

Impaired Implicit Memory for Gist Information in Amnesia

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In 2 experiments using a converging associates paradigm, the authors evaluated implicit memory for gist information in amnesic patients. In Experiment 1, participants saw multiple sets of associates, each converging on a nonpresented theme word, and were then tested using an implicit word stem completion test and an explicit cued recall test. Amnesic patients showed intact implicit and impaired explicit memory for studied words, but memory for nonpresented lures was impaired, regardless of retrieval instructions. To evaluate whether impaired implicit memory for lures was due to accelerated forgetting of gist information, short study lists were used in Experiment 2, each consisting of a single set of associates. Amnesics' implicit memory for lures was again impaired. These results point to an inability to encode robust gist representations as the cause of impaired gist memory in amnesia.

Keywords: amnesia, false memory, gist memory, implicit memory

During the last decade, studies of amnesia focusing on the amount of information amnesic individuals can remember have been complemented by studies evaluating qualitative aspects of patients' performance, such as the nature of the errors and distortions that sometimes accompany their remembering. One fruitful line of research in this regard has been the study of false recognition in amnesia—the mistaken claim of remembering items similar to those that were studied. These studies have demonstrated that under conditions in which normal participants show high levels of false recognition, amnesic patients show markedly reduced levels of false recognition (for a review, see Schacter, Verfaellie, & Koutstaal, 2002). The finding that amnesic patients show parallel impairments in veridical and false recognition suggests that the medial temporal and diencephalic regions that mediate veridical memory are also important for the storage and retrieval of the semantic and/or perceptual information that underlies false memory in healthy participants.

Most studies of false memory in amnesia have used a converging associates paradigm, originally developed by Deese (1959) and later revived and modified by Roediger and McDermott (1995) (the Deese–Roediger–McDermott [DRM] paradigm).¹ In our initial study using the DRM paradigm (Schacter, Verfaellie, & Pradere, 1996), amnesic patients and controls studied lists of associated words that all converged on a nonpresented theme word and were then tested with studied words, the unstudied theme word of

each list and other unstudied words that were unrelated to the studied words. As expected, amnesic patients endorsed fewer studied items than controls and more unstudied items. More important, they also endorsed fewer nonpresented theme words than did the controls. These findings, and similar findings in subsequent studies using perceptually related materials (Koutstaal, Schacter, Verfaellie, Brenner, & Jackson, 1999; Koutstaal, Verfaellie, & Schacter, 2001; Schacter, Verfaellie, & Anes, 1997), have been taken as evidence for impairments in the encoding, maintenance, and/or retrieval of gist information (cf. Reyna & Brainerd, 1995) in amnesia. When presented with many converging associates, normal participants establish a well-organized representation of the features that are common among items on the study list—the perceptual or conceptual gist of the study list. Because the nonpresented theme word is consistent with the gist of the study list, normal participants experience a strong sense of familiarity or recollection. Amnesic patients, in contrast, have poorer memory for the gist information and thus show reduced levels of false recognition.

In a more recent study, we evaluated whether the gist impairment in amnesia is due either to an inability to form robust gist representations at encoding or to impaired access to gist representations at retrieval. Our evaluation was done by comparing the performance of amnesic patients under standard recognition instructions with that under meaning retrieval instructions (Verfaellie, Schacter, & Cook, 2002). The meaning retrieval instructions were similar to those used by Brainerd and Reyna (1998) in which participants were asked to ignore whether an item was actually studied but instead to endorse any item that shared the meaning of studied items. We reasoned that if amnesics' deficit is due to an inability to encode strong gist information, then meaning retrieval

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¹ Findings in the converging associates paradigm differ from those in studies in which participants study only a single word that is semantically, associatively or physically similar to the lure (Cermak, Butters, & Gerrein, 1973; Verfaellie, Rapsack, Keane, & Alexander, 2004). In the latter studies, amnesic patients showed higher levels of false recognition than did controls (see Schacter et al., 2002; Verfaellie et al., 2004).

instructions should not ameliorate the reduction in false recognition seen under standard retrieval instructions. However, if amnesic individuals are able to encode gist information but are unable to access that information under standard retrieval instructions—because standard instructions emphasize access to distinctive, item-specific information rather than to the gist information that is shared among items—then meaning instructions should lessen or possibly eliminate the false recognition impairment in amnesia. We found that false memory in the amnesic group was as impaired in the meaning retrieval condition as in the standard retrieval condition: Meaning instructions led to enhanced false recognition, but did so to the same extent in the control group and the amnesic group. Thus, it was concluded that poor gist memory in amnesia is due to an impairment in the formation and maintenance of gist representations.

One argument that might be offered against this conclusion is that although the meaning retrieval instructions de-emphasized retrieval of item-specific information, the task, nonetheless, required intentional retrieval of gist information. It is possible that amnesics' impairment in false memory, just as their impairment in veridical memory, is due to the use of an explicit memory test that requires participants to refer back intentionally to the study phase. By this reasoning, amnesic participants may have a deficit in intentional retrieval of gist information but not in unintentional (or implicit) retrieval. Preserved unintentional retrieval, in turn, would imply that amnesic participants are able to establish some kind of gist representation at encoding, and that establishment of such a representation is not dependent on the medial temporal and diencephalic regions damaged in amnesia. To evaluate this possibility, we examined implicit memory for gist information in amnesic patients in the present study.

Several studies in normal cognition have provided evidence that participants can show priming for a nonpresented theme word in implicit memory tasks such as fragment completion (McDermott, 1997), stem completion (McDermott, 1997; McKone & Murphy, 2000; Smith, Gerkens, Pierce, & Choi, 2002), and anagram solution (Lovden & Johansson, 2003). More important, such priming is obtained even when precautions are taken to reduce the chance of contamination by explicit memory (Lovden & Johansson, 2003; McKone & Murphy, 2000; Smith et al., 2002). In the present study, we evaluated implicit memory for studied items and non-presented theme words in the context of a stem completion task.² Stem completion priming for studied items has repeatedly been demonstrated to be intact in amnesia (e.g., Carlesimo, Marfia, Loasses, & Caltagirone, 1996; Graf, Shimamura, & Squire, 1985; Graf, Squire, & Mandler, 1984) and thus provides an appropriate reference condition against which to evaluate implicit memory for nonpresented theme words. We hypothesized that if the impairment in gist memory is due to the intentional retrieval demands associated with the tasks that have been used to evaluate false memory in amnesia, then amnesic participants should show priming for nonpresented theme words, just like nonamnesic controls. Alternatively, if the impairment in gist memory is due to an inability to form robust gist representations, then amnesics' gist memory should continue to be impaired even when tested implicitly. That is, amnesic participants should show impaired priming for nonpresented theme words, even though priming for studied words is intact.

In conjunction with the implicit stem completion task, we administered an explicit stem cued recall task. The inclusion of an explicit task that is similar in format to the implicit stem completion task allowed us to evaluate directly the effect of retrieval instructions on false memory in both the amnesic and the control group. Additionally, the use of a cued recall task may shed light on an apparent inconsistency with regard to amnesics' explicit retrieval of gist information: Although impaired gist memory in amnesia has been consistently demonstrated in studies of false recognition, two studies of false recall revealed that amnesic patients intruded the theme word as often as (Schacter, Verfaellie, & Pradere, 1996) or more often than (Melo, Winocur, & Moscovitch, 1999) did controls. Findings in a free-recall task are difficult to interpret, however, because it is not clear how to take into account intrusions of unrelated words, which in at least one study (Schacter, Verfaellie, & Pradere, 1996) also differed across groups. The use of a cued recall task allowed us to evaluate gist memory in a task that requires overt generation of responses yet provides a straightforward way to adjust for baseline differences in response rate, as both studied and unstudied cues are provided on the cued recall test.

The status of implicit and explicit memory for gist information in amnesia was evaluated in two experiments that differed in terms of study list length. In Experiment 1, the study list consisted of multiple sets of associates, each converging on a theme word. In an effort to reduce the average delay between an item's study and test presentation, we used short study lists in Experiment 2, each consisting of a single set of associates to a theme word, and testing occurred immediately after presentation of each list.

Experiment 1

Method

Participants. Fourteen amnesic individuals (10 men and 4 women) and 18 individuals with intact memory abilities (8 men and 10 women) participated in the experiment. Of the 14 amnesic participants, 6 had a diagnosis of Korsakoff syndrome, and the remaining 8 patients had a variety of nonalcoholic etiologies, including anoxia ($n = 5$), encephalitis ($n = 2$), and bithalamic stroke ($n = 1$). Demographic and clinical neuropsychological data for the individual patients are provided in Table 1. The combined group of amnesics had a mean age of 62.0 years, with a mean education of 14.0 years. The mean verbal IQ of the patient group, as measured by the Wechsler Adult Intelligence Scale—Third Edition (WAIS-III; Wechsler, 1997a), was 102. Their attentional abilities, measured by the Wechsler Memory Scale—Third Edition (WMS-III; Wechsler, 1997b) Working Memory Index, were also intact, as indicated by a mean score of 104. Their episodic memory functioning was severely

² A reviewer questioned our selection of the stem completion task to assess priming for gist information, as stem completion priming depends on both perceptual and conceptual processes. Arguably, a more purely conceptual priming task, such as word association, would be more appropriate for this purpose. Our selection was guided by the fact that several studies have demonstrated priming of critical lures in stem completion, whereas only a single study has demonstrated priming of critical lures in word association. Moreover, in the stem completion study on which our experiment was modeled (McKone & Murphy, 2000), priming was more robust than in word association (McDermott, 1997).

Table 1
Summary of Neuropsychological Characteristics of Amnesic (AM) Patients in Experiment 1

Patient	Etiology	WAIS-III			WMS-III			
		Age	Edu.	VIQ	GM	AD	VD	WM
AM01	Anoxia	43	14	90	45	52	53	93
AM02	Anoxia	39	16	86	49	52	53	93
AM03	Anoxia	46	14	111	59	52	72	96
AM04	Anoxia	72	18	113	75	80	72	102
AM05	Anoxia	52	12	83	52	55	56	91
AM06	Bithalamic stroke	60	12	84	73	67	84	99
AM07	Encephalitis	59	12	106	69	77	68	111
AM08	Encephalitis	74	18	135	45	58	53	141
AM09	Korsakoff	81	14	105	66	64	62	121
AM10	Korsakoff	75	14	99	59	58	65	115
AM11	Korsakoff	52	18	111	69	64	72	81
AM12	Korsakoff	78	14	103	72	71	68	115
AM13	Korsakoff	56	12	97	66	74	62	108
AM14	Korsakoff	83	9	100	72	74	75	91
Controls ($n = 18$)		60.7	14.4	106				

Note. WAIS-III = Wechsler Adult Intelligence Scale—Third Edition; WMS-III = Wechsler Memory Scale—Third Edition; Edu. = Education; VIQ = Verbal IQ; GM = General Memory; AD = Auditory Delayed; VD = Visual Delayed; WM = Working Memory.

compromised, as indicated by a mean General Memory Index of 62, a mean Visual Delayed Index of 65, and a mean Auditory Delayed Index of 64.

The control group consisted of 9 individuals with a history of alcoholism and 9 individuals with no known history of alcoholism. The control group was matched to the amnesic group in terms of age ($M = 60.7$ years), education ($M = 14.4$ years), and WAIS-III Verbal IQ ($M = 106$; $ts < 1.2$).

Materials and design. Stimuli consisted of 16 sets of 16 words used by McKone and Murphy (2000), with minor modifications to accommodate cultural and language differences. Each set contained 15 words to be presented for study and a critical lure that was not presented for study. The study words were all highly associated to the critical lure. All lures were at least five letters long, had distinct three-letter stems, and their stems had at least 8–10 completions. For each set, one associate that met the same stem completion requirements as the lures was selected as the studied target. The target could occupy all but the first two or last two positions on the study list. Studied targets had unique stems that were distinct from the stems for the critical lures and were matched to the lures in terms of average word frequency and average baseline stem completion rate. The 16 sets were divided into two groupings of eight, and each group was used to create a 120-word list. Across the two lists, targets and critical lures were matched as closely as possible in terms of word frequency and baseline completion rate. Words on each list were ordered such that for each set of associates, the strongest associates to the lure occurred first. For each participant, one of the lists was used as the study list and the other as the unstudied list. The assignment of list to condition was counterbalanced across participants.

The test list for the implicit stem completion task and the explicit cued recall task consisted of the same 32 three-letter word stems. Of these stems, 8 could be completed to a word that had been presented in one of the studied sets (targets), and 8 could be completed to the related nonpresented theme word from each studied set (lures). The remaining 16 stems corresponded to words pertaining to unstudied sets and consisted of 8 stems that could be completed to a word from an unstudied set (target distractors) and 8 stems that could be completed to a theme word corresponding to an unstudied set (lure distractors). The order of the test words was randomly determined, except for the fact that for half of the sets, the stem corresponding to the list word was presented before the stem corresponding to the theme word, and for the other half of the sets, the order was reversed. Stems were presented in a different order in the implicit and explicit test list.

Procedure. Participants took part in three phases: a study phase, an implicit stem completion phase, and an explicit cued recall phase. During the study phase, participants were shown groups of converging associates, one at the time. Words were presented on the computer screen at a rate of 2 s per word, and each group of associates was separated by an asterisk that was shown for 5 s. Participants were asked to read each word aloud and to indicate verbally how many meanings they believed each word to have. In the stem completion phase, which started approximately 3 min after the study phase had ended, participants were asked to complete stems with the first word that came to mind as quickly as they could. This phase was introduced as a filler task unrelated to the memory experiment in which individuals were participating. After participants completed this implicit memory task, explicit memory was tested immediately by means of a cued recall test. Participants were told that some stems could be completed with a word from the previously studied list and were instructed to complete stems only with words that they remembered from the study list. They were told that many stems would not correspond to studied words and were instructed to leave these stems blank. At the end of the experiment, participants were given a questionnaire to assess whether they used explicit memory during the implicit test phase. The questionnaire was modeled after that developed by McKone and Murphy (2000). Data from participants who indicated using explicit memory to complete stems during the stem completion task were eliminated from the results.

Results and Discussion

Results of 2 control participants (1 nonalcoholic, 1 alcoholic) were eliminated because they indicated using explicit memory during the implicit task. Completion rates in the implicit and explicit test for the remaining participants are shown in Table 2. This table combines the results of individuals with and without a history of alcohol use because preliminary analyses revealed no differences among control participants or amnesic participants as a function of alcohol history.

Implicit memory. As can be seen in Table 2, both amnesic participants and controls showed higher completion rates for studied than for unstudied targets. Control participants also showed higher completion rates for lures corresponding to studied associates than to unstudied associates, but amnesic participants did not.

Table 2
Proportion of Stems Completed to List Targets and Critical Lures by Amnesic Patients and Control Participants in the Implicit and Explicit Retrieval Test in Experiment 1

Variable	Implicit test			Explicit test		
	Studied	Unstudied	Studied – Unstudied	Studied	Unstudied	Studied – Unstudied
List target						
Amnesics	0.48 (0.03)	0.34 (0.04)	0.14 (0.06)	0.31 (0.05)	0.18 (0.04)	0.13 (0.07)
Controls	0.46 (0.05)	0.26 (0.03)	0.20 (0.01)	0.46 (0.05)	0.11 (0.03)	0.35 (0.05)
Lure						
Amnesics	0.24 (0.05)	0.28 (0.05)	-0.04 (0.06)	0.17 (0.05)	0.17 (0.04)	0 (0.06)
Controls	0.30 (0.04)	0.18 (0.03)	0.13 (0.05)	0.32 (0.05)	0.05 (0.02)	0.27 (0.06)

Note. In the implicit test, Studied – Unstudied reflects priming; in the explicit test, Studied – Unstudied reflects corrected recall. Standard error of the mean appears in parentheses.

An analysis of variance (ANOVA) on completion rates to list targets revealed a significant main effect of study condition, $F(1, 28) = 13.65, p < .01, \eta_p^2 = .33$, confirming that there was significant priming for targets. Neither the effect of group nor the Group \times Study interaction was significant ($F_s < 1.76$). Priming for targets was significant both in the control group, $t(15) = 2.96, p < .01$, and in the amnesic group, $t(13) = 2.28, p < .05$. An ANOVA on completion rates to lures revealed a significant Group \times Study Condition interaction, $F(1, 28) = 4.34, p < .05, \eta_p^2 = .13$. For control participants, the completion rate for lures corresponding to studied associates was higher than for lures corresponding to unstudied associates, $t(15) = 2.34, p < .05$, demonstrating significant priming for critical lures. For amnesic participants, in contrast, there was no priming for critical lures, as the completion rate for lures did not differ, depending on whether the corresponding list of associates had been studied, $t(13) < 1$. A direct comparison of the magnitude of lure priming across groups indicated significantly greater priming in the control group than in the amnesic group, $t(28) = 2.08, p < .05$.

Unexpectedly, baseline completion rates for lures corresponding to unstudied associates were higher in the amnesic group than in the control group, making the priming results for lures more difficult to interpret. To evaluate priming for lures in the absence of baseline differences, we examined group means after excluding the 3 amnesic patients whose baseline scores fell outside the range of those for controls. For this subgroup of patients, the completion rate for lures associated with unstudied lists was .22 and did not differ significantly from that for the controls ($M = .18$), $t(25) < 1$. The completion rate for lures associated with studied lists was .18 in this subgroup of amnesic patients, again indicating an absence of lure priming. The Group \times Study Condition interaction for this restricted analysis was marginally significant, $F(1, 25) = 3.56, p = .07, \eta_p^2 = .13$.

Explicit memory. As can be seen in Table 2, both amnesic patients and controls produced more target completions on the cued recall test for studied than for unstudied targets, but this difference was much greater in control participants than in the amnesic participants. Control participants also produced more lures corresponding to studied associates than to unstudied associates, but amnesic patients did not. An ANOVA on target production rates revealed a significant effect of study condition, $F(1, 28) = 29.22, p < .01, \eta_p^2 = .51$, as well as a significant Group \times Study Condition interaction, $F(1, 28) = 6.44, p < .05, \eta_p^2 = .19$.

This interaction reflected the fact that production rates for studied targets were higher for the control participants than for the amnesic participants, $t(28) = 1.98, p < .06$, whereas production rates for unstudied targets did not differ in the two groups, $t(28) = 1.5$. A direct comparison of corrected cued recall of targets across groups revealed higher cued recall in the control group than in the amnesic group, $t(28) = 2.54, p < .05$. An ANOVA on lure production rates also revealed a significant effect of study condition, $F(1, 28) = 9.99, p < .01, \eta_p^2 = .26$, and a significant Group \times Study Condition interaction, $F(1, 28) = 10.17, p < .01, \eta_p^2 = .27$. The production rate for lures corresponding to studied associates was higher for the control participants than for the amnesic participants, $t(28) = 2.07, p < .05$, and the production rate for lures corresponding to unstudied associates was higher for the amnesic participants than for the control participants, $t(28) = 2.67, p < .05$. A direct comparison of corrected cued recall of lures across groups indicated higher cued recall of lures in the control group than in the amnesic group, $t(28) = 3.17, p < .01$.

Effect of retrieval instructions. To examine directly the effect of retrieval instructions on the status of performance in amnesia, we compared baseline-corrected completion rates in the two groups as a function of retrieval instructions (implicit vs. explicit). Results of the ANOVA for list targets revealed a marginal effect of group, $F(1, 28) = 3.33, p < .08, \eta_p^2 = .11$, and a marginal Instruction \times Group interaction, $F(1, 28) = 3.44, p < .08, \eta_p^2 = .11$. Priming for targets did not differ across groups ($F = 1$), but corrected cued recall was higher in control participants than in amnesic participants, $F(1, 28) = 13.20, p < .01$. A similar ANOVA for lures revealed a main effect of group, $F(1, 28) = 10.04, p < .01, \eta_p^2 = .26$, and a marginal effect of retrieval instructions, $F(1, 28) = 3.80, p < .07, \eta_p^2 = .12$. The Group \times Test interaction was not significant, $F(1, 28) = 1.38$. Amnesic participants completed fewer lures than did controls regardless of the nature of the retrieval instructions.

Experiment 1 revealed two main findings. First, amnesic patients showed intact priming for studied words but failed to show priming for nonpresented theme words, suggesting that their implicit memory for gist information is impaired. Second, retrieval instructions affected the status of veridical memory and gist memory in amnesia in different ways. Whereas memory for studied items was impaired when tested explicitly but not when tested implicitly, memory for nonpresented theme words was impaired regardless of retrieval instructions.

The impairment in implicit memory for gist information in amnesia is unlikely to be due to the use of explicit memory in the control group, as participants who indicated in the questionnaire to have used explicit memory were eliminated from the study. Moreover, implicit memory for studied items was intact in amnesia. One would expect explicit contamination to have had a similar effect on the performance of studied items and nonpresented lures, but clearly, performance for the two types of items was dissociated.

Another possible concern in interpreting amnesics' impairment in implicit memory for gist information relates to the higher baseline completion rate for lures in the amnesic group. Higher stem completion baseline performance has been observed in several amnesic studies (Carlesimo, 1994; Squire, Shimamura, & Graf, 1987) but remains poorly understood. Nonetheless, it is unlikely to explain amnesics' failure to show lure priming, as the results did not change when only a subgroup of amnesic patients was considered with baseline scores similar to those of controls. Furthermore, the high baseline completion rate for lures is unlikely to provide a functional ceiling, as amnesics' baseline completion rate for targets was even higher, yet priming for targets was normal. Our findings, therefore, point unequivocally to an impairment in implicit memory for gist information under conditions in which implicit memory for veridical information is intact.

Our findings further suggest that amnesics' impairment in gist information is not a function of the retrieval demands of the memory task. The fact that amnesic patients showed impaired retrieval of gist information in a cued recall task suggests that their explicit retrieval deficit is not limited to recognition tasks but can be seen in tasks that require overt generation of information from memory as well. More important, the fact that memory for gist information was impaired under implicit as well as explicit retrieval instructions suggests that the impairment in gist memory in amnesia is not due to an inability to intentionally retrieve gist information. Rather, these findings favor the view that amnesic patients are unable to form a strong gist representation at encoding.

Before accepting an encoding interpretation, however, an alternative possibility needs to be ruled out. Because multiple sets of associates were presented in one extensive study list, the study-test delay was considerable. It is possible, therefore, that amnesics' gist impairment is due to accelerated forgetting of gist information. By this view, amnesic participants may form robust gist representations just as control participants, but they may be unable to maintain such representations over time. We evaluated this possibility in Experiment 2, in which short study lists were presented, each consisting of a set of associates corresponding to a single nonpresented theme word. Implicit memory for the studied items and nonpresented theme words was tested immediately following presentation of each study list. If amnesics' impairment in implicit memory for gist is due to accelerated forgetting of gist information, then priming for lures should be intact under these conditions.

Experiment 2

Method

Participants. Seventeen amnesic patients (11 men and 6 women) and 20 patients with intact memory abilities (12 men and 8 women) participated in the experiment.³ Of the 17 amnesic participants, 6 had a diagnosis of Korsakoff syndrome, and the remaining 11 patients had a variety of nonalcoholic etiologies, including anoxia ($n = 8$), encephalitis

($n = 2$), and bithalamic stroke ($n = 1$). Demographic and clinical neuropsychological data for the individual patients are presented in Table 3. The combined group of amnesics had a mean age of 59 years, with a mean education of 14.5 years. The mean verbal IQ of the patient group, as measured by the WAIS-III was 100. Their attentional abilities, measured by the WMS-III Working Memory Index, were also intact, as indicated by a mean score of 97. Their episodic memory functioning was severely compromised, as indicated by a mean General Memory Index of 58, a mean Visual Delayed Index of 63, and a mean Auditory Delayed Index of 61.

The control group consisted of 9 individuals with a history of alcoholism and 11 individuals with no known history of alcoholism. The control group was matched to the amnesic group in terms of age ($M = 61.8$ years), education ($M = 14.2$ years), and WAIS-III Verbal IQ ($M = 107$; $t_s < 1.6$).

Materials and design. The stimuli were identical to those used in Experiment 1 and were again divided into two sets. Each set of stimuli consisted of eight 15-word lists. One set of lists was used as studied lists during the implicit task and as unstudied lists during the explicit task, whereas the other set of lists was used as studied lists during the explicit task and as unstudied lists during the implicit task. Assignment of set of lists to studied and unstudied condition for each task was counterbalanced across participants.

Corresponding to each set of stimuli, there were eight test lists consisting of 14 three-letter word stems. Each test list comprised four critical stems and 10 filler stems. One of the critical stems could be completed to the studied target, one to the lure corresponding to the studied list, one to a word from an unstudied list (target distractor), and one to the lure of the corresponding unstudied list (lure distractor). The filler stems were included to reduce the possibility that participants might detect a relationship between each study and test list, and these stems did not overlap with stems of any studied items. The order of the critical stems in the test list was pseudorandomly determined so that for half of the lists, the stem corresponding to the list target was presented before the stem corresponding to the lure, whereas for the other half of the lists, the order was reversed.

Procedure. Participants took part in an implicit and an explicit task that were administered at least 1 week apart. The implicit session was always administered first. Each session consisted of eight brief study-test runs. During the study phase of each run, participants were shown a group of 15 converging associates, one at a time. Words were presented on the computer screen at a rate of 2 s per word. Participants were asked to read each word aloud and to indicate verbally how many meanings they believed each word to have. Each study phase was followed immediately by a test phase. During implicit tests, participants were asked to complete each stem with the first word that came to mind. To reduce the possibility of explicit contamination in the implicit test, study and test phases were portrayed as two independent experiments that would alternate in order to maintain participants' interest. During explicit tests, participants were told that some stems could be completed with a word from the previously studied list and were instructed to complete stems only with words that they remembered from the immediately preceding study list. They were told that many stems would not correspond to studied words and were instructed to leave these stems blank. At the end of the implicit session, participants completed the same questionnaire used in Experiment 1 to assess whether they used explicit memory. Data from participants who indicated using explicit memory to complete stems during the stem completion task were eliminated from the results.

Results and Discussion

Implicit data from 3 participants (1 nonalcoholic control, 1 alcoholic control, 1 Korsakoff patient) were eliminated because

³ One Korsakoff patient and 1 alcoholic control participant were no longer available at the time of the explicit memory test. Only their implicit test data are included.

Table 3
Summary of Neuropsychological Characteristics of Amnesic (AM) Patients in Experiment 2

Patient	Etiology	WAIS-III			WMS-III			
		Age	Edu.	VIQ	GM	AD	VD	WM
AM01	Anoxia	66	20	111	52	64	56	83
AM02	Anoxia	75	12	107	59	64	65	83
AM03	Anoxia	20	10	91	45	46	56	79
AM04	Anoxia	45	14	90	45	52	53	93
AM05	Anoxia	40	16	86	49	52	53	93
AM06	Anoxia	47	14	111	59	52	72	96
AM07	Anoxia	74	18	113	75	80	72	102
AM08	Anoxia	54	12	83	52	55	56	91
AM09	Bithalamic stroke	62	12	84	73	67	84	99
AM10	Encephalitis	60	12	106	69	77	68	111
AM11	Encephalitis	76	18	135	45	58	53	141
AM12	Korsakoff	83	14	105	66	64	62	121
AM13	Korsakoff	77	14	99	59	58	65	115
AM14	Korsakoff	50	14	80	57	58	72	69
AM15	Korsakoff	58	12	97	66	74	62	108
AM16	Korsakoff	62	16	92	47	58	56	85
AM17 ^a	Korsakoff	54	18	111	69	64	72	81
Controls (n = 20)		61.8	14.2	106.9				

Note. WAIS-III = Wechsler Adult Intelligence Scale—Third Edition; WMS-III = Wechsler Memory Scale—Third Edition; Edu. = Education; VIQ = Verbal IQ; GM = General Memory; AD = Auditory Delayed; VD = Visual Delayed; WM = Working Memory.

^a AM17 was excluded from data analysis due to explicit memory contamination.

these individuals indicated using explicit memory during the implicit memory task. Completion rates in the implicit and explicit task for the remaining participants are shown in Table 4. As in Experiment 1, data from participants with and without a history of alcohol use were combined because preliminary analyses revealed no differences among control participants or amnesic patients as a function of alcohol history.

Implicit memory. As illustrated in Table 4, both amnesic patients and controls showed higher completion rates for studied than for unstudied targets. Control participants also showed higher completion rates for lures corresponding to studied associates than to unstudied associates, but amnesic participants did not. An ANOVA on completion rates to list targets revealed a significant effect of study condition, $F(1, 32) = 87.13, p < .01, \eta_p^2 = .73$, indicating that there was significant priming for targets. Neither the effect of group nor the Group \times Study Condition interaction was significant ($F_s < 1$). Priming for targets was significant in both the

control group, $t(17) = 8.33, p < .01$, and the amnesic group, $t(15) = 5.44, p < .01$. An ANOVA on completion rates to lures revealed a marginally significant effect of study condition, $F(1, 32) = 3.66, p = .06, \eta_p^2 = .10$, indicating that lures corresponding to studied lists were given as completions more often than lures corresponding to nonstudied lists. Although the Group \times Study Condition interaction failed to reach significance, $F(1, 32) = 2.73, p = .11, \eta_p^2 = .08$, it can be seen in Table 4 that lure priming was driven almost entirely by the performance of control participants. For control participants, the completion rate was higher for lures corresponding to studied than to unstudied associates, $t(17) = 2.69, p < .05$, but this was not the case for amnesic patients, $t(15) < 1$.

As in Experiment 1, baseline completion rates for lures were higher for amnesic patients than for controls. This was partially due to a single amnesic patient whose baseline score was outside the range of controls and partially to the high baseline completion

Table 4
Proportion of Stems Completed to List Targets and Critical Lures by Amnesic Patients and Control Participants in the Implicit and Explicit Retrieval Test in Experiment 2

Variable	Implicit test			Explicit test		
	Studied	Unstudied	Studied – Unstudied	Studied	Unstudied	Studied – Unstudied
List target						
Amnesics	0.61 (0.04)	0.29 (0.03)	0.32 (0.06)	0.50 (0.07)	0.14 (0.09)	0.36 (0.08)
Controls	0.64 (0.04)	0.31 (0.03)	0.33 (0.04)	0.80 (0.05)	0.02 (0.01)	0.78 (0.05)
Lure						
Amnesics	0.27 (0.04)	0.26 (0.04)	0.01 (0.05)	0.36 (0.06)	0.08 (0.03)	0.28 (0.06)
Controls	0.27 (0.04)	0.15 (0.04)	0.12 (0.04)	0.45 (0.07)	0.01 (0.01)	0.44 (0.07)

Note. In the implicit test, Studied – Unstudied reflects priming; in the explicit test, Studied – Unstudied reflects corrected recall. Standard error of the mean appears in parentheses.

rate to two stimuli in a majority of patients. When the data were rescored with these sources of outliers removed, the baseline rate in amnesic patients was .19, which was not significantly different from the baseline rate for controls ($M = .14$), $t(31) < 1$. Amnesics' completion rate for lures corresponding to studied associates was .22, whereas the corresponding value in control participants was .25. Again, patients failed to show priming for critical lures, $t(14) < 1$, whereas control participants showed robust priming, $t(17) = 2.2$, $p < .05$. The Group \times Condition interaction, however, was not significant in this restricted analysis, $F(1, 31) = 1.32$.

Explicit memory. As illustrated in Table 4, target production rates on the cued recall test were higher for studied than for unstudied targets both in amnesic patients and in controls, but corrected cued recall, indicated by the difference between these conditions, was much higher for the control group than for the amnesic group. A similar pattern was seen for lure productions: Again, both groups produced more lures corresponding to studied associates than to unstudied associates, but corrected cued recall was higher in the control group than in the amnesic group. An ANOVA on target production rates revealed a significant effect of study condition, $F(1, 30) = 150.61$, $p < .01$, $\eta_p^2 = .83$; a marginal effect of group, $F(1, 20) = 3.95$, $p < .06$, $\eta_p^2 = .12$; and a significant Group \times Study Condition interaction, $F(1, 30) = 20.75$, $p < .01$, $\eta_p^2 = .41$. The amnesic group produced fewer studied targets, $t(30) = 3.62$, $p < .01$, and more unstudied targets, $t(30) = 3.14$, $p < .01$, than did the control group, resulting in significantly impaired corrected target recall, $t(30) = 4.56$, $p < .01$. An ANOVA on lure production rates revealed a significant effect of study condition, $F(1, 30) = 61.48$, $p < .01$, $\eta_p^2 = .67$, and a marginal Group \times Study Condition interaction, $F(1, 30) = 2.99$, $p < .10$, $\eta_p^2 = .09$. The two groups did not differ in their production of lures corresponding to studied targets, but the amnesic group produced more lures corresponding to unstudied targets than did the control group, $t(30) = 2.60$, $p < .05$, leading to marginally impaired corrected lure recall, $t(30) = 1.70$, $p < .10$.

Effect of retrieval instructions. To examine directly the effect of retrieval instructions on the status of performance in amnesia, we compared baseline-corrected completion rates in the two groups as a function of retrieval instructions (implicit vs. explicit). Results of the ANOVA for targets revealed significant effects of group, $F(1, 30) = 15.60$, $p < .01$, $\eta_p^2 = .34$; retrieval instructions, $F(1, 30) = 18.18$, $p < .01$, $\eta_p^2 = .38$; and a significant Group \times Instruction interaction, $F(1, 30) = 10.87$, $p < .01$, $\eta_p^2 = .27$. Priming for targets did not differ across groups ($F < 1$), but corrected cued recall was higher for control participants than for amnesic participants, $F(1, 30) = 25.30$, $p < .01$. A similar ANOVA for lures revealed significant effects of group, $F(1, 30) = 6.35$, $p < .05$, $\eta_p^2 = .17$, and retrieval instructions, $F(1, 30) = 22.40$, $p < .01$, $\eta_p^2 = .43$. The Group \times Instruction interaction was not significant ($F < 1$). The amnesic group completed fewer lures than did controls, regardless of retrieval instructions.

Despite the use of short study lists that contained associates converging on a single nonpresented theme word, the pattern of results obtained in this experiment was essentially identical to that obtained in Experiment 1. Amnesic patients again failed to show priming for nonpresented theme words but showed intact priming for studied words. Furthermore, memory for studied items was impaired in amnesia when tested explicitly but not when tested

implicitly, whereas memory for lures was impaired in amnesia regardless of retrieval instructions. Thus, the impairment in implicit memory for gist information demonstrated by amnesic patients in Experiment 1 cannot be ascribed to rapid forgetting of gist information in association with the use of long study lists. Rather, we propose that amnesics' impairment can best be understood as an inability to encode a strong gist representation that can support implicit or explicit memory.

General Discussion

Our findings extend the scope of the gist impairment in amnesia by demonstrating that gist memory is impaired not only when tested explicitly but also when tested implicitly. The pervasiveness of the gist impairment in amnesia is striking given that the impairment in veridical memory is limited to tasks requiring explicit retrieval. It suggests that the impairment in gist memory cannot be explained merely as a byproduct of impaired veridical memory.

The use of an implicit memory task eliminated the need for strategic processes associated with intentional retrieval yet did not lessen or eliminate the impairment in gist memory in the amnesic group. Moreover, the impairment was evident not only when memory was tested following presentation of multiple lists of converging associates (see Experiment 1) but also when it was tested immediately following presentation of a single set of associates (see Experiment 2). We reasoned that amnesics' impairment in implicit retrieval of gist information in Experiment 1 may have been due to enhanced susceptibility to interference or accelerated forgetting, but eliminating these factors did not alter their pattern of performance. Thus, we conclude that a deficit in the encoding of gist information is the likely cause of amnesics' impairment.

How, then, is encoding of gist information distinct from encoding of verbatim information? According to fuzzy-trace theory (Reyna & Brainerd, 1995), encoding of verbatim information involves processing of the specific features of study items, whereas encoding of gist information involves processing of commonalities among study items. A related distinction is that between item-specific processing and relational processing (Einstein & Hunt, 1980; Hunt & McDaniel, 1993), and that distinction has also been invoked to explain differences between veridical and false memory (Arndt & Reder, 2003). In the context of an implicit stem completion task, processing of item-specific features leads to activation of a perceptual and/or lexical representation that mediates priming of verbatim information. Processing of similarities among different items leads to activation of a focused thematic representation that mediates priming for gist information. Consistent with this view, priming for nonpresented theme words is found in tasks in which priming is at least in part conceptually based but not in tasks in which priming is purely perceptually based (Hicks & Starns, 2005; McKone, 2004; Zeelenberg & Pecher, 2002). Surprisingly, one study has suggested that the thematic representations that support stem completion priming are modality specific, as priming for lures was substantially reduced on a visual stem completion test when the study associates had been presented auditorily (McKone & Murphy, 2000), but this finding has not been replicated (Hicks & Starns, 2005).

In light of the foregoing analysis, amnesics' impairment in encoding gist information can be understood as an inability to engage in the inferential processes whereby different items are

related and compared with one another and organized into a focused representation. A similar deficit has been posited to account for findings of impaired verbal category learning in amnesia (Kitchener & Squire, 2000). In that study, participants heard short verbal descriptions of imaginary animals belonging to the same category and were then asked to classify novel verbal descriptions according to whether they belonged to that category. Amnesic patients failed to acquire categorical knowledge—a finding that stands in stark contrast to their intact nonverbal category learning (Knowlton & Squire, 1993; Reed, Squire, Smith, Jonides, & Patalano, 1999; Squire & Knowlton, 1995). Kitchener and Squire (2000) proposed that verbal category learning differs from nonverbal category learning in that similarities cannot be directly apprehended, but performance depends instead on relational processing that allows one to compare and contrast exemplars across learning trials. Our findings, taken together with those of Kitchener and Squire, suggest that the effects of impaired relational encoding are not limited to explicit memory tasks but may affect performance on implicit memory tasks as well.

Aside from fuzzy-trace theory (Brainerd & Reyna, 2001; Reyna & Brainerd, 1995), which we have used as an explanatory framework for the findings in amnesia, another influential view (Balota et al., 1999; Roediger, Balota, & Watson, 2001) states that false memories are the result of an automatic spreading activation process whereby study items activate their associates. Because the lure is highly associated with many study items, activation summates across study items to make the lure strongly activated, and this activation leads to the production or endorsement of the lure on subsequent memory tests. Although several findings suggest that associative activation plays a role in the formation of false memories (e.g., Gallo & Roediger, 2003; Robinson & Roediger, 1997), it is not immediately obvious how such a view can account for the impairment in false memory in amnesia, as automatic activation processes have been shown to be intact in amnesic patients (Verfaellie, Cermak, Blackford, & Weiss, 1990; Verfaellie, Reiss, & Roth, 1995).

One way to accommodate an activation view is to suggest that amnesic patients activate the lure, just as nonamnesic individuals do, but that this activation dissipates abnormally rapidly over time. By this view, amnesics' impaired implicit memory for lures could be thought of as an item-specific impairment in conceptual priming. We do not favor such a view, however, as the available evidence suggests that amnesic patients show intact conceptual priming in the context of a word stem completion task (Carlesimo et al., 1996; Graf et al., 1985; Verfaellie, Keane, & Cook, 2001). Alternatively, it is possible that lure activation in itself is not sufficient for priming to occur but instead that it needs to elicit a covert verbal response that is consciously generated during the study phase (Underwood, 1965). Amnesic patients may fail to consciously generate the nonpresented lure. Although there is some evidence to suggest that covert verbal responses may be important for lure priming to occur (Lovden & Johansson, 2003), we know of no evidence to suggest that amnesic patients have impairments in the activation of phonological representations.

Regardless of the specific mechanism of impairment that is postulated, an activation account posits that the impairment in false memory in amnesia is due to impaired memory for specific, individual items rather than to degraded gist representations. Although an activation account possibly can be modified to accom-

modate the findings of the present study, it cannot account for the fact that false recognition for nonverbal materials is also impaired in amnesia (Koutstaal et al., 1999, 2001), as false recognition in these paradigms is unlikely to result from generating specific items at study. Thus, for the sake of parsimony, we favor an explanation of impaired false memory in amnesia in terms of degraded gist representations. In that context, the present study points to the importance of elaborative encoding processes that lead to the detection of semantic similarity across study items and the formation of gist representations—a process that is impaired not only in amnesic patients but also in patients with semantic dementia (Simons et al., 2005).

Our findings also provide further insight into the neural mechanisms associated with the encoding of gist information. It has been proposed that the extraction of gist depends on semantic processes mediated by lateral temporal neocortex (Melo et al., 1999; Simons et al., 2005) and possibly also on organizational processes mediated by prefrontal cortex (Melo et al., 1999). Only 2 patients in this study had lesions extending into lateral temporal cortex, and neither of them showed evidence of a semantic deficit. A number of patients, especially those with Korsakoff syndrome, exhibited signs of frontal dysfunction. To examine whether frontal impairment may be responsible for the impairment in implicit memory for gist, we calculated a composite frontal score for each patient, consisting of the patients' mean rank on four measures of frontal lobe functioning (Wisconsin Card Sort number of categories and percentage of perseverative errors, Controlled Oral Word Association Test total number of responses, and Trail Making Test—Part B reaction time). There was no significant correlation between the presence versus absence of priming for gist information and the composite frontal score in either Experiment 1 ($r = -.34$) or Experiment 2 ($r = -.12$). Thus, there was no evidence that the impairment in gist encoding was directly related to frontal deficits. Instead, our findings suggest that the encoding of gist information depends on memory-related structures in the medial temporal lobes and diencephalon. These findings extend previous results emphasizing the role of limbic structures in the retrieval of gist information (Cabeza, Rao, Wagner, Mayer, & Schacter, 2001; Schacter, Buckner, Koutstaal, Dale, & Rosen, 1997; Schacter, Reiman, et al., 1996; Slotnick & Schacter, 2004; for a review, see Schacter & Slotnick, 2004) and are generally consistent with the notion that the hippocampus and related structures play a critical role in the encoding of relationships between elements that are separated in time or space (Cohen, Poldrack, & Eichenbaum, 1997; Johnson & Chalfonte, 1994).

The present study not only extends the scope of the gist impairment in amnesia but also establishes limits on the implicit memory abilities of amnesic patients. Implicit memory for item-specific information in amnesia is supported by preexisting neocortical representations that can be directly activated by the study input, but implicit memory for gist information is impaired because it depends on the activation of a focused gist representation, the formation of which critically depends on the hippocampus and related structures.

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