

Recognition and Recall in Amnesics

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Although there is considerable agreement that performance in direct memory tasks (e.g., recall, recognition) is more disrupted by amnesia than performance in indirect memory tasks (e.g., mirror reading, word completion), one may be able to further circumscribe the deficit within the domain of direct memory tasks. The present article explores whether recall is disproportionately disrupted by amnesia compared to recognition. If amnesia affects memory uniformly across different direct memory measures, recall of normal controls should not differ from the recall of amnesics when recognition scores of these two groups are equated. On the other hand, if recall is disproportionately disrupted, normal recall should be superior to amnesic recall even when recognition is equated. The present study equated amnesic recognition with that of controls by providing amnesics with 8 s of study time and normal subjects with 0.5 s. Amnesics with Korsakoff's syndrome, amnesics with other etiologies, and appropriate controls were examined. Normal recall was superior to amnesic recall even when no differences were found in recognition. The results further specify the selective nature of amnesia.

Amnesia is a selective, rather than global, deficit (Squire, 1982). There is considerable agreement that amnesia typically is characterized by a greater disruption in "direct memory tasks," in which memory is probed with instructions to recall or recognize past events, than in "indirect memory tasks," in which memory is indexed by, for example, the effect of prior exposure on word completion (Graf, Squire, & Mandler, 1984; Squire, Shimamura, & Graf, 1985). The present experiment examines whether the deficit found in direct memory tests can be further circumscribed.

Some theorists of amnesia posit that amnesic performance on direct memory tests differs only quantitatively from normal performance (see Hirst, 1982, for a review of the differing theories). This position gathers support from several studies that simulate amnesic performance in normals by depressing their memory to amnesic levels (Mayes & Meudell, 1981a, 1981b; Mayes, Meudell, & Som, 1981; Squire, Nadel, & Slater, 1981; Squire, Wetzel, & Slater, 1978; Woods & Piercy, 1974). For instance, Mayes and Meudell (1981a, 1981b) equated recognition of amnesics and controls by delaying the testing of the controls and then examined cued recall. They found that although the presence of cues aided amnesic memory, the facilitation was as great for the normals as it was for the amnesics. Building on these and related findings, Mayes et al. (1981) concluded that

"whenever the pattern of amnesic memory performance has been found to differ from that of normal controls' tested at the same short retention intervals, it has so far proved possible to show 'amnesic' patterns in normal subjects at prolonged retention intervals" (Mayes et al., 1981, p. 652; but see Schacter, Harbluck, & McLachlan, 1984). From this perspective, amnesia is a quantitative memory disruption reflected equally across different tests of direct memory.

In contrast, a number of theories of amnesia posit a circumscribed memory deficit that should adversely affect different direct memory tests to different degrees, such as retrieval deficit theories (Warrington & Weiskrantz, 1970, 1978), context theories (Hirst & Volpe, 1982; Huppert & Piercy, 1976; Kinsbourne & Wood, 1975), and some encoding theories (Butters & Cermak, 1980; Johnson, 1983; Johnson, Kim, & Risse, 1985). Support for such theories comes in part from a comparison of recognition and free recall. Although every investigator of amnesia has reported that amnesic free recall is substantially below that of normal controls, several researchers have documented strikingly good recognition in amnesic patients (Brooks & Baddeley, 1976; Cohen & Squire, 1980; Hirst & Volpe, 1982; Huppert & Piercy, 1976; Jacoby & Witherspoon, 1982; Johnson & Kim, 1985; Volpe & Hirst, 1983a, 1983b; Volpe, Holtzman, & Hirst, in press). Indeed, in some instances, amnesic recognition does not differ significantly from normal recognition (Hirst & Volpe, 1982; Huppert & Piercy, 1976; Johnson & Kim, 1985).

These sporadic reports of good recognition, however, are not sufficient support for the claim that amnesia disproportionately disrupts recall. To make the necessary argument, one must equate amnesic and normal memory on one of the measures—for example, recognition—and then determine whether normal recall is significantly better than amnesic recall. The experiment reported herein does just this, but it departs from the stud-

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ies of Mayes and Meudell, in which the scores of amnesics and normal controls were equated by delaying the testing of controls. This technique often results in quite low performance. In the present study, we elevated amnesic recognition until it equaled normal recognition and then compared free recall of amnesics and controls.

Huppert and Piercy (1977) have shown that even patient H.M.'s (Milner, Corkin, & Teuber, 1968) recognition can be raised to normal levels if he is given sufficient study time. We followed a similar procedure, allowing the amnesics 8 s to study what the controls had only 0.5 s to study. To increase the generality of the results, we tested two groups of amnesics: One consisted of amnesics with alcoholic Korsakoff's syndrome; the other was a group of amnesics with varying etiology. The one factor uniting members of the second group was the absence of any history of alcoholism, and for this reason, they are referred to as the nonalcoholic amnesic group. Little significance should be attached to the label "nonalcoholic," because any differences that may emerge between the two groups may or may not relate to their drinking history. Our interest is in the nature of recall and recognition with amnesia and the extent to which the relation between recall and recognition found to hold for one amnesic group holds for another.

Method

Subjects

The two experimental groups consisted of seven amnesic patients without a history of alcoholism and nine amnesic patients with alcoholic Korsakoff's syndrome. The two control groups consisted of seven normal adults matched to the nonalcoholic amnesics and nine alcoholics matched to the Korsakoffs.

The only cognitive complaint of the nonalcoholic amnesics was poor memory. They volunteered for the study. The experiment was completed at least 1 year after each patient's acute injury. In the 6 years that we have followed the patients, their memory deficit has remained stable. Two men and one woman suffered diffuse injury after global cerebral hypoxic ischemia (lack of adequate oxygen supply and obstruction to circulation). One man and two women had rupture and repair of anterior communicating artery aneurysm (arterial dilation due to pressure of blood on weakened tissues, forming a sac of clotted blood). One man suffered closed head trauma with prolonged coma. (For neurological details of the patients, see Volpe & Hirst, 1983a, 1983b, or write to B. T. Volpe, Department of Neurology, Cornell Medical College, New York, New York 10021.)

The average age of the patients was 51.0 years. Although there were differences in etiology, neuropsychological examinations were remarkably similar. They showed no perceptual or linguistic impairment in neuropsychological and neurological examination, had above-normal intelligence, did not confabulate, and were aware of their mnemonic problems. They had an average of 17.7 years of education. Their Wechsler Adult Intelligence Scale (WAIS) average full-scale score was 117.9 (range, 103–131); Ravens Progressive Matrices, 81% (range, 50–95); Boston Diagnostic Aphasia Evaluation, normal; Token test (for aphasia), no errors. The patients showed no signs of frontal lobe damage on a set of standard neuropsychological tests. Our patients achieved six categories on the Wisconsin card-sorting assessment with an average of 13.1 errors (range, 6–42; Milner, 1963, reported that the patients with frontal lobe damage that she studied achieved 1.3 categories with an average of 78 errors). Our patients could also accurately interpret proverbs and performed on the upper quartile on the controlled word association task (Benton, 1968). Frontal lobe damage typically impairs performance on these tasks.

Memory deficits were assessed using tests probing the free recall of pictures and words. The patients could recall an average of 1.6 unrelated words out of 10 after 30 s of distraction (which is significantly different from controls' 5.8 words, using Mann-Whitney U test, $U = 0$, $p < .002$), and 1.1 out of 20 objects depicted in complex pictures (control = 7.6, $U = 0$, $p < .002$). Five patients did not remember a single difficult word pair on the paired associate learning task of the Wechsler Memory Scale (WMS); two remembered one pair.

The alcoholic Korsakoff patients (eight men and one woman) were located at various nursing homes. Their average age was 66.1 years. The patients had a mean of 11.3 years of education. Their average WAIS verbal comprehension deviation quotient (VDQ) and perceptual organization deviation quotient (PDQ) were 104.6 and 101.4, respectively. The patients were severely disturbed in their ability to recall new information. For example, five patients were tested for paired-associate learning and paragraph recall with the lists and procedures used by Cohen and Squire (1981). For our patients, paired-associate learning scores, averaged over three lists of 10 unrelated pairs, were .20, .93, and 1.13 for three trials, respectively (similar to the values reported by Cohen and Squire, 1981, for their Korsakoff sample). For the recall of three paragraphs, the mean number of idea units out of a total of 21 per paragraph were 4.3 for immediate recall and 0.0 for 12-min delayed recall. Again, these values were close to those reported by Cohen and Squire (4.1 for immediate recall and 0.0 for delayed recall). All Korsakoff patients were tested on the WMS. Seven of the patients failed to recall a single idea on delayed prose learning; one recalled an average of 1.5 idea units; another recalled an average of 2.0 idea units. In the delayed hard (unrelated) paired-associate learning test, seven of the patients failed to recall a single item after the third trial, and two recalled one item.

The nonalcoholic control group (nine men) was matched to the nonalcoholic amnesics on years of education and age. Their average age was 53.2 years, and they had received 16.4 years of education. The alcoholic controls were members of Alcoholics Anonymous with long-term drinking histories. They reported being sober for at least 90 days prior to the experiment. Their average age was 63.1, with 11.4 years of education. Their average WAIS VDQ was 115.9 and PDQ was 102.9. Their mean paired-associate scores across three trials were 5.15, 7.70, and 8.56. Means for immediate- and delayed-paragraph recall were 9.53 and 8.22, respectively.

Except for the nonalcoholic amnesics, subjects were paid for their participation.

Materials

Each of four lists contained 40 words. In two of the lists, the words were unrelated. In each of the two other lists, the words fell into eight categories, five words per category. No word appeared in more than one list, and the categories were different in the two categorized lists. The words in the two unrelated word lists were one or two syllables long and of a frequency greater than 20 occurrences per million (Kučera & Francis, 1967). The words in the two categorized lists were taken from Battig and Montague norms (1969) and again were of a frequency greater than 20 occurrences per million. Each word in the list was typed on a 3 in. \times 5 in. index card. The order of the words in the four lists was randomly determined (with the constraint that words from the same category in the categorized lists were presented successively, following a card announcing the category label). There were two orderings for each list.

A two-item forced-choice recognition test was constructed for each list. For the categorized lists, each distractor was from the same category as the target. The distractors for the unrelated lists were selected from Kučera and Francis (1967) to match the targets in syllable length and word frequency. Targets and distractors were typed on cards with left-right positions of each equated across items.

Design and Procedure

For all subjects, a word list was presented to the subject, one word at a time, and subjects were told to study the words so that they could remember them when tested. The experimenter presented the words by showing the index cards at a prescribed rate. This task was followed by 5 min of distraction, in which subjects solved simple arithmetic problems. Subjects were then allowed approximately 3 min to recall the words from the studied list. Finally, they were given the forced-choice recognition test. On the recognition test, they were asked to rate their confidence in the response, with 1 being *least confident* and 3 being *most confident*.

There were four conditions in the study. The conditions differed in the rate of presentation of the words (8 s or 0.5 s) and the structure of the lists (categorized or unrelated). Words were presented at a rate of either 8 s per word or 0.5 s per word (pilot work had indicated that these values would equate the recognition of amnesics and controls). List structure and presentation rate were within-subject variables. Each subject studied four lists—a categorized list presented at the 8-s rate, and one presented at the 0.5-s rate, an unrelated list presented at the 8 s rate, and one presented at the 0.5-s rate.

The pairing of lists with rate of presentation, the order of the words in the lists, and the order of the conditions were all partially counterbalanced. A fully balanced design was specified, and a subject was placed in a cell as he or she became available. Unfortunately, the availability of the amnesics did not allow us to complete the design, but analysis of the data indicated that word order and the order of the conditions did not affect performance.

Results

Results are first presented for the conditions in which amnesics were given 8 s to study the words and controls given 0.5 s study time. These conditions were expected to equate recognition performance of amnesics and controls. Because this strategy was differentially successful for the two amnesic groups, results are presented separately for the two groups of amnesics and their corresponding controls.

Nonalcoholic Subjects

Table 1 shows the data for the nonalcoholic subjects. As anticipated, the manipulation of presentation rate successfully equated recognition performance of amnesics and controls. In an analysis of variance (ANOVA), main effects were found for structure, $F(1, 12) = 6.68, p < .03$, and type of test, $F(1, 12) > 50, p < .001$. There was also an interaction of Structure \times Type of Test, $F(1, 12) = 22.36, p < .001$. Thus, in keeping with prior findings (see Kintsch, 1970), the effect of categorization on memory was more pronounced in the recall scores than the recognition scores. Finally, and most important, there was an interaction of Memory Test \times Subject Group, $F(1, 12) = 11.25, p < .01$; in the amnesic group recall was more disrupted than was recognition. Indeed, as Table 1 shows, whereas the recognition scores of amnesics and controls were equivalent, for both categorized and unrelated lists, controls recalled more than did amnesics. Recall of the unrelated lists was low for both amnesics and controls; the amnesics only recalled on the average 1.2 words, whereas controls recalled an average of 2.8 words. Although any observable difference at these low values must be approached cautiously, it is worth noting that performance of

Table 1
Recall and Recognition of Nonalcoholic Amnesics and Controls

Group	Recall		Recognition	
	Proportion correct	SD	Proportion correct	SD
	Categorized			
Amnesics	.12	.07	.82	.10
Controls	.28	.12	.81	.10
	Unrelated			
Amnesics	.03	.03	.86	.10
Controls	.07	.05	.84	.06

Note. For the nonalcoholic amnesics, study time was 8 s. For controls, study time was 0.5 s.

controls was twice that of amnesics, and that this difference is significant, $t(6) = 7.23, p < .01$.

Alcoholic Subjects

An increase in study time did not effectively equate the recognition of Korsakoffs and their corresponding controls. For the unrelated words, for instance, at the 8-s rate the Korsakoffs recognized 70% of them, whereas at the 0.5 s rate the alcoholic controls recognized 82%. The two groups could be equated, however, by selecting a subset of the subjects in the two groups. Specifically, the three Korsakoff patients with the lowest recognition for the unrelated list were discarded from the sample as well as the three controls with the highest recognition scores for the unrelated list. This maneuver did not appreciatively affect mean age, VDI, PDQ, or educational background. Further analysis is confined to these two yoked groups of amnesics and controls.

Table 2 shows the relevant data. In an ANOVA, there were main effects for subject group, $F(1, 10) = 5.71, p < .04$, and type of test, $F(1, 10) > 50, p < .001$. There was also a significant interaction of Structure \times Type of Test, $F(1, 10) = 7.01, p < .02$. As with the nonalcoholic subjects reported above, recall scores were more affected by structure than were recognition scores.

The important finding in the ANOVA is that there was an interaction of Type of Test \times Subject Group, $F(1, 10) = 26.79, p < .001$. This result indicates that when recognition was equated, Korsakoffs' recall was worse than would be expected from matched controls' performance. However, there was also a significant three-way interaction of Structure \times Test \times Subject Group, $F(1, 10) = 6.21, p < .04$, suggesting that the interaction Test \times Subject Group was a function of the structure in the to-be-remembered list.

To investigate this more closely, we conducted separate ANOVAs on the data for the categorized lists and the unrelated lists. For the categorized lists, there was indeed an interaction of Test \times Subject Group, $F(1, 10) = 18.47, p < .01$. However, for the unrelated list, the interaction of Test \times Subject Group was not significant, $F(1, 10) = .04, p < .05$. Korsakoffs, however,

Table 2
*Recall and Recognition of Alcoholic Korsakoff
 Amnesics and Alcoholic Controls*

Group	Recall		Recognition	
	Proportion correct	SD	Proportion correct	SD
Categorized				
Korsakoffs	.02	.04	.74	.08
Controls	.22	.15	.73	.05
Unrelated				
Korsakoffs	.005	.01	.74	.04
Controls	.04	.04	.78	.07

Note. For the Korsakoff amnesics, study time was 8 s. For controls, study time was 0.5 s.

recalled little of the original list. One subject recalled one word; the other five recalled nothing. This floor effect may have masked the size of the difference between the recall of the Korsakoffs and their controls. An analysis of the log ($x + 1$) transform of the unrelated data came closer to verifying the pattern evident in the means; the interaction of Type of Test \times Subject Group reached an acceptable significance level, $F(1, 10) = 4.13$, $p < .07$.

Performance Including the Presentation Rate Variable

Although the results relevant to the major question motivating this study have been presented, the design also permitted us to look at the effects of presentation rate and potential interactions between presentation rate and other variables. The data for all conditions are shown in Table 3 (the nonalcoholic groups in the upper portion and the "yoked" alcoholic groups in the lower). Separate analyses of the data from the two types of amnesic populations (nonalcoholic and alcoholic, respectively, in the analyses that follow) produced generally consistent results. Amnesics performed worse than controls, $F(1, 12) = 49.37$ and $F(1, 10) = 46.15$, $p < .001$; recognition scores were higher than recall scores, $F(1, 12) > 50$, $p < .001$ and $F(1, 10) > 50$, $p < .001$; 8-s presentation yielded better performance than 0.5-s presentation, $F(1, 12) = 74.76$, $p < .001$ and $F(1, 10) = 132.91$, $p < .001$. There was also a significant interaction of Subject Group \times Presentation Rate, $F(12) = 11.33$, $p < .01$ and $F(1, 10) = 10.52$, $p < .01$, indicating that increasing study time benefited controls more than amnesics.

There were several interactions involving list structure: Structure \times Subject Group, $F(1, 12) = 8.65$, $p < .02$ and $F(1, 10) = 16.09$, $p < .01$; Structure \times Type of Test, $F(1, 12) = 80.64$, $p < .001$ and $F(1, 10) = 10.51$, $p < .01$; and Structure \times Type of Test \times Subject Group, $F(1, 12) = 14.28$, $p < .01$, and $F(1, 10) = 37.38$, $p < .001$. These interactions indicate that controls were more likely to benefit from list structure than were amnesics; recognition performance was not affected by list structure, whereas recall was better for categorized lists than for unrelated lists; and the advantage of controls from greater list structure was confined to recall.

Confidence Ratings, Proactive Interference, New Intrusions, and Conditional Probabilities

Confidence ratings were analyzed separately for the nonalcoholic and the yoked alcoholic group. The analysis is confined to those conditions in which recognition scores of amnesics and controls were equated—that is, the 8-s presentation condition for the amnesics and the 0.5-s presentation condition for the controls. Both the average confidence ratings for the correctly recognized items and the average rating for errors were considered. The data are presented in Table 4. For the nonalcoholic subjects, confidence ratings for the correctly recognized items were significantly higher than the confidence ratings for errors, $F(1, 12) = 60.57$, $p < .001$, and confidence ratings on the recognition tests for the related word lists were higher than those for the unrelated word lists, $F(1, 12) = 9.46$, $p < .01$. Interestingly, confidence ratings for the nonalcoholic amnesics did not differ significantly from their controls, $F(1, 12) = .14$, $p > .05$. The interactions of Group (amnesic vs. control) \times Relatedness, and Group \times Correctness were also not significant, $F(1, 12) = .04$, $p > .05$; $F(1, 12) = 3.44$, $p > .05$, respectively. Thus, it would appear that the confidence ratings of the nonalcoholic amnesics did not differ from those of the controls and that the pattern of the ratings was similar for both groups. Both amnesics and controls were more confident when being tested on a related list than an unrelated list. Both groups were also more confident in their response when it was correct than when it was incorrect.

For the alcoholic groups, again, the confidence ratings of the Korsakoffs and their controls did not differ significantly from each other, $F(1, 10) = .26$, $p > .05$. Moreover, as with the nonalcoholic group, correct responses were given higher confidence ratings than incorrect responses, $F(1, 10) = 24.43$, $p < .001$. In contrast to the nonalcoholic group, confidence ratings for test items for related lists did not differ from those for unrelated lists, $F(1, 10) = .35$, $p > .05$. The nearly significant interaction of Relatedness \times Group, $F(1, 10) = 4.58$, $p < .06$, indicates that whereas the nonalcoholic controls showed a trend toward a relatedness effect (confidence ratings of 2.11 for unrelated vs. 2.21 for related), the Korsakoffs did not (2.13 for unrelated vs. 1.95 for related). This pattern of results is consistent with the recognition scores of the nonalcoholic subjects: Korsakoffs did not benefit from categorized word list, whereas the alcoholic controls did. Finally, although correct responses had higher confidence ratings than incorrect responses, there was a significant interaction of Group \times Correctness, $F(1, 10) = 5.12$, $p < .05$, indicating that the difference between confidence ratings of correct and incorrect responses was larger for the controls than it was for the Korsakoffs. The present design does not allow a closer examination of this latter result, but it could arise either because the two groups were not as equated as indicated by the recognition scores or because the response biases of Korsakoffs may differ from those of the alcoholic controls.

There were on the average 1.13 prior list intrusions for all subjects. An ANOVA failed to reveal any significant main effects or interactions. An average of 1.97 new items intruded in the subjects' recall. Again, an ANOVA on the data concerning "new intrusions" failed to find any significant main effects or interactions.

Finally, in order to examine the effect recall had on recognition, we examined the conditional probability of recognizing

Table 3
Recall and Recognition With Varying Study Time and List Structure for Different Groups

Group	Categorized				Unrelated			
	Recall		Recognition		Recall		Recognition	
	Proportion correct	SD	Proportion correct	SD	Proportion correct	SD	Proportion correct	SD
Nonalcoholics								
Amnesics								
0.5 s	.05	.07	.68	.08	.01	.01	.70	.11
8 s	.12	.07	.82	.10	.03	.03	.86	.10
Controls								
0.5 s	.28	.12	.81	.10	.07	.05	.84	.06
8 s	.60	.20	.97	.04	.33	.13	.95	.04
Alcoholics								
Korsakoffs								
0.5 s	.03	.04	.64	.06	.005	.01	.45	.20
8 s	.02	.04	.74	.08	.005	.01	.74	.04
Controls								
0.5 s	.22	.15	.73	.05	.04	.04	.78	.07
8 s	.52	.15	.96	.03	.10	.09	.88	.07

a recalled item. The analysis was confined to the 8-s related condition for nonalcoholic amnesics and the 0.5-s related condition for their controls because the amnesic recall scores in all other conditions were so low that one could not meaningfully calculate the conditional probabilities. The probability of recognizing a recalled item was .95 for the nonalcoholic amnesics and .97 for their controls. The probability of recognizing an item that had not been recalled was .79 for the amnesics and .78 for their controls. Thus, the prior recall test appeared to influence recognition equally for the nonalcoholic amnesics

and their controls, although no strong statement to this effect can be made because of potential item selection effects.

Discussion

The results suggest that recognition and recall are differentially affected by amnesia. When recognition of amnesics and controls was equated, recall of the controls was between 200% and 1,200% greater than for the amnesics. This finding indicates that amnesia (at least of the sort represented by the patients we studied) does not lead to a uniform depression of memory. Amnesia affects recall more than it affects recognition.

The present results suggest several empirical and theoretical considerations. For instance, the present results raise several empirical questions when they are examined in conjunction with the work on amnesic cued recall (Mayes & Meudell, 1981a, 1981b; Mayes et al., 1981; Squire et al., 1978; Warrington & Weiskrantz, 1974; Weiskrantz & Warrington, 1975; Woods & Piercy, 1974). This work showed that cued recall scores of amnesics and controls were equal when recognition scores were equated by delaying the testing of controls. The finding indicates that memory traces formed by amnesics are only quantitatively different from those of normals. If, as several memory theorists have argued (see Tulving, 1983), recall and recognition are merely on a continuum, with cued recall somewhere in between, then the finding with cued recall should carry over to studies of free recall and recognition. That is, in our experiments, free recall of amnesics and controls should have been similar in those conditions in which recognition was similar. Clearly, this was not the case.

The differences between our findings and the work on cued recall may reflect differences in the processes involved in cued and free recall, but it may also reflect the different methodologies used in the cued recall work and the present method. As noted in the introduction, the cued recall investigators equated

Table 4
Confidence Ratings With Varying Presentation Rates for Different Groups

Group/response	Categorized		Unrelated	
	Rating	SD	Rating	SD
Nonalcoholics				
Amnesics ^a				
Correct	2.33	.33	2.13	.27
Incorrect	1.93	.30	1.73	.21
Controls ^b				
Correct	2.49	.17	2.12	.44
Incorrect	1.70	.32	1.62	.43
Alcoholics				
Korsakoffs ^a				
Correct	2.00	.50	2.25	.47
Incorrect	1.91	.48	2.03	.37
Controls ^b				
Correct	2.37	.43	2.38	.37
Incorrect	2.07	.49	1.85	.44

^a 8-s presentation rate.

^b 0.5-s presentation rate.

amnesics and controls by delaying testing. Consequently, performance of all subjects was quite low. Indeed, in some cases, it was not significantly different from chance. Squire et al. (1978), for instance, reported that forced-choice recognition performance of Korsakoffs and controls was between 7 and 8 correctly identified items out of 12 (by binomial, for 7, $p = .194$; for 8, $p = .073$). The results, then, may have been influenced by floor effects. (Interestingly, our data were least sensitive to the interaction of Subject Group \times Type of Test in the condition where amnesics' performance was the lowest—Korsakoffs' recall of unrelated lists.) The present work indicates that floor effects can be avoided. It remains to be seen what will happen to amnesic cued recall as study time is increased to equate amnesic and normal recognition.

In the present study, the alcoholic amnesics were worse than the nonalcoholic amnesics. This difference could be traced to the effects of alcohol, the density of the amnesia, or merely the advantage the nonalcoholic subjects obtained with their 6 years of additional formal education (see Moscovitch, 1982 for a discussion of the differences between Korsakoffs and nonalcoholic amnesics). Whatever the cause of the difference, it did not affect the pattern of the results. The recall of the Korsakoffs may have been close to the floor in some conditions, but in every instance, recall of the controls was at least twice as good as recall of the amnesics. Thus, the finding that the recall deficit of amnesics is out of proportion to their recognition deficit appears to be characteristic of both amnesic groups.

One qualification is necessary, however. The clearest results were found in the nonalcoholic amnesics, who suffered the least dense amnesia, and significant results were found for the Korsakoff group only when a "yoking" procedure eliminated the most severe amnesics in that group. Consequently, the relatively preserved recognition that we observed may be a characteristic specific to "mild" amnesics. Of course, dividing amnesics into "mild" and "severe" is fraught with difficulty inasmuch as (a) there is no acceptable means of measuring the severity of amnesia, (b) the density of amnesia is a continuum rather than a dichotomy, and (c) even so-called mild amnesics have memory problems severe enough to make normal life impossible and supervision a necessity. Moreover, even if the present findings were confined to mild amnesia, they would still bear on the nature of the breakdown of memory with brain damage insofar as they suggest that recall and recognition are mediated by different processes. Thus, severity of amnesia might be a product of the number of relatively independent types of cognitive processes affected, rather than, say, the product of the extent of the damage to a single type of process or mechanism. This possibility suggests that, despite some logical difficulties of defining severity of amnesia independent from the measures of interest, an attempt should be made to repeat the present study on a group of "severe" amnesics.

The comparison between memory for categorized and unrelated word lists also raises theoretical as well as empirical issues. Amnesics can use categorization strategies to their benefit (Warrington & Weiskrantz, 1971), and Korsakoffs may fail to categorize lists spontaneously (Butters & Cermak, 1980). The present result extends these findings by demonstrating that both alcoholic and nonalcoholic amnesics are less likely than controls to use the structure in a list to improve recall. Along with work on the encoding of spatial and temporal information and

the effect of deep processing on amnesic memory (Butters & Cermak, 1980; Hirst & Volpe, 1984a, 1984b), the present results suggest that amnesics and controls may engage in different strategies or draw on different skills when memorizing and remembering material.

Neither recognition nor recall is a distinct process that can be damaged as a unit. Success at either depends on a complex of processes, some of which underlie both recognition and recall, others of which are unique to one or the other form of memory. Presumably, the 0.5-s presentation rate in this study affected the processing of the controls in such a way that their recognition and the processes underlying it came to resemble that of the amnesics at an 8-s presentation rate. Nevertheless, even when these recognition processes were equated, the findings indicate that an aspect of processing underlying successful recall was disrupted by amnesia.

Several models offer specific hypothesis about the nature of the disrupted processing. For instance, the present findings are consistent with models of amnesia that posit only shallow encoding with amnesia (Butters & Cermak, 1980) or those positing disruption to the component of memory responsible for the occurrence and later retrieval of "reflective activity" (Johnson, 1983; Johnson et al., 1985). During study, a subject must find relations among items within the list, between list items and more general world knowledge, or between the list items and the context in which the lists are studied. At test, the subject must generate cues to reactivate intralist relations, extralist relations, or contextual relations. These self-generated activities during acquisition and test are members of the general class of "reflective" processes, which are presumably more important in recall than in recognition (Johnson, 1983; Smith, 1979; Smith, Glenberg, & Bjork, 1978). It is not that recognition never benefits from reflective activity—clearly it does (see, for instance, Paivio, 1971, for a discussion of the effects of imaging on recognition). However, under many circumstances, recognition may draw primarily on a record of prior perceptual processing, whereas recall is generally more dependent on a record of reflective processing.

The present results are also consistent with models of memory in which recognition depends in part on processes like priming (Jacoby, 1982, 1984; Mandler, 1980). It appears that priming may be preserved with amnesia (Graf et al., 1984; Squire et al., 1985). According to these models, preserved priming may be enough to allow the amnesic to differentiate targets from distractors, but would have little or no effect on retrieval or recall. Consequently, recognition would be less disrupted by amnesia than recall (see however, Squire et al., 1985).

In sum, the present study shows that the recall of both alcoholic and nonalcoholic amnesics is much worse than one would expect from their recognition. The results suggest that amnesia reflects not a general depression of memory but rather a selective disruption of an aspect of memory critical to successful recall.

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