Korsakoff amnesics, exhibit impaired baseline performance. We observed a similar pattern of results in the present experiment. The overall baseline identification rate for the four Korsakoff patients (.250) was significantly lower than the control baseline [.437; $t(14) = 1.82$, $p < .05$], whereas the baseline rate for the eight non-Korsakoff patients did

not differ significantly from the control baseline [.380; $t(18) < 1$]. We also observed a trend for a larger priming effect in the Korsakoff subgroup. Collapsed across the familiar and unfamiliar voices, Korsakoff patients' overall priming score (i.e., proportion correct for studied words minus proportion correct for nonstudied words) was .188, whereas non-Korsakoff patients' overall priming score was .108. Control subjects' overall priming score was .136, which did not differ significantly from the priming scores of either subgroup of amnesic patients, $t(s) < 1$.

Recognition memory

The proportions of hits and false alarms for amnesics and controls are displayed in Table 2. Consistent with previous research, amnesic patients exhibited much lower levels of recognition accuracy than did control subjects. The amnesic patients exhibited trends for voice familiarity effects in their responses to both old and new items: They tended to say "old" more often to studied and nonstudied words that were spoken by the familiar speaker's voice than by the unfamiliar speaker's voice. However, control subjects exhibited no such trends.

To examine the apparent trend for voice familiarization effects in the amnesics, an ANOVA was performed on the proportion of subjects' "old" responses to studied and nonstudied words. There were highly significant effects of Item Type (studied *vs.* nonstudied), $F(1,22) = 43.55$, $MSE = .045$, $p < .001$, and Subject Group, $F(1,22) = 12.54$, $MSE = .086$, $p < .005$. However, the main effect of Speaker's Voice was not significant, $F(1,22) < 1$, $MSE = .014$, and the Subject Group × Speaker's Voice interaction did not attain significance, $F(1,22) = 2.44$, $MSE = .014$, $p = .133$. We also carried out a separate ANOVA on the data from the amnesic patients alone, and failed to observe a significant effect of Speaker's Voice, $F(1,22) = 1.20$, $MSE = .027$.

We also performed an ANOVA on corrected recognition scores that were computed by subtracting the false alarms from the hits for each subject. As with the priming analysis, corrected recognition scores could be computed either by subtracting a single false alarm rate from the hit rates in the familiar and unfamiliar voice conditions for each subject group, or by subtracting separately the proportion of false alarms in the familiar and unfamiliar voice conditions from the corresponding hit rates for studied words. We analyzed the data both ways and observed similar outcomes, so we report only the results of the former analysis. This ANOVA revealed a highly significant effect of subject group, $F(1,22) = 11.68$, $MSE = .054$, $p < .005$, a nonsignificant effect of speaker's voice, $F(1,22) < 1$, $MSE = .054$, and a nonsignificant interaction between the two variables, $F(1,22) < 1$, $MSE = .054$. The patterns of recognition performance for Korsakoff and non-Korsakoff amnesic subgroups was virtually identical: both exhibited substantial impairments of recognition accuracy.

Table 2. Proportion of "Yes" responses to studied words (Hits) and nonstudied words (False Alarms) on the Yes/No Recognition Test as a function of speaker's voice in Experiment 1

	Studied words			Nonstudied words		
Subject group	F	U	M	F	U	M
Amnesic patients	.444	.409	.427	.298	.215	.257
Control subjects	.743	.764	.754	.347	.361	.354

F = Familiar voice; U = Unfamiliar voice; M = Mean.

Discussion

The main purposes of Experiment 1 were to determine whether amnesic patients show normal voice familiarization effects when binding between words and voices is not required, and to assess the generality of previous findings of normal auditory priming in amnesic patients. The experiment is inconclusive concerning the first point, because we failed to observe a significant voice familiarization effect. Both control subjects and amnesic patients showed trends in the predicted direction, but there was considerable variability in the data, so the trends did not attain or approach statistical significance. With respect to the second point, our data replicate and extend Schacter et al.'s (1994) previous finding of spared auditory priming in amnesia: The magnitude and pattern of priming effects in amnesics and controls was indistinguishable. However, this finding must be treated cautiously because of between-group differences in baseline identification performance that were largely attributable to the low levels of baseline performance by the four Korsakoff patients.

The finding of impaired baseline identification performance in Korsakoff patients replicates previous observations in studies of auditory priming (Schacter et al., 1994, 1995) and visual priming (Hamman et al., 1995). In addition, we also observed a trend for higher levels of priming in Korsakoff than non-Korsakoff patients. According to Ostergaard and Jernigan's (1993) argument that low levels of baseline performance artifactually increase priming scores, these observations indicate that priming scores in the Korsakoff amnesics have been inflated by their overall lower level of baseline identification accuracy. Such an effect may have contributed to inflated priming scores in the entire amnesic group. Contrary to this argument, however, there are good reasons to believe that in our experiment the low baseline identification rate may have artifactually *decreased*, rather than increased, priming scores in the Korsakoff patients and in the entire amnesic group. Chapman et al. (1994) have pointed to the existence of a statistically artifactual curvilinear relationship between overall accuracy and priming scores in accuracy data of the kind that we have collected. Priming scores tend to be artifactually increased when perfor-

mance is at or close to 50% correct, and artifactually decreased as performance moves above or below the 50% level. In our experiment, control subjects' overall accuracy (.505) was almost exactly 50%, whereas amnesic patients' overall accuracy (.403) was considerably lower than 50%, largely because of the low overall accuracy level in the Korsakoff group (.344). To the extent that baseline artifacts operated in Experiment 1, they may have worked against the finding of normal priming by amnesic patients.

Internal analysis of the data from Experiment 1 revealed one potentially important factor that may have contributed both to the small voice familiarization effects that were observed, and to the impaired levels of baseline performance in the Korsakoff amnesics. We found that subjects in both groups consistently exhibited more difficulty identifying words spoken by the female speaker than by the male speaker. This observation is perhaps not surprising, because (a) the female speaker's voice had a higher fundamental frequency than did the male speaker's voice, (b) many of our subjects are elderly, including all of the Korsakoff patients, and (c) it is known that aging is associated with decreased sensitivity to higher frequencies in the speech signal. It is possible that problems associated with hearing words enunciated by the female speaker worked against finding voice familiarization effects and exacerbated impairments of baseline identification performance in the Korsakoff patients. We addressed these concerns in Experiment 2.

Experiment 2

Experiment 2 was identical to Experiment 1 except that two different male speakers were used. Studied words were spoken by one of the two speakers. On the subsequent identification and recognition tests, half of the words were spoken by the familiar male voice and half were spoken by the unfamiliar male voice.

According to our analysis, using two male speakers ought to eliminate the baseline differences between amnesics and controls that were observed in Experiment 1, but we should still observe normal priming effects in the amnesic group. This is because we believe that the artifact produced by low baseline levels of performance in Experiment 1 worked against finding normal priming in the amnesic group (Chapman et al., 1994). In contrast, according to Ostergaard and Jernigan's (1993) analysis, elimination of baseline differences between amnesics and controls should yield evidence of impaired priming in the amnesic patients.

Method

Subjects

The same 12 amnesic patients who participated in Experiment 1 also took part in Experiment 2. A group of 12 control subjects was also tested; half of them had taken part in Experiment 1 and half had not. Six of the control subjects had a history of alcoholism, and 6 had no history of alcoholism. The mean age of the control subjects was 53.9 yr, they averaged 13.4 yr of education, and their mean verbal IQ on the WAIS-R was 107.3.

Materials, design, and procedure

As in Experiment 1, the target materials consisted of 48 familiar words that were divided into two subsets of 24 words each. The two subsets were matched for frequency, first letter, number of syllables, and length (Kucera & Francis, 1967). The words were recorded and filtered in the same manner described in Experiment 1.

Two male speakers were recorded to yield two versions of each of the two study lists, the filter identification test, and the recognition test. A word that was spoken by one male speaker on one version of a tape was spoken by the second male speaker on the other version of the tape, and vice versa. Each of the four study list tapes contained 24 words spoken clearly by one of the male speakers. 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 recognition tasks, half of the words were presented in the same voice as on the study task (male 1/male 1 or male 2/male 2) and half of the words were presented in the different voice (male 1/male 2 or male 2/male 1); 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. As in Experiment 1, all words were presented using a cassette deck and headphones. The design and procedure of Experiment 2 were identical to those used in Experiment 1.

Results and discussion

Filter identification

Table 3 displays the proportion of studied and nonstudied words identified correctly by amnesic patients and control subjects as a function of the familiarity of the speaker's voice. The first point to note is that there was

Table 3. Proportion of studied and nonstudied words identified correctly on the Filter Identification Test as a function of speaker's voice in Experiment 2

	Studied words			Nonstudied words		
Subject group	F	U	M	F	U	M
Amnesic patients	.764	.692	.728	.562	.582	.572
Control subjects	.728	.653	.691	.527	.488	.508

F = Familiar voice; U = Unfamiliar voice; M = Mean.

no hint of impaired baseline performance in the amnesic patients. In fact, amnesic patients identified a numerically higher proportion of nonstudied words (.572) than did control subjects (.508). There was also little evidence of a voice familiarity effect for the nonstudied words. An ANOVA was performed on the proportion of correct identifications for nonstudied words, and it revealed nonsignificant main effects of Subject Group, $F(1,22) = 1.15$, $MSE = .044$ and Speaker's Voice, $F(1,22) < 1$, $MSE = .044$, as well as a nonsignificant interaction between the two variables, $F(1,22) < 1$, $MSE = .044$. Importantly, baseline performance of Korsakoff patients (.541) was only slightly lower than that of non-Korsakoff amnesics (.588) and was slightly higher than that of control subjects. These findings are consistent with our suggestion that the impaired baseline performance of Korsakoff patients in Experiment 1 was attributable to their difficulties discriminating the words spoken by the female speaker.

The data in Table 3 also reveal strong similarities between the magnitude and pattern of priming in amnesics and controls: Both exhibited relatively large priming effects, and both showed trends for more priming when words were spoken by the familiar voice than by the unfamiliar voice. An overall ANOVA showed a highly significant main effect of Item Type (studied *vs.* nonstudied), $F(1,22) = 18.91$, $MSE = .005$, $p < .001$, confirming that priming occurred. The Item Type × Subject Group interaction did not approach significance, $F(1,22) < 1$, $MSE = .037$, indicating that amnesic patients and control subjects exhibited identical patterns of priming. No other effects approached significance ($Fs < 1.14$).

To examine priming more specifically, we subtracted the proportion of nonstudied words identified correctly from the proportion of studied words identified correctly. Because there were no differences in the proportion of nonstudied words identified correctly in the familiar and unfamiliar voice conditions, we subtracted the mean proportion of nonstudied words identified correctly from the proportion of studied words identified in the familiar and unfamiliar voice conditions, respectively. The analysis indicated a nonsignificant effect of Subject Group, $F(1,22) < 1$, $MSE = .039$, and a marginally significant effect of Speaker's Voice, $F(1,22) = 3.46$, $MSE = .019$, $p = .076$. In addition, there was a nonsignificant Subject Group × Speaker's Voice interaction, $F(1,22) < 1$, $MSE = .019$. These results confirm that amnesic patients showed normal priming, and provide some evidence for voice-specific priming in both amnesics and controls.

The finding of intact priming in amnesic patients together with normal levels of baseline performance is consistent with our claim that amnesics' impaired baseline performance in Experiment 1 did not artifactually increase their priming scores, although it might have artifactually decreased them. Note also that in Experiment 2, control subjects' overall accuracy (.599) was closer to the 50% level than was amnesic patients' overall accuracy (.650), which could have resulted in a small inflation of priming scores in the control group (Chapman et al., 1994). In any case, our findings are contrary to Ostergaard and Jernigan's (1993) claim that normal priming in amnesic patients is an artifact of low baseline performance.

Recognition

Table 4 displays the proportion of hits and false alarms by amnesic patients and control subjects. As in Experiment 1, amnesics exhibited a substantial impairment in recognition accuracy. There was no systematic effect of voice familiarity on recognition performance. Both patients' and controls' hit rates were slightly higher for familiar than for unfamiliar voices, but their false alarm rates exhibited a trend in the opposite direction. Separate analyses of hit rates and false alarm rates revealed that neither the main effect of Speaker's Voice nor the Speaker's Voice × Subject Group interaction approached significance for either hits or false alarms, all $Fs < 1.03$.

To analyze recognition accuracy, we performed an ANOVA on corrected recognition scores. Because there were no significant differences between the proportions of false alarms to familiar and unfamiliar voices, we subtracted a single mean false alarm rate for each of the subject groups from the appropriate hit rates in the familiar- and unfamiliar-voice conditions. The analysis revealed a highly significant main effect of Subject Group, $F(1,22) = 52.64$, $MSE = .037$, $p < .001$, a nonsignificant effect of Speaker's Voice, $F(1,22) < 1$, $MSE = .037$, and a nonsignificant interaction between these two variables, $F(1,22) < 1$, $MSE = .037$.

General discussion

We began these experiments with two objectives: to determine whether Schacter et al.'s (1994) finding of spared auditory priming could be replicated and to assess whether amnesic patients show voice-specific priming under conditions that do not require binding between words and voices. With respect to the first objective, our data are relatively clear: Amnesic patients exhibited normal priming in both experiments. The results from Experiment 1 could not be interpreted unequivocally because of the low lev-

Table 4. Proportion of "Yes" responses to studied words (Hits) and nonstudied words (False Alarms) on the Yes/No Recognition Test as a function of speaker's voice in Experiment 2

	Studied words			Nonstudied words		
Subject group	F	U	M	F	U	M
Amnesic patients	.486	.472	.479	.257	.347	.302
Control subjects	.862	.821	.842	.249	.278	.264

F = Familiar voice; U = Unfamiliar voice; M = Mean.

els of baseline performance in Korsakoff amnesics. Our suggestion that the low level of baseline performance could be attributed to problems discriminating words spoken by the female speaker received strong support from Experiment 2, where we used only male speakers and found entirely normal levels of baseline performance in both Korsakoff and non-Korsakoff amnesics. Most importantly, amnesic patients exhibited normal levels and patterns of priming in this experiment. These results confirm Schacter et al.'s (1994) finding that amnesic patients can show spared auditory priming under conditions in which they also exhibit normal baseline performance.

With respect to our second objective of examining the role of binding processes in voice-specific priming, our experiments are best characterized as suggestive but not conclusive. Although the first experiment revealed only nonsignificant trends for voice familiarity effects in amnesics and controls, the second experiment revealed a stronger, marginally significant voice familiarity effect in both subject groups for studied words but not for nonstudied words. To the extent that this latter phenomenon is reliable, it raises a number of key issues. Most importantly, the finding is consistent with our hypothesis that amnesic patients failed to exhibit voice-specific priming in Schacter et al.'s (1995) experiment because the design of that experiment made it necessary to bind together words and voices during the study task in order to later exhibit voice-specific priming. In Schacter et al.'s (1995) experiment, simple familiarity with a speaker's voice was not sufficient to support voice-specific priming because all of the test voices were equally familiar; in our experiments, simple familiarity with the speaker's voice could support voice-specific priming because one test voice was familiar and the other was not. Thus, our data are consistent with the idea that voice familiarity effects are mediated by an acoustic PRS subsystem that is preserved in amnesic patients, whereas voice-specific priming effects that require episodic binding between words and voices depend on medial temporal/diencephalic structures that are damaged in amnesia. To provide a stronger test of our hypothesis, it will be necessary to develop a paradigm that yields stronger evidence of voice familiarity effects.

Alternative possibilities also need to be considered. For example, in Schacter et al.'s (1995) experiment, subjects heard words from six different speakers during the study task, whereas in our study there was only a single speaker. Thus, the amnesic patients' failure to exhibit voice-specific priming in the former experiment might be attributable to the multiple speakers rather than to the requirement for binding between words and voices. Future research should attempt to distinguish between these two possibilities.

Our observation that the voice familiarity effect in Experiment 2 appeared to occur only for studied words raises further questions about the nature of the processes underlying this effect. It is possible that the effect is mediated solely by acquisition of information about the speaker's voice—information that affects processing of the phonetic content of a speaker's utterance. Indeed, Nygaard et al. (1994) have provided evidence that familiarity with a speaker's voice influences phonetic analysis of what the speaker says. However, if the voice familiarity effect is mediated exclusively by altered processing of the phonetic content of whatever the familiar speaker says, then it should be observed for both studied and nonstudied words. To the extent that the effect is observed only for studied words, there is reason to believe that something more than altered phonetic analysis is involved.

Because our data are somewhat equivocal, they must be interpreted cautiously. Nevertheless, one potentially useful perspective is provided by the idea that information from different kinds of cues can be combined in either an additive or interactive manner (e.g., Ratcliff & McKoon, 1988). Curran and Schacter (in press) have considered the hypothesis that amnesic patients may be able to combine activation from different sources additively but not interactively (see Humphreys et al., 1989). Curran and Schacter point out that when a different-voice condition is created by repairing studied words and voices, as in the Schacter et al. (1995) experiment in which amnesics exhibited impaired voice-specific priming, the amount of additive activation in same- and different-voice conditions is identical: both the words and the voices appeared previously on the study list. To exhibit more priming in the same- than in the different-voice condition in such an experiment, it is necessary to interactively combine word and voice information. If control subjects are capable of such interactive activation but amnesic patients are not, then the former but not latter would exhibit voice-specific priming, as Schacter et al. (1995) observed. In contrast, in the present experiments, there was greater additive activation in the familiar-voice condition than in the unfamiliar-voice condition. Accordingly, reliance on additive activation between words and voices could produce a voice familiarity effect without any need for interactive activation. Thus, even if amnesic patients rely solely on additive activation, this analysis suggests that they could show normal voice familiarity effects, and we have provided suggestive evidence that they can. Further exploration of the processes underlying implicit memory for words and voices will likely provide important insights in the nature of preserved and impaired memory processes in amnesia.

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