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Spatial Location Memory in Amnesia: Binding Item and Location Information Under Incidental and Intentional Encoding Conditions

Barbara L. Chalfonte

Princeton University, USA

Mieke Verfaellie

Memory Disorders Research Center, Boston University School of Medicine and Boston Department of Veterans Affairs Medical Center, USA

Marcia K. Johnson

Princeton University, USA

Lisa Reiss

Memory Disorders Research Center, Boston University School of Medicine and Boston Department of Veterans Affairs Medical Center, USA

Items located within an array were presented to alcoholic Korsakoff and nonalcoholic mixed-etiology amnesics and to alcoholic and normal controls. Recognition memory for the locations of items was tested after incidental and intentional encoding. When equated on item recognition, neither Korsakoff amnesics nor alcoholic controls benefited from intentional, relative to incidental, encoding instructions. Furthermore, Korsakoff amnesics showed neither disproportionately impaired incidental nor intentional location recognition memory relative to alcoholic controls. In contrast, mixed-etiology amnesics profited significantly from intentional location acquisition relative to incidental instructions, and were impaired somewhat in incidental, but not intentional, location memory relative to normal controls. We discuss these data in relation to Mayes' (1992) contextual memory deficit hypothesis and Hirst's (1982) automatic encoding deficit account, and propose an alternative framework in which the location memory deficit observed in mixed-etiology amnesics is interpreted as a disruption to the ability to bind item and location information.

Requests for reprints should be sent to Barbara L. Chalfonte, Department of Psychology, Mount Holyoke College, South Hadley, MA USA 01075. e-mail: bchalfon@mhc.mtholyoke.edu

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INTRODUCTION

Amnesics show general memory deficits on a range of tasks (e.g. Cohen & Eichenbaum, 1993; Squire, 1987). Among these, one that has received special attention is amnesics' deficits in memory for spatial location (see Schacter & Nadel, 1991 for a review). Deficits in memory for spatial location include recollection of the location of experimental items (e.g. Hirst & Volpe, 1984a; Smith & Milner, 1981, 1984; Warrington & Baddeley, 1974), knowledge of the locations of previously known landmarks (e.g. Hirst & Volpe, 1984b), and navigation of experimental mazes (e.g. Corkin, 1965; Milner, 1965). Neurophysiological evidence implicates the hippocampus in the computation and storage of spatial location information (e.g. O'Keefe & Nadel, 1978). Given that human amnesia often includes lesions to the hippocampus and adjacent cortical areas, the resulting profound disruption of memory for spatial location does not seem controversial. The picture concerning spatial location memory in amnesia remains ambiguous, however, because two important questions have not yet clearly been answered. First, is memory for spatial location disproportionately disrupted relative to memory for other kinds of information, or does disrupted memory for spatial location represent one facet of a more general memory impairment? Second, what effects does the intention to remember spatial location have on memory for the information? The present experiment addresses these questions.

Is Spatial Location Memory Disproportionately Disrupted?

Consider, first, the literature on spatial location memory in patients with focal, unilateral temporal lobe lesions. Using a standard method to assess spatial location memory (Mandler, Seegmiller, & Day, 1977), Smith and Milner (1981, 1984, 1989) directed patients with either right or left temporal lobe lesions to name and to estimate the price of 16 visually distinct, toy objects placed in various locations on an unmarked board. Immediately following the study phase, subjects were asked to recall the object names. No differences in object recall were noted between controls and either lesion group—all groups recalled about half of the object names. Subjects were then given a cued-recall test of location memory; they were asked to place the objects onto the board in the same positions in which they were seen originally. On this task, the mean displacement between the placed object and its original location was no different in normal controls and patients with left temporal lobe lesions; in contrast, patients with right temporal lobe lesions performed more poorly than either of these groups. Kesner, Hopkins, and Chiba (1992) reported a similar pattern of results in their patients with unilateral temporal lesions. Subjects who are not profoundly amnesic, but who have lesions to the right temporal lobe, demonstrated a spatial location memory deficit in the absence of impaired memory for objects. These data suggest that spatial location memory is disproportionately disrupted relative to memory for other information insofar as memory for objects in lesioned patients was intact relative to controls, and memory for the locations of the objects was impaired.

The question remains, however, whether this same pattern holds for patients who are densely amnesic. Smith and Milner (1981) tested HM using the same paradigm described earlier. They found that HM's spatial location memory was grossly impaired relative to controls. However, HM was also unable to recall any of the objects' names. Because HM's memory for the objects was poorer than controls', it remains unclear whether (a) HM's memory is generally impaired, or (b) location memory is disproportionately impaired, but could not be observed because memory for objects was not equivalent or experimentally equated between HM and the control subjects (e.g. Mayes & Meudell, 1981).

Cave and Squire (1991) equated amnesic and control subjects' memory for objects by testing amnesics immediately after acquisition and by testing controls after a 3–5 week delay. Under these conditions, the two groups of amnesic subjects, one with bilateral hippocampal lesions and the other with bilateral diencephalic damage (primarily alcoholic Korsakoff patients), had the same level of object recall performance as normal control subjects. When object recall was equated, amnesics' cued-recall memory for the objects' spatial location was also equivalent to that of normal controls. One problem, however, was that performance on the spatial location task was very close to floor levels, potentially obscuring differences in the amnesic and control subjects' performance. Indeed, different results were obtained by Shoqeirat and Mayes (1991) when they equated the object recognition performance of amnesics of mixed etiology and controls by providing multiple presentations of the objects to amnesics, thereby avoiding floor effects. In this case, amnesics performed worse than controls on several measures of spatial location memory. If Shoqeirat and Mayes' (1991) data provide a more accurate reflection of amnesics' impairment, then these data, like those for patients with unilateral lesions, suggest that memory for spatial location may be disproportionately impaired in patients who are densely amnesic.

What are the Effects of Intention-to-remember on Spatial Location Memory?

The studies reviewed in the previous section provisionally suggest that memory for spatial location may be disproportionately impaired. Globally amnesic patients in these studies acquired the spatial location information incidental to making price estimates of and naming toy objects (Cave & Squire, 1991; Smith & Milner, 1981) or naming shapes and making relative size comparisons (Shoqeirat & Mayes, 1991). However, these studies did not address whether there is any benefit of encoding location intentionally rather than incidentally, and if so, whether controls and amnesics benefit to the same degree. Consider that Hasher and Zacks (1979) suggested that location may be encoded automatically when object information is encoded; by definition, instructing subjects to intentionally study location in addition to object information should not improve memory for spatial location. If this supposition holds for amnesics as well as for normals, then there should be no change in the pattern of location memory performance when subjects intentionally study location. Mayes, Meudell, and MacDonald (1991) evaluated amnesic and control subjects' spatial location memory after instructing them to study both words and the quadrant in which the words were located. By showing fewer words to amnesics, their level of word recognition was similar to that of controls. As in the study by Shoqeirat and Mayes (1991), amnesics' cued-recall of spatial location was impaired relative to that of controls. Under intentional encoding instructions, amnesics apparently also show disproportionately impaired memory for spatial location.

Although intentional memory for spatial location may be impaired in amnesics relative to controls, perhaps amnesics still profit from intentional relative to incidental encoding in a way that control subjects do not. Smith (1988) examined HM's memory for the locations of toy objects after estimating their prices, an incidental encoding condition, and after estimating their prices and intending to remember the locations of the objects, an intentional encoding condition. Neither HM nor the controls performed any differently on the spatial location test whether location information was acquired intentionally or incidentally. In both cases, HM's location memory was poorer than controls', although whether this location deficit was disproportional is uncertain because memory for the objects was not tested.

Hirst and Volpe (1984a) tested groups of mixed-etiology amnesics and controls using a similar method in which subjects were directed to remember only the items or the items and their locations. In this case, Hirst and Volpe found no effect of encoding instructions on controls' memory for spatial location, but the amnesics profited by intentionally encoding spatial location relative to incidental acquisition. Object memory performance for the amnesics was about half of that for the controls. In a similar study, MacAndrew and Jones (1993) investigated the effect of intentionally encoding spatial location for alcoholic Korsakoff amnesics and controls. Unlike Hirst and Volpe (1984a), MacAndrew and Jones found that neither their amnesics nor controls benefited from intentional location instructions on cued-recall tests of spatial location memory. The investigators attempted to equate memory for the objects, but controls continued to demonstrate somewhat better recognition and recall memory for the objects even after a 24-hour delay. Kovner, Dopkins, and Goldmeier (1988) tested both mixed-etiology and Korsakoff amnesics in comparison to controls. For all three groups, they found no effects of intentionality on the ability to replace pictorial objects into their former locations. Memory for these pictorial objects was not evaluated.

With the exception of Hirst and Volpe (1984a), investigators have not found any benefit of intentional acquisition of location information relative to incidental acquisition for either amnesics or controls. However, final resolution of this issue, and of the question of a disproportionate location memory deficit in amnesia, must take into account important methodological concerns and potential differences in amnesic populations.

Factors That May Influence Spatial Location Memory Results

In order to determine whether spatial location memory is disproportionately impaired, memory for location must be compared to memory for some other type of information that has been equated between subject groups (e.g. Mayes & Meudell, 1981). Although non-spatial, item memory has been equated between amnesic and control groups in some studies (Cave & Squire, 1991; Mayes et al., 1991; Shoqeirat & Mayes, 1991), it has not in others (Hirst & Volpe, 1984a; Kovner et al., 1988; Smith, 1988; Smith & Milner, 1981). Moreover, the method by which amnesic and control performance is equated has taken one of two approaches: in the first case, amnesics are given more repetitions of material and less material than controls (Mayes et al., 1991). In the other, amnesics are tested immediately and the testing of controls is delayed (Cave & Squire, 1991; MacAndrew & Jones, 1993). Sometimes a combination of these approaches has been used (Shoqeirat & Mayes, 1991). The drawback of providing more repetitions for amnesics is that memory is based on different encoding conditions for amnesics and controls. One drawback of delaying testing is that testing conditions differ for amnesics and controls, and another is that performance on tests may approach floor especially after long delays (e.g. Cave & Squire, 1991). Providing additional repetitions of the material to amnesics has the clear benefit of avoiding floor performance, and this approach is less likely than delayed testing to change the cognitive processes used at test for one experimental group relative to the other (e.g. Verfaellie & Treadwell, 1993).

When determining whether spatial location memory is disproportionately impaired, it is crucial to test memory for location and the comparison case (e.g. object memory) in the same way. Unless object memory (for example) and location memory are assessed using the same type of test, any observed differences may be a consequence of the type of test used rather than a consequence of any amnesia-related deficit in memory for location. Previous studies vary in the similarity between tests used to evaluate object memory and those used to evaluate location memory. Studies using object recall and location cued-recall (i.e. replace items into their original locations) evaluated memory for these kinds of information in a more similar manner (e.g. Cave & Squire, 1991; Hirst & Volpe, 1984a; Smith & Milner, 1981) than those studies using object recognition and location cued-recall (e.g. Mayes et al., 1991; Shoqeirat & Mayes, 1991). In order to fairly evaluate deficits in memory for location compared to memory for objects, object memory and location memory should be evaluated using the same kind of test.

Finally, some consideration must be given to the etiology of the amnesic group being tested. For example, Hirst and Volpe (1984a) found that for their mixed-etiology, nonalcoholic amnesics, memory for location improved with intentional instructions whereas MacAndrew and Jones' (1993) Korsakoff patients did not benefit. Although these two studies varied in a number of ways, a potentially important difference concerns the sub-type of amnesic patient tested. Some researchers have argued that Korsakoff amnesics differ from nonalcoholic bitemporal amnesics in the ability to encode contextual information (e.g. Parkin & Leng, 1992). For example, Korsakoff amnesics perform worse than bitemporal amnesics when making judgements of temporal order (Shimamura, Janowsky, & Squire, 1990; Squire, 1982) or when performing a recognition memory task that requires the encoding of distinctive temporal context (Parkin, Leng, & Hunkin, 1990). Perhaps the disruption of memory for spatial location represents another dimension along which these amnesia subtypes differ. Cave and Squire's (1991) amnesics with hippocampal lesions had numerically poorer location memory than their diencephalic amnesics. In contrast, Kovner et al.'s (1988) Korsakoff amnesics had numerically poorer memory for spatial location than did their mixed-etiology amnesics. Although these two studies portray somewhat different pictures, these findings taken together nonetheless suggest that the nature of the spatial location memory deficit may be different in nonalcoholic, mixed-etiology amnesics than in Korsakoff patients.

Experimental Overview

Taking into account the issues already discussed, the goal of the present experiment was to observe (a) whether memory for spatial location was disproportionately impaired in amnesics, and (b) whether intentional encoding of location yielded superior location memory performance relative to incidental encoding. To do this, we tested two different groups of amnesic subjects, Korsakoff patients and nonalcoholic, mixed-etiology patients, under incidental and intentional study conditions. Item recognition performance between the amnesic groups and their controls was equated by providing an extra repetition of the material and by reducing the amount of material to be studied by amnesics. Using a paradigm developed by Chalfonte and Johnson (1996) to study location memory in young and older adults, all groups of amnesic and control subjects saw several coloured objects located within an array and were tested in three conditions: study Item/test Item; study Item/test Item&Location (incidental location); and study Item&Location/test Item&Location (intentional location). Memory was assessed with a recognition test for Item&Location as well as for Item alone.

METHOD

Subjects

Six alcoholic Korsakoff amnesics (all male) and five nonalcoholic, mixed-etiology amnesics (two female, three male) participated in this study, all of whom were outpatients at the Memory Disorders Research Center at the Boston University Medical Center. The Korsakoff amnesics averaged 63.0 years of age at the time of testing and had 11.0 years of education. They had an average Wechsler Adult Intelligence Scale–Revised (WAIS–R) verbal IQ of 94.2. The mixed-etiology amnesics averaged 45.8 years of age and had 16.8 years of education. Two of these patients suffered from anoxia secondary to cardiac arrest. In neither patient did neuroimaging studies reveal focal structural abnormalities; however, available neuropathological data for patients who have sustained anoxia suggests that these patients may have medial temporal lesions (Cummings, Tomiyasu, Read, & Benson, 1984; Zola-Morgan, Squire, & Amaral, 1986). Two patients were post-encephalitic and neuroimaging studies suggested that both had extensive damage to the medial and lateral temporal lobes. The last patient became amnesic following an episode of status epilepticus. MRI scans were consistent with extensive loss of tissue in the left temporal lobe, although bilateral hippocampal damage is possible given recent neuropathological evidence (Victor & Agamanolis, 1990). We suspect that all subjects in this group suffered from hippocampal damage, although because lesions probably involved surrounding cortical areas as well, we refer to this group simply as ‘‘mixed-etiology’’. Their WAIS–R verbal IQ averaged 111.4. Individual IQ and Wechsler Memory Scale–Revised (WMS–R) index scores for both amnesic groups appear in Table 1. None of the amnesic patients had any evidence of colour blindness.

Eighteen alcoholic control subjects (all male) were selected to match the Korsakoff amnesics with respect to age ($M = 60.7$) and education ($M = 13.2$). These subjects were chronic alcoholics living in private homes or in local public halfway houses. None evidenced any signs of neurological or psychiatric illness, and all had abstained from alcohol for at least one month prior to their participation in this experiment. Their WAIS–R verbal IQ averaged 111.1. A total of 18 healthy controls (6 female, 12 male) were also tested. None of these control subjects manifested any signs of neurological or psychiatric conditions. They were selected to match the mixed-etiology amnesics for age ($M = 49.1$) and education ($M = 14.6$). Their average WAIS–R verbal IQ was 106.9. Group means for control subjects are presented in Table 1. None of the control subjects reported any evidence of colour blindness.

TABLE 1
Characteristics of Amnesic and Control Patients

Groups	Age (years)	Education (years)	WAIS–R Verbal IQ	WMS–R Attention	WMS–R General	WMS–R Delay
Korsakoff Amnesics						
AA	66	9	93	109	76	62
LB	59	9	87	93	84	65
PB	67	14	87	93	82	60
RL	51	8	96	83	65	51
GP	72	14	119	110	89	62
RD	63	12	83	99	66	50
Means	63.0	11.0	94.1	97.8	77.0	58.3
S.D.	7.3	2.7	13.0	10.4	9.8	6.3
Alcoholic Controls (N = 18)						
Means	60.7	13.2	111.1	106.3	109.3	112.0
S.D.	7.9	1.7	14.7	13.6	14.5	20.0
Mixed-etiology Amnesics						
DS	30	16	95	120	65	50
DF	43	16	111	107	81	69
PD	56	20	121	90	65	61
SS	65	18	126	114	102	50
PS	35	14	104	115	90	50
Means	45.8	16.8	111.4	109.2	80.6	56.0
S.D.	14.6	2.3	12.5	11.7	16.1	8.7
Normal Controls (N = 18)						
Means	49.1	14.6	106.9			
S.D.	14.6	2.5	14.5			

WAIS–R, Weschler Adult Intelligence Scale–Revised; WMS–R, Wechsler Memory Scale–Revised. The WAIS–R and each of the subtests of the WMS–R have a mean score of 100 with a standard deviation of 15 in the normal population.

Study Materials

We began with a set of 36 drawings of common objects (Snodgrass & Vanderwart, 1980) that occupied 36 locations of a 7×7 grid, and were presented in 36 unique colours. The lines composing the objects were coloured and the interior was white[1]. Colours that were naturally associated with particular objects were avoided (e.g. a leaf might appear in a shade of pink, but not in a shade of green or brown). Although colour was not tested in the current experiment, this feature was present because we used the same materials that

[1] Information about the RGB components defining the colours of the items as well as other parameters can be obtained from the experimenters.

have been used in other work from our laboratory (e.g. Chalfonte & Johnson, 1994, 1996). From these stimuli, three different study arrays were generated for the control subjects, each set consisting of 30 randomly selected items each in a unique location. These pictorial objects were presented in a two-dimensional 19cm $\times$ 19cm grid representing 49 locations in seven rows and seven columns, with objects randomly assigned to locations with the restriction that no more than five objects were placed in any row or column. Because, by necessity, some locations would be occupied multiple times across the three study arrays, items were also repeated across the three arrays to the same degree in order that both features (i.e. item, location) would be treated in an equivalent manner. The items in the three study arrays were matched on Snodgrass and Vanderwart's (1980) name agreement and familiarity norms and there were a similar number of naturally-occurring and man-made objects in the three arrays. Also, the ratio of objects located in the perimeter of the array to objects located more centrally in the array was similar in the three study arrays. An example stimulus array is seen in Fig. 1.

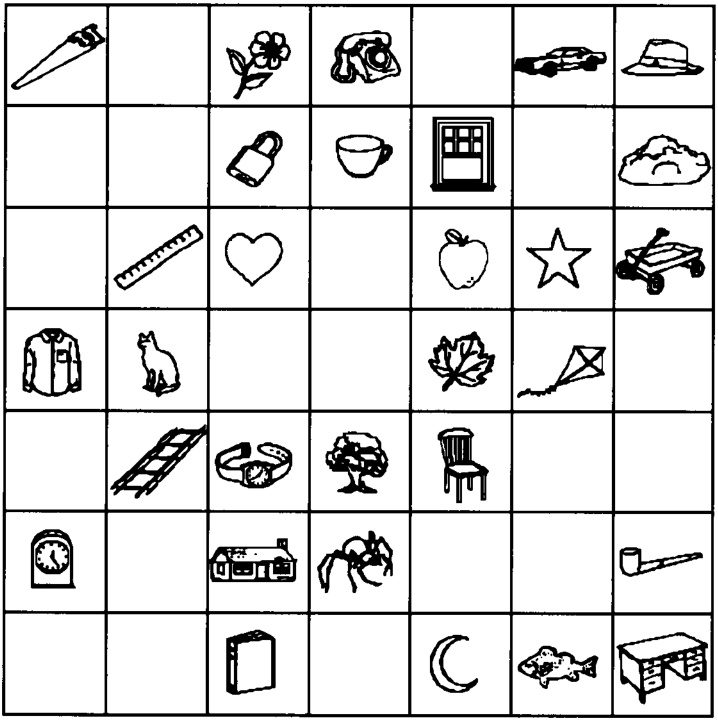

FIG. 1. Example study array, for control subjects, in which 30 uniquely coloured items were located within the 7 $\times$ 7 grid. Black lines composing the items seen here were coloured in the actual array. Amnesic subjects received arrays that were subsets of the control arrays.

All amnesic subjects saw subsets of the stimuli presented to control subjects. We wanted to create versions of the study materials for amnesics in such a way that items and locations (and colours) would be reduced in comparable ways. This was done by reducing the number of items presented and the number of locations filled (and the number of colours seen). At first glance, location might seem to be treated "differently" because we did not also reduce the size of the grid (i.e. from 7×7 to 5×5). However, location differs from item (and colour) because not only are the stimulus locations defined but the non-stimulus locations are defined as well (the blank boxes of the grid). In contrast, stimulus items are defined, but non-stimulus items are not—you do not see items that you need not learn. Because of this difference, we simply reduced the number of to-be-learned stimulus items and locations and did not change the number of not-to-be-learned locations, because we could not do the same for items. Thus, amnesic subjects saw 12 of the 30 objects occupying locations within the 7×7 array that were presented to control subjects. Three such arrays were generated for the amnesic groups. Given that controls saw more items at study (30) than did amnesics (12), using three sets of the study materials ensured that the pairings of items and locations seen by the controls would be seen by the amnesics across the three conditions. The subsets of items presented to the amnesics had the same average name agreement and familiarity norms, and ratio of man-made to natural items, as those presented to controls. Likewise, the ratio of perimeter-located to centrally-located items for amnesics was similar to that for controls.

Test Materials

Examples of the two recognition tests used for control subjects are shown in Fig. 2. For the Item recognition test, 10 items from the array as well as 10 new items were arranged in five rows with four items each (without grid lines)—the items were black and white only. Subjects were instructed to indicate items that had appeared anywhere in the original array. The Item&Location recognition test consisted of a 7×7 array with 10 black and white items from the studied array in their original locations, 5 items from the array in new locations, and 5 new items in previously filled locations. The old items placed in new locations were re-located 3–4 spaces from their original location into previously unfilled locations. Subjects were instructed to indicate an item only if both the item and its location corresponded to the studied array. Three sets of these recognition tests corresponding to the three study arrays were constructed. Amnesic subjects saw subsets of the recognition tests given to controls. For the Item recognition test, there were eight items from the array and eight new items. For the Item&Location recognition test, amnesics saw eight studied items in their original locations, four items from the array in new locations, and four new items in previously filled locations. As for the controls, old items placed in new locations were re-located 3–4 spaces from their original location into previously

(a)

(b)

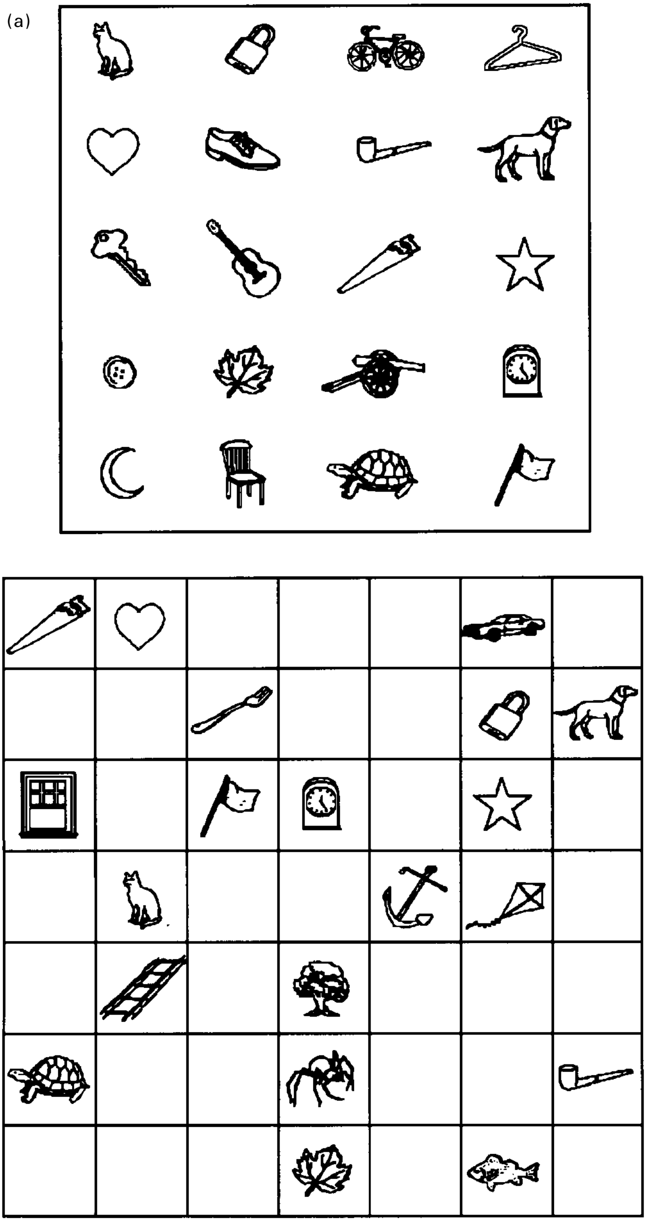

FIG. 2. Example recognition tests for control subjects. (a) Item Only–10 target items and 10 distractor items presented in black and white. (b) Item&Location–10 targets of old items in their original locations; 5 distractors of old items in new locations; 5 distractors of new items in previously filled locations; all items were presented in black and white. Amnesic subjects received recognition tests that were subsets of the tests given to controls.

unfilled locations. Again, there were three sets of recognition tests generated, one for each study set.

In order to assess subjects' ability to perceptually discriminate between items and locations independent of any memory demands, a matching task was constructed for both features. A subset of 10 target items and locations were selected from those used in the study/recognition test materials. Corresponding distractor features, also selected from the study/test materials, ranged in their similarity to the target features from very similar to very dissimilar. Both a target feature page and a two-alternative choice page were in view simultaneously. Thus, item matching was tested by showing subjects one item in black and white centred on the target page and asking them to choose between the same item paired with a distractor item of varying similarity. Items and their distractors ranged from very similar in shape (e.g. ball and apple) or concept (e.g. star and moon) to very dissimilar in shape (e.g. tree and kite) or concept (e.g. pipe and spider). Location matching was tested by showing subjects a target ''X'' placed within one location of a 7×7 array and asking them to choose between an ''X'' located in the same place and a distractor ''X'' in varying proximity. The target and distractors ranged from very close in proximity (e.g. in adjacent squares of the grid) to very far (e.g. seven squares apart in the grid). Twenty item and location matching trials were randomly intermixed.

Design and Procedure

Both control and amnesic subjects of both sub-types, alcoholic Korsakoff and mixed-etiology, were tested in each of the three study/test conditions: study Item/test Item; study Item/test Item&Location (incidental location); and study Item&Location/test Item&Location (intentional location). This yielded a 2 (subject) $\times$ 3 (study/test condition) mixed design for each amnesia sub-group. Amnesic patients were all tested in the order (1) study Item/test Item, (2) study Item/test Item&Location (incidental location), and (3) study Item&Location/test Item&Location (intentional location). This was done to prevent subjects from studying aspects of the array additional to what they were instructed to study. Of the 18 control subjects, six were tested in the same order as the amnesic patients, completing the item condition first. Six more controls were tested in the incidental location condition first, and six other controls were tested in the intentional location condition first. This ordering was used because, for control subjects but not amnesic subjects, there was some overlap between the items seen in the three study/test conditions. Thus, having some control subjects complete each condition first allowed us to determine whether there were any effects of item repetition on performance. No significant effects were noted however, thus results are reported here collapsed across order of testing.

For each condition, subjects were told which feature(s) to study and then were presented with the study array. Subjects in the Item Only condition and in the incidental location condition were directed to study the items only. Subjects in the intentional location condition were directed to study both the items and their locations. Control subjects were given one 90-second acquisition phase. To equate memory for item information, in addition to reducing the study array, amnesic patients were given two 90-second acquisition phases, separated by a two-minute interval in which they conversed with the experimenter. Immediately following the acquisition phase(s), subjects completed the recognition test. The interval between each of the study/test conditions was at least one day for all subjects and was longer than seven days for most subjects. After subjects completed the three study/test conditions, they were given the matching task to evaluate their ability to perceptually identify specific item and location features independent of any memory demands.

RESULTS

The dependent variable computed for each subject was a corrected recognition score (proportion hits minus proportion false alarms) in each study/test condition. All mean performance values and standard errors for the groups are seen in Figs. 3 and 4. For all statistical analyses reported here, the significance level was set at 0.05 unless otherwise specified.

For Korsakoff amnesics and alcoholic controls (Fig. 3), a 2 (subject type) $\times$ 3 (condition) ANOVA showed no main effect of subject type, $F(1,22) = 1.10$, $MS_e = 0.12$, a main effect of condition, $F(2,44) = 12.34$, $MS_e = 0.04$, and no interaction, $F(2,44) < 1$. The main effect of condition reflects the fact that both Korsakoff amnesics and controls performed better in the study Item/test Item condition than in the two location conditions. Planned comparisons for each condition separately showed that by reducing the study array and increasing acquisition time, item recognition memory was successfully equated for Korsakoff patients and alcoholic controls, $F(1,44) = 1.45$, $MS_e = 0.04$. Under these study conditions yielding equated item memory, we found that Korsakoff amnesics showed no evidence of disproportionately impaired recognition memory for incidentally-encoded, $F(1,44) = 2.17$, $MS_e = 0.04$, or intentionally-encoded location, $F(1,44) < 1$. Moreover, Korsakoff amnesics' memory for item and location did not benefit by intentionally studying location relative to incidental acquisition, $F(1,44) < 1$. There was also no difference between alcoholic controls' incidental and intentional location acquisition, $F(1,44) = 2.23$, $MS_e = 0.04$.

Korsakoff amnesics and alcoholic controls were equated for age and education, but the verbal IQ of the controls (M = 111.1) was higher than that of the amnesics (M = 94.1), $F(1,22) = 7.84$, $MS_e = 191.7$, $P < 0.05$. Therefore, we selected a subset of six alcoholic controls that matched the Korsakoff patients on verbal IQ (M = 97.8, SD = 9.9) as well as age (M = 61.8, SD = 6.0) and years of education (M = 13.0, SD = 1.7). The pattern of recognition performance was the same for

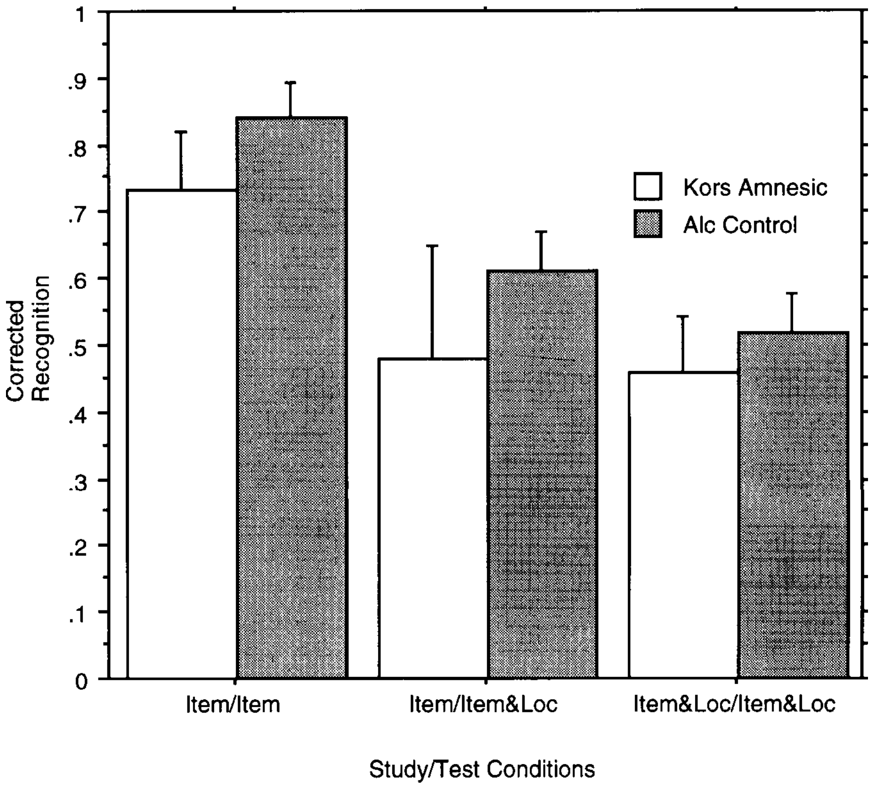

FIG. 3. Mean corrected recognition performance and standard errors for Korsakoff amnesics and alcoholic controls.

these amnesic and matched control subjects as for the complete groups: a 2 (subject type) × 3 (condition) ANOVA showed that there was no reliable effect of subject type, $F(1,22) < 1$, but there was a reliable effect of condition, $F(2,20) = 5.05$, $MS_e = 0.05$. The interaction did not reach significance, $F(2,20) < 1$. Korsakoff patients and their matched controls not only had equivalent item recognition performance ($M_{Kors} = 0.73$, $SE = 0.09$; $M_{Ctrls} = 0.72$, $SE = 0.15$), but incidental location ($M_{Kors} = 0.48$, $SE = 0.17$; $M_{Ctrls} = 0.53$, $SE = 0.12$) and intentional location recognition performance ($M_{Kors} = 0.46$, $SE = 0.08$; $M_{Ctrls} = 0.42$, $SE = 0.10$) also did not differ. Likewise, there was no effect of incidental versus intentional acquisition of location for these matched groups.

For mixed-etiology amnesics and normal controls (Fig. 4), a 2 (subject type) × 3 (condition) ANOVA showed a main effect of subject type that approached significance, $F(1,21) = 3.35$, $MS_e = 0.10$, $P = 0.08$, a reliable main effect of condition, $F(2,42) = 10.02$, $MS_e = 0.03$, as well as a significant

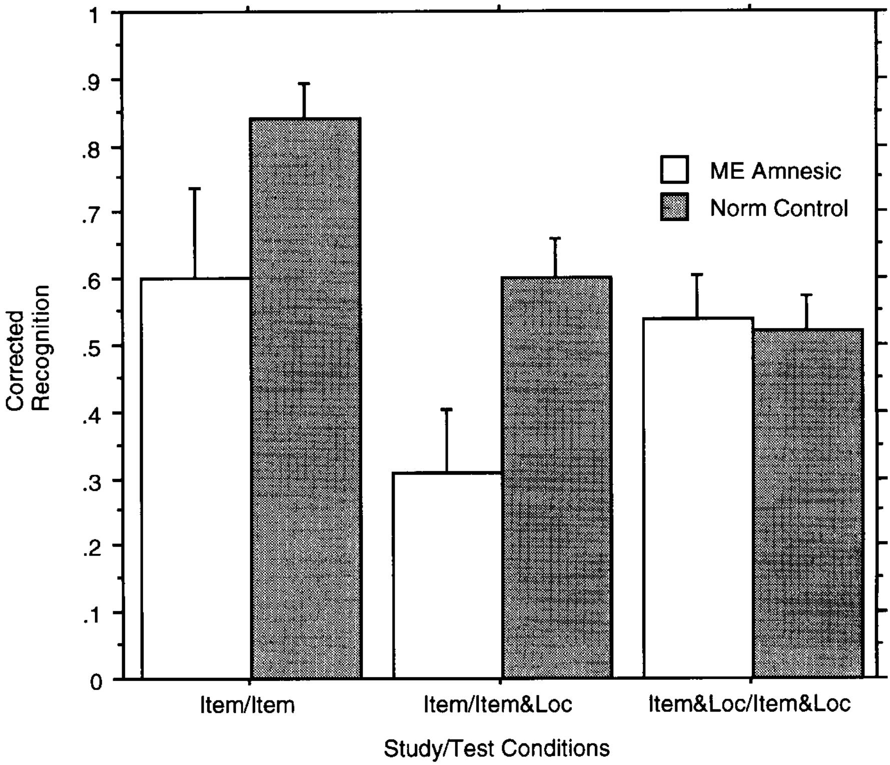

FIG. 4. Mean corrected recognition performance and standard errors for nonalcoholic mixed-etiology amnesics and normal controls.

interaction, $F(2,42) = 3.57$, $MS_e = 0.03$. Like the other amnesic and control groups, these subjects showed better recognition memory in the study Item/test Item condition relative to the two location conditions. Moreover, control subjects generally outperformed the mixed-etiology patients except in the intentional location condition, as reflected in the interaction. Planned comparisons for each condition separately showed that reducing the study array and increasing study time did not successfully equate mixed-etiology patients' and normal controls' item memory, $F(1,42) = 7.38$, $MS_e = 0.03$. In addition to impaired item memory, amnesic patients showed impaired recognition memory for incidentally-encoded, $F(1,42) = 10.78$, $MS_e = 0.03$, but not intentionally-encoded location recognition memory, $F(1,42) < 1$. Unlike Korsakoff patients, mixed-etiology amnesics did benefit significantly from intentionally studying location relative to incidental acquisition, $F(1,42) = 6.78$, $MS_e = 0.03$. For normal controls, performance in the incidental and intentional location acquisition conditions did not differ, $F(1,42) < 1$.

Given that mixed-etiology amnesics and normal controls were not equated on item recognition performance, we selected a subset of five controls who matched the amnesics on item recognition performance (M = 0.64, SE = 0.15), age (M = 45.8, SD = 10.4), and years of education (M = 13.2, SD = 1.1) to better evaluate the possibility of disproportionately impaired location memory. When item recognition was equated, the pattern of performance remained the same as in the analysis including all subjects: mixed-etiology patients tended to be impaired in their incidental acquisition of location information, $F(1,16) = 3.07$, $MS_e = 0.05$, $P < 0.10$, and they continued to benefit by encoding location intentionally rather than incidentally, $F(1,16) = 5.63$, $MS_e = 0.05$. Normal controls did not show an effect of encoding instructions, $F(1,16) < 1$.

Recognition Responses

Subjects were not required to make a specific number of recognition responses, and if amnesic patients were more cautious making recognition responses than control subjects, then this could have contributed to their poorer performance. Of course, amnesics did not perform more poorly in all conditions; only the mixed-etiology amnesics performed worse than their controls in the Item condition and in the incidental location condition. Thus, evidence of an interaction between subject type and condition would be necessary in the analysis of the mixed-etiology group to support any notion of response cautiousness accounting for the reported performance in location memory. To compute recognition responses, we took the number of targets and distractors selected and divided it by the total number of targets and distractors available for each subject type (16 possible responses for amnesics; 20 possible responses for controls).

Using a 2 (subject type) × 3 (condition) repeated measures ANOVA, for Korsakoff patients and alcoholic controls, there was no main effect of subject type, $F(1,22) < 1$. There was a main effect of condition, $F(2,44) = 7.10$, $MS_e = 0.01$, where subjects made more responses on the Item recognition test (M = 0.47, SE = 0.02) than on either Item&Location test (incidental: M = 0.42, SE = 0.03; intentional: M = 0.37, SE = 0.03). There was no interaction between subject type and condition, $F(2,44) = 2.44$, $MS_e = 0.01$.

Similarly, for mixed-etiology patients and normal controls, there was no main effect of subject type, $F(1,21) < 1$, but there was a significant effect of condition, $F(2,42) = 3.67$, $MS_e = 0.01$, again with more responses being made on the Item recognition test (M = 0.46, SE = 0.02) than on either Item&Location test (incidental: M = 0.39, SE = 0.02; intentional: M = 0.39, SE = 0.02). There was no interaction, $F(2,42) < 1$. In short, these results suggest that willingness to respond does not account for differences in location memory performance between amnesic and control subjects.

Matching Task

Subjects' ability to discriminate between different items and locations independent of any memory demands was also assessed. For Korsakoff amnesic and alcoholic control subjects, a 2 (subject type) $\times$ 2 (feature type) repeated measures ANOVA showed an effect of subject type that approached significance, $F(1,22) = 3.30$, $MS_e = 0.07$, $P < 0.09$, but no effect of feature type or interaction, $F(1,22) < 1$. The difference in performance between the Korsakoff amnesics and the controls is due to one Korsakoff patient mismatching one item (of ten) and one location (of ten). All other amnesics and controls achieved 100% matching performance. All mixed-etiology and normal controls also achieved 100% matching performance for both the items and the locations. Given the very high level of matching proficiency of all subjects for both features, there is no evidence that an inability to discriminate item or location features significantly influenced the encoding or recognition of these features.

Correlation Analyses

We considered factors, other than amnesia sub-type, that may have accounted for the different patterns of location memory in the Korsakoff and mixed-etiology amnesics. In the following correlation analyses across the amnesia sub-types, we used performance in the incidental location condition as well as the difference between performance in the intentional and incidental conditions, because these measures best highlighted the different patterns of location memory for the two amnesic groups. First, correlations between these location measures and a measure of amnesia density (difference between WMS–R attention and general indices) were not reliable (Ps > 0.30), arguing against the notion that increasingly severe amnesia is associated with disproportionately poorer location memory. Second, we correlated location memory performance with age because our previous work showed that older adults, relative to college-aged subjects, have impaired memory for locations (Chalfonte & Johnson, 1994, 1996). The correlations between age and location memory were not reliable (Ps > 0.30). Finally, one possible difference between Korsakoff and mixed-etiology amnesics is the often-reported contribution of frontal lobe damage to alcoholic Korsakoff amnesia. Indeed, the Korsakoff amnesics tested in the current experiment performed more poorly than mixed-etiology amnesics on frontal tests (verbal fluency, Wisconsin card sort, Trails B) as measured by a composite performance score consisting of subjects' average ranking on the three tests, $F(1,9) = 18.63$, $MS_e = 3.16$, $P < 0.005$ (Korsakoff: M = 4.42, SE = 0.77; mixed-etiology: M = 9.06, SE = 0.74, where higher scores indicate less impairment on the frontal tasks). This measure of frontal impairment, however, was not significantly correlated with location memory measures (Ps > 0.30), and in fact, contrary to what might be expected, location memory performance was typically better in subjects with more frontal impairment (i.e. Korsakoff patients) than in subjects with less frontal impairment (i.e. mixed-etiology amnesics).

DISCUSSION

Recognition memory for non-spatial information (i.e. item information) was equated between amnesic groups and their controls in the current study. Under these circumstances, mixed-etiology amnesics tended to demonstrate disproportionately impaired memory for the spatial location of items. The location deficit in mixed-etiology amnesics, however, was apparent only when location information was acquired incidentally. Korsakoff patients, in contrast, showed no evidence of disproportionately disrupted location memory relative to alcoholic controls, regardless of whether spatial location was encoded incidentally or intentionally. The differences in the patterns of location memory performance between amnesics and controls and between the two amnesic groups was not due to differences in willingness to make recognition responses or in the ability to distinguish perceptually among items or locations. Moreover, the location recognition performance of the amnesics was not correlated with subject characteristics such as amnesia density, age, or frontal involvement.

Disproportionate Disruption of Spatial Location Memory

Our initial review of prior work suggested that memory for spatial location may be disproportionately impaired in human amnesia. That is, under conditions that are sufficient to equate amnesics' and controls' memory for non-spatial information, amnesic patients' memory for spatial information is more impaired than that of controls. Closer examination of earlier findings in light of the present results suggest that this may be true only for a subset of amnesic patients. Our data suggest that mixed-etiology amnesics, with the temporal lobe as the probable locus of damage, may have disproportionately impaired spatial location memory, whereas Korsakoff amnesics do not. Cave and Squire's (1991) data suggest a similar pattern: their hippocampal amnesics performed numerically poorer on a spatial location task than did their diencephalic amnesics when the non-spatial performance of both groups was equated with that of control subjects. Mayes et al. (1991) found that their Korsakoff patients tended to be less impaired than their patients who were amnesic due to anterior communicating artery aneurysms, and performed numerically better than their post-encephalitic patients. MacAndrew and Jones (1993) reported that Korsakoff patients performed no worse than controls on a leniently scored location memory task. We note, however, that Shoqeirat and Mayes (1991) found no reliable location memory differences between their amnesic sub-groups. Nevertheless, taken together, these studies suggest that memory for spatial location is disproportionately disrupted for some amnesia sub-types but not others. Our data are consistent with the conclusion that memory for spatial location is more likely to be disproportionately impaired in amnesics with disruption of temporal lobe structures than in amnesics with disruption of diencephalic regions.

Intention and Location Memory

The intention-to-remember spatial locations did not improve location memory for our Korsakoff patients. This is consistent with the findings of MacAndrew and Jones (1993) and Kovner et al. (1988). Likewise, Smith (1988) found that intentional instructions did not improve HM's location memory performance and Kovner et al. (1988) found that intentional instructions did not help mixed-etiology amnesics. In contrast, our data indicated that mixed-etiology amnesics' recognition memory for spatial location was better when the information was intentionally acquired than when it was incidentally acquired. This result is consistent with Hirst and Volpe's (1984a) similar findings for mixed-etiology amnesics. Thus, our data in combination with previous findings suggest that effects of intention-to-remember may represent another dimension, like disproportionately impaired location memory, on which amnesia sub-groups differ. Intention-to-remember spatial locations may not improve spatial location memory for Korsakoff amnesics, but may improve (at least under some conditions) spatial location memory for mixed-etiology amnesics.

Spatial Location Memory and Intention in Accounts of Global Amnesia

One account of human amnesia in which memory for spatial location plays a central role is Mayes' (1992; Mayes, Downes, Shoqierat, Hall, & Sagar, 1993) contextual memory deficit hypothesis (CMDH). According to Mayes' CMDH, amnesics suffer from a primary deficit in contextual information processing; relative to target information, memory for contextual information (e.g. location, colour, size) is disproportionately impaired. Mayes (1992) further argues that it is the type of information (i.e. contextual) and not the type of processing that is important, so that directing amnesic subjects to intentionally encode contextual information should not improve their performance. The data from the mixed-etiology amnesics in the current experiment contradict the latter processing prediction from the CMDH. With regard to the former prediction, it remains to be determined whether memory for types of contextual information other than location (e.g. colour, size) is also disproportionately disrupted as a consequence of various amnesia etiologies. Mayes et al. (1993) recently found that amnesics had poorer memory for the colour and size, as well as the location, of objects relative to controls, but whether memory for any of these contextual features was *disproportionately* poorer than memory for the objects themselves was not established.

In another account of human amnesia, the intention-to-remember plays a central role. To explain the processing deficits observed in amnesia, Hirst's (1982; Hirst & Volpe, 1984a) automatic encoding deficit account suggested that encoding may require more effort for amnesics than for normal individuals. Intact adults may be able to encode some contextual information automatically (Hasher & Zacks, 1979), but amnesics may have to encode this same information effortfully. If processing resources are limited and amnesics expend resources encoding information that is usually encoded automatically, then amnesic patients must experience some trade-off between remembering item information and remembering location information. According to Hirst, this would lead to overall poorer memory for the item and its location. Given multiple presentations and fewer items to remember, mixed-etiology amnesics in the present study were able to remember the objects, but their incidental location memory was impaired. They were able to overcome this deficit, however, with intentional encoding instructions. This finding is more consistent with the notion that mixed-etiology amnesics have a deficit in the allocation of processing resources, rather than a deficit in the availability of resources *per se*. Whatever the ultimate value of Hirst's disrupted automatic processing account, it does not hold for all amnesics, because our Korsakoff patients were not especially disadvantaged in the incidental location condition.

Insofar as these (Mayes' and Hirst's) accounts do not entirely capture the data presented here, how might we understand the pattern of results we obtained? First consider that complex memory, like memory for the location of a particular item, requires memory for location itself as well as cognitive processes for binding the location and the item information together. Binding is what provides the memorial experience that features ''belong together''. If both location information alone and binding processes are important for intact item and location memory, then the deficit in the incidental encoding condition observed in mixed-etiology amnesics could therefore result from either a memory deficit for location information specifically or from a deficit in binding. Two findings suggest that the primary deficit may be in the processes important for binding. First, Shoqeirat and Mayes (1991) tested amnesics' and controls' memory for location only. Memory for location only was considered correct when some object was placed into a previously filled location, regardless of which item was placed into the particular location. They reported that this kind of memory for spatial location, which we call location feature memory, was much less impaired than memory for the locations of specific objects, which we refer to as memory for bound item and location. Second, we have found that when location feature memory is impaired (in older adults), intentional encoding does not improve recognition of item and location as was seen here with the mixed-etiology amnesics (Chalfonte & Johnson, 1994). Consequently, we suspect that the deficit observed in mixed-etiology amnesics reflects an impairment in incidental binding of item and location into complex memories, beyond any location feature deficit they may have (cf Ungerleider & Mishkin, 1982).

Next, consider the additional deficit that mixed-etiology amnesics showed on item memory. Old/new item recognition presumably relies on memory for item information bound to environmental context, including item to environmental location (e.g. Anderson & Bower, 1974). Mixed-etiology amnesics' item recognition memory remained poor despite attempts to equate their performance with controls by increasing exposure time and by reducing the number of to-be-learned stimuli. Mixed-etiology amnesics' inability to bind item and location information incidentally, likely contributes to their poor item recognition performance as well. It is also the case, without efforts to equate amnesic and control performance, that Korsakoff amnesics as well as mixed-etiology amnesics have profound deficits on recognition memory in general; they require more or longer exposures, or shorter retention intervals to even begin to approximate the performance of controls. Thus not only hippocampal but also diencephalic regions appear to be part of a circuit crucial for binding aspects of experience into complex memories (cf Eichenbaum & Bunsey, 1995; Johnson & Chalfonte, 1994). Perhaps this ''binding circuit'' is differentially recruited in conjunction with relevant cortical areas depending on the features being processed (e.g. location, size, colour). If so, then lesions to particular areas of this circuit would be more likely to disrupt binding of some features than others. Lesions to hippocampal regions, for example, would be more likely to disrupt item and location binding than lesions to the diencephalic area as suggested by the present results (cf Nadel, 1992; O'Keefe & Nadel, 1978). Moreover, lesions to the hippocampal region may also be more likely to disrupt item and location binding than say, item and colour binding. This latter possibility remains to be tested. That is, an important question is whether the disproportionate incidental binding deficit found for mixed-etiology amnesics is general to all information or is specific to location. Furthermore, it should be noted that damage outside the hippocampal area may also contribute to the deficits that mixed-etiology amnesics exhibit. Therefore, another central question is whether lesions to areas other than the hippocampus, especially lesions to other areas of this presumed binding circuit, disproportionately disrupt the binding of spatial and/or other types of information.

Finally consider that mixed-etiology amnesics benefited from intentional encoding of item and location information whereas controls (and Korsakoff patients) did not. This evidence is consistent with other studies finding that neurologically intact adults do not score higher on recognition tests of item and location after intentional encoding relative to incidental encoding (Chalfonte & Johnson, submitted). Thus the additional processing engaged by intentional instructions does not produce representations of item and location information that are superior for recognition to those yielded by incidental processing[2]. Evidently, for mixed-etiology amnesics, we can see the benefits from

[2] In controls, intentionally generated representations may, however, be superior under other testing conditions (e.g. cued-recall, Chalfonte & Johnson, 1994).

intentionally generated representations because their incidentally derived representations of item and location information are so impoverished.

SUMMARY

We assessed whether memory for spatial location was disproportionately impaired in Korsakoff patients and mixed-etiology amnesics, and whether amnesics' spatial location memory benefited from intentional encoding relative to incidental acquisition. To do this, we equated item recognition memory by increasing the number of times that amnesics saw the study materials and by decreasing the number of items and locations that they had to learn, and then tested spatial memory via recognition. We found evidence for disproportionately disrupted spatial location memory in one amnesia sub-type—mixed-etiology, bitemporal amnesia—but not in another—Korsakoff amnesia. This finding helps resolve discrepancies in the literature about whether or not spatial location memory is indeed disproportionately disrupted in amnesia. We also found that intentional, relative to incidental, encoding instructions improved item and location recognition memory, again in one amnesia sub-type—mixed-etiology, bitemporal amnesia—and not in another—Korsakoff amnesia. Although several other researchers have failed to find this pattern, we note that the type of test used to evaluate spatial location memory may be important; other researchers have used cued-recall tests whereas we used recognition tests. Finally, we suggest that binding is generally disrupted in amnesia as a consequence of lesions in a ''binding circuit'' involving hippocampal and diencephalic regions, and that hippocampal damage in mixed-etiology amnesics may disproportionately disrupt item and location binding.

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