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Intact Baseline Performance and Priming in Amnesia: Reply to Ostergaard and Jernigan

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In their comment on the article by S. B. Hamann, L. R. Squire, and D. L. Schacter (1995), A. L. Ostergaard and T. L. Jernigan (1996) reaffirmed their position that baseline perceptual identification performance and priming are impaired in amnesia. They also suggested certain shortcomings in the experiments of Hamann et al., who found normal baseline performance and normal priming in amnesia across a wide range of performance accuracies. In reply, the authors of this article suggest that the position of Ostergaard and Jernigan rests on selective consideration of data, inaccurate assumptions concerning 1 patient's priming performance (A.B.), and debatable concerns about the masking stimuli, ceiling effects, and presentation time of study items that were used. In addition, the authors of the present article suggest that Ostergaard and Jernigan have based their own experimental work on a task and test method that may not be optimal for studying priming.

On the basis of their experimental work, Jernigan and Ostergaard (1993) challenged the idea that priming effects in implicit memory tests are frequently intact in amnesic patients. They contended that amnesic patients are actually impaired at priming but that this deficit is typically masked because amnesic patients also have impaired baseline performance on the tasks used to assess priming. Impaired baseline scores would then artifactually inflate priming performance. Hamann, Squire, and Schacter (1995) subsequently reported four experiments examining baseline performance and priming effects in a perceptual identification task with amnesic patients and controls. We found that amnesic patients exhibited normal baseline performance across a wide range of stimulus conditions and accuracy levels. The only exception to this finding was a deficit limited to patients with Korsakoff's syndrome when relatively small stimuli were used ($<3°$ of visual angle). Finally, in Experiment 4, Hamann et al. (1995) demonstrated both normal baseline performance and normal priming effects for all of the amnesic patients. On the basis of these findings, we concluded that sparing priming in amnesia is not an artifact of impaired baseline scores.

The original proposal by Jernigan and Ostergaard (1993) was based on data from a multiple regression analysis that involved a control group and three patient groups: amnesic patients (9 out of 11 of whom were Korsakoff patients), patients with Huntington's disease, and patients with Alzheimer's disease. The indirect nature of their argument might be missed in a reading of their comment, which states, "When priming scores were corrected for . . . processing deficits, we [Jernigan & Ostergaard, 1993] found a relationship between priming and both recognition memory and mesial temporal lobe damage" (p. 125). This relationship was not demonstrated for the amnesic group alone but depended on additional data from the control group and the two groups of demented patients.

Jernigan and Ostergaard (1993) were correct to point out the possibly distorting effects of baseline performance on priming scores. However, our report (Hamann et al., 1995) demonstrated that (a) non-Korsakoff amnesic patients demonstrate both intact baseline scores and intact priming; and (b) patients with Korsakoff's syndrome exhibit impaired baseline scores only in a restricted set of stimulus conditions. Nevertheless, in their comment, Ostergaard and Jernigan (1996) continue to defend the inferences from their earlier report (Jernigan & Ostergaard, 1993), and they suggest a number of shortcomings in our experiments. Below we summarize several points.

Experiment 1: Patient A.B.

In our article, the non-Korsakoff amnesic patients always performed normally. However, 1 of these patients in our Experiment 1 (A.B.) initially exhibited much poorer baseline perceptual identification performance than did the controls and the other 3 non-Korsakoff patients. As explained in Footnote 1 of Hamann et al. (1995), 19 months after his initial testing, we learned that A.B. had worn nonprescription glasses during the testing sessions and that his family had ceased purchasing prescription lenses for him because he regularly

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lost or broke them. Subsequently, we acquired appropriate prescription lenses for him and tested him again on the materials in Experiment 1. Then he performed entirely normally. To assess whether his normal performance was attributable to prior exposure to the materials, we also tested him with novel materials and again observed normal baseline identification performance.

These observations suggest that A.B.'s initial poor performance was attributable to the fact that he was not wearing the prescription glasses that he once wore regularly and still required. In light of this new information, it became clear that his initial data were flawed and misleading. Hence, we presented his new data and placed his original data in a footnote, together with a brief explanation of what had occurred.

Ostergaard and Jernigan (1996) suggest that the original data from A.B. are more valid than the newer data, and they present his original data in their Figure 1 because (a) A.B. passed our initial acuity requirements; (b) at least 2 other participants exhibited poorer acuity than A.B.; and (c) no attempts were made to improve the visual acuity of other subjects. These arguments raise two separate questions: (a) Should the initial data from A.B. be considered as representative of his actual capacity for baseline perceptual identification? (b) Do the results of Experiment 1 demonstrate conclusively that amnesic patients can exhibit normal baseline performance? Ostergaard and Jernigan (1996) offer a positive response to the first question and a negative response to the second.

With respect to the first question, it is unclear why one would rely on data obtained under what turned out to be clearly inappropriate testing conditions. Subsequently, we interviewed anew all 31 controls in Experiments 1–4 and determined that only A.B. had previously worn prescription glasses that were not available during the experiment.[1] Thus, none of the controls who ordinarily wore prescription glasses were without them during the test sessions, and only patient A.B. was operating with a handicap in terms of his visual habits and his needs in everyday life.

Ostergaard and Jernigan (1996) also predict that if A.B. were tested with prescription glasses, he would fail to exhibit normal priming. Their reasoning was that when A.B.'s baseline performance is brought to normal levels, his priming performance should suffer. However, our Experiment 4 (Hamann et al., 1995) already showed that A.B. exhibits normal priming and intact baseline performance, even without his prescription glasses (in a condition that used larger stimuli than in Experiment 1). In any case, we have directly tested their prediction and found it to be incorrect. Specifically, we retested A.B. twice with his new glasses by using the procedure from Experiment 4, and on both occasions he exhibited intact baseline performance and intact priming (see Table 1). A.B. was first retested with the same materials and procedure that he had received when he initially participated in Experiment 4. For the second retest, A.B. was given the materials from Experiment 2.

The second question concerns whether Experiment 1 shows that non-Korsakoff amnesic patients exhibit normal baseline performance. At several points in their commentary, Ostergaard and Jernigan (1996) point to nonsignificant trends for amnesic patients to be impaired at baseline performance or priming performance relative to controls. They attribute these trends to low statistical power and imply that real deficits may be present. Although low power for detecting differences is always a concern and is especially important when marginally significant or near-significant results are obtained, such concerns are less serious when differences between groups do not begin to approach statistical significance. In any case, Ostergaard and Jernigan (1996) express concern about the fact that the nonsignificant differences in our Experiment 1 tended to favor the control group, not the amnesic group. We return to this issue below, but note here that the nonsignificant differences in baseline performance in Experiment 2 consistently favored the amnesic patients.

Experiment 2

In Experiment 2, we used a different kind of mask than in Experiment 1 and also used target words that differed in size and font from the words in Experiment 1. The results revealed entirely normal baseline performance in both Korsakoff and non-Korsakoff amnesic patients across a wide range of performance. Importantly, Patient A.B. exhibited normal baseline performance in this experiment, even without his prescription glasses. Yet Ostergaard and Jernigan (1996) mention Experiment 2 only in passing and ignore it when generalizing about the alleged lack of power in our experiments. The key point is that the 4 non-Korsakoff amnesic patients in Experiment 2 performed slightly more accurately than did the control group at each of the six exposure durations, and the patients with Korsakoff's syndrome performed slightly more accurately than the control group at all but one exposure duration. We do not attribute any special meaning to the nonsignificant advantage exhibited by amnesic patients in nearly all the conditions of Experiment 2; it presumably represents measurement error. We note simply that Ostergaard and Jernigan (1996) attributed significance to such fluctuations when they favored the controls but apparently overlooked these same nonsignificant trends when they favored the amnesic patients.

Ostergaard and Jernigan (1996) also suggest that our results are compromised by ceiling effects. However, in Experiment 2, ceiling effects were not present. Indeed, the performance of the control group was less than 80% correct for four of the six exposure durations and only slightly above 80% for the remaining two exposure durations. The amnesic patients exhibited normal perceptual identification performance at all six exposure durations across virtually the entire range of performance. The data from Experiment 4 are also not subject

[1] Thirty-one controls participated in Experiments 1–4 of Hamann et al. (1995). Twenty-nine of them wore eyeglasses or contact lenses during testing and 2 did not. Those 2 reported having regular eye examinations, which indicated that prescription lenses were unnecessary. All 29 individuals who wore corrective lenses reported that their prescriptions were up to date and that they could see and read comfortably. Additionally, 19 of the 29 indicated that they received annual eye examinations, and the other 10 indicated having regular, less frequent examinations. Finally, 4 participants reported that their glasses were nonprescription, but their eye doctors had suggested that nonprescription glasses were adequate in their cases.

Table 1
Patient A.B.'s Baseline Identification Performance and Priming (Proportion Correct) on Two New Priming Tests

	Exposure duration (ms)							
	50		67		83		Mean	
Variable	P	B	P	B	P	B	Priming	Baseline
Initial test	.47	.20	.77	.63	.97	.80	.19	.54
Retest 1	.57	.27	.77	.60	.90	.83	.18	.57
Retest 2	.60	.37	.83	.73	.97	.83	.16	.64
Controls	.61	.44	.82	.65	.81	.65	.17	.58

Note. Initial test = A.B.'s performance in Experiment 4 without prescription glasses (Hamann et al., 1995). Retest 1 = A.B.'s performance when retested 24 months later with prescription glasses and using the same materials and procedure as in the initial test. Retest 2 = A.B.'s performance in the same session with the same procedure as in Retest 1, again with prescription glasses, but with the materials from Experiment 2 (Hamann et al., 1995). Controls = corresponding data from the controls in Experiment 4 of Hamann et al. (1995). P = Primed performance; B = Baseline performance.

to ceiling effects. Baseline identification performance did not exceed 80% for any group, and primed performance did not exceed 90% for any group. Ostergaard and Jernigan (1996) ignore these data and instead focus their comments about ceiling effects on Experiment 3. As a result, they generalize incorrectly: "In all conditions in which performance was not contaminated by ceiling effects, amnesia patients consistently showed poorer identification performance than controls" (p. 28).

Finally, Ostergaard and Jernigan (1996) offer the opinion that the ampersand mask used in Experiments 2–4 did not effectively control exposure time of the test stimuli. We discuss this matter below (see Experiment 3). However, in Experiment 2, this issue is irrelevant. Whatever the effective exposure times in Experiment 2, the key point is that the range of exposure durations used yielded a wide range of performance accuracy from nearly 0% correct to about 80% correct. Because fixed exposure durations were used, the only way that the mask could have biased the results in favor of the amnesic patients would be if the mask somehow behaved in a systematically different manner for amnesic patients than for controls. There is no evidence or reason to suppose that this occurred.

In short, Ostergaard and Jernigan (1996) largely overlooked Experiment 2; yet it provided clear evidence against their proposal that baseline performance is impaired in amnesic patients. Indeed, the results of Experiment 2 provided strong evidence that amnesic patients, including patients with Korsakoff's syndrome, can exhibit normal baseline performance across a wide range of performance accuracy.

Experiment 3: Methodological Problems?

The purpose of Experiment 3 was simply to clarify the conditions under which patients with Korsakoff's syndrome exhibit spared or impaired perceptual identification performance. Ostergaard and Jernigan (1996) would dismiss the data from Experiment 3 (which found normal performance in patients with Korsakoff 's syndrome at visual angles larger than 3°) because they claim that the normal performance is an artifact of ceiling effects.

We make two observations. First, Ostergaard and Jernigan (1996) have apparently misconstrued the purpose of Experiment 3. The results of Experiment 1 had indicated that patients with Korsakoff's syndrome exhibited impaired baseline perceptual identification performance when words subtended 1° of visual angle. The results of Experiment 2 had indicated that the same patients exhibited normal performance when words subtended 10° of visual angle. Thus, we carried out Experiment 3 only to identify the stimulus size at which patients with Korsakoff's syndrome begin to perform in an impaired manner. Experiment 3 was not intended (or needed) to demonstrate normal baseline performance in patients with Korsakoff's syndrome because Experiment 2 had already done so.

Second, Ostergaard and Jernigan (1996) claim that the ampersand mask used in Experiments 2–4 was "not effectively controlling the exposure time of tachistoscopically presented [test] stimuli" (p. 127). They estimated that subjects were allowed an additional 50 ms of exposure duration because of the ineffective ampersand mask. If their assertion were correct that the ampersand mask provided 50 ms more exposure time than the random pattern mask, then in Figure 2 the controls tested at a 50-ms exposure duration with the ampersand mask (their score was 37% correct) should have performed at least as well as the controls in Figure 1 tested at a 100-ms exposure duration with the pattern mask (their score was 71% correct). (Indeed, the controls in Figure 2 should have performed above the level of the controls in Figure 1 because the data in Figure 2 were obtained with larger, easier-to-identify stimuli). Yet, neither of these outcomes occurred. In any case, the issue of the ampersand mask is irrelevant because Experiments 2 and 4 were both effective at testing performance across a wide range of accuracies, as stated above. Unless the ampersand mask affected amnesic patients differently than controls, the nature of the mask could not have influenced the results.

Experiment 4: Impaired Priming?

In Experiment 4, we demonstrated normal baseline performance and normal priming effects in amnesic patients within a single study. Once again, baseline performance of the amnesic patients was normal across the entire performance curve. All

nonsignificant differences favored the amnesic group. In addition, ceiling effects did not occur. Baseline performance scores of controls never exceeded 60% correct, and baseline performance of amnesic patients never exceeded 75% correct. Finally, because fixed exposure durations were used, the only way that the amnesic patients could have benefited disproportionately from an allegedly unreliable mask would be if variations in mask effectiveness had somehow favored the amnesic patients systematically. We know of no basis for such an idea.

Ostergaard and Jernigan (1996) raised two objections to our conclusion in Experiment 4 that amnesic patients exhibited normal priming. First, they noted that we did not strictly control presentation time of the words during the study task (which asked participants to rate how much they liked each word). Second, they argued again that the failure to find significant differences between groups may be due to low statistical power. With respect to presentation time, Ostergaard and Jernigan (1996) suggest that (a) amnesic patients may have required more time for the study task than did controls; and (b) a longer presentation time at study might have improved priming scores in amnesic patients, even though "in normal subjects [it] generally does not" (see Roediger & McDermott, 1993). However, their first suggestion is incorrect. We recorded the amount of time taken for the study task and found that although amnesic patients were a little slower than controls, the amount of time taken to rate each stimulus did not differ significantly among groups. The average time taken to rate each stimulus at study (i.e., the mean of the median presentation times) was 2,521 ± 436 s for the 11 amnesic patients and 1,963 ± 171 for controls, $t(19) = 1.15, p > .10$. In addition, there was no correlation between priming performance and the amount of study time taken for the rating task ($r = .05$; priming scores averaged across the three out of four exposure duration conditions that were free of floor effects). The results were similar within the amnesic group as well ($r = .04$). Moreover, when the amnesic patient who took the longest to rate stimuli was excluded from the analysis (Patient M.G. with a median presentation time of 6,019 ms), the remaining amnesic patients averaged only 209 ms longer than the control group, $t(18) = .62, p > .10$.

With respect to their concerns about low statistical power, Ostergaard and Jernigan (1996) note that the overall mean priming score in Experiment 4 (i.e., the proportion of studied words identified correctly minus the proportion of nonstudied words identified correctly) was numerically greater in the control group (.17) than in the non-Korsakoff amnesic patients (.15), which was in turn numerically greater than in the patients with Korsakoff's syndrome (.12). They state that these nonsignificant trends may reflect real differences that cannot be detected because of low statistical power. However, the overall F value was <1 when the full amnesic group was compared with the control group, was also <1 when non-Korsakoff amnesic patients were compared with controls, and was 1.14 when patients with Korsakoff's syndrome were compared with the control group. When between-group differences fail even to approach conventional levels of statistical significance, there seems little basis for treating them as other than experimental error.

Neuroanatomy

The view that baseline performance is impaired in amnesia was apparently based on the idea that striatal damage is common in amnesic patients (Ostergaard & Jernigan, 1996). Yet in their original study (Jernigan & Ostergaard, 1993), striatal damage was not detected in the amnesic patient group itself. Furthermore, the relationship between striatal volume and impaired baseline perceptual identification performance (i.e., processing deficits) did not reach significance for the amnesic group alone ($p = .08$; Ostergaard & Jernigan, 1996). Finally, in recent years the brains of a number of well-documented amnesic patients have been submitted to detailed and exhaustive histopathological examination, and no bilateral damage in the basal ganglia has been detected (Victor & Agamanolis, 1990; Patient R.B., Zola-Morgan, Squire, & Amaral, 1986; Patients L.M. and G.D., Rempel-Clower, Zola-Morgan, & Squire, 1994; Patient W.H., Rempel-Clower, Zola-Morgan, Squire, & Amaral, 1995), who participated in the study by Hamann et al., 1995). This is not to claim that striatal damage never results from the events or injuries that cause amnesia; the point is simply that striatal damage is by no means a common finding in amnesic patients, especially in the kinds of well-circumscribed amnesic patients who are recruited to standing groups for continuous study.

The view that priming is impaired in amnesia was based on the significant association observed between temporolimbic damage and priming scores in the pooled data from 30 subjects, including 10 demented patients (Jernigan & Ostergaard, 1993). We suggested that this association may have been carried by the demented patients. The fact that the association was markedly reduced ($p = .14$) when the 7 patients with Alzheimer's disease were removed from the data analysis (Ostergaard & Jernigan, 1996) provides some support for our suggestion.

Concluding Comments

Ostergaard and Jernigan (1996) suggest that none of the data reported by Hamann et al. (1995) is inconsistent with their views. As outlined in the preceding sections, this suggestion rests on a selective consideration of data, overinterpretation of nonsignificant results, and questionable and sometimes irrelevant criticisms. In this section, we try to offer some constructive comments. First, as noted by Jernigan and Ostergaard (1993), baseline performance measures are critically important and must be considered carefully whenever comparisons are made between different groups. Second, baseline identification performance is sometimes impaired in patients with Korsakoff's syndrome. Third, amnesia is not a fixed, unidimensional condition. The various injuries and diseases that damage the medial temporal lobe and diencephalic memory systems can vary in severity. As a result, amnesic patients vary in the severity of their memory impairment, and they also can vary in terms of how much damage occurs to other brain structures (such as the prefrontal cortex or neostriatum) that do not cause amnesia per se but do influence the pattern of cognitive impairment that is observed.

Finally, considering the variety of priming tasks on which

amnesic patients have been found to perform normally, it is surely time to move the so-called baseline performance issue beyond the single perceptual task and testing method on which it has so far rested. As discussed in detail elsewhere (Haist, Musen, & Squire, 1991), this testing method (Jernigan & Ostergaard, 1993; Ostergaard, 1994) is unique in that the whole stimulus is presented several times in succession until it can be identified. As a result, on both baseline and priming tests, controls may gain an advantage over amnesic patients by explicitly remembering partial information from the preceding test presentations and from the study list. When amnesic patients perform differently from controls on this particular task, the result may mean nothing more than that the task is not optimal for studying priming. When Ostergaard (1994) used an alternate procedure for testing priming that did not involve repeated presentation of whole stimuli, amnesic patients and controls exhibited virtually identical baseline scores and priming scores.

In summary, the empirical record shows that amnesic patients commonly have fully intact baseline and priming performance and that these abilities are fully dissociable from declarative (explicit) memory. The question of what brain systems support baseline performance and priming remain important questions for further work.

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