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Memory and executive functions in amnesic and non-amnesic patients with aneurysms of the anterior communicating artery

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Summary

Ruptured and repaired anterior communicating artery (ACoA) aneurysm can result in devastating impairments involving memory, executive function, confabulation and personality change. Importantly, traditional cerebral areas implicated in amnesia are not damaged, yet amnesia can still be manifested. While ACoA patients show normal visual–constructional skills (i.e. copy scores) on the Rey–Osterrieth complex figure test, recall is often impaired. What is unclear is whether impaired recall is attributable to problems in encoding, accelerated rates of forgetting, retrieval or some combination. To disentangle these issues, we examined 10 patients with ruptured aneurysms of the ACoA, using the Rey-organizational and extended memory procedure which uses an organizational procedure for enhancing immediate recall and provides added sensitivity by combining recall with non-recall measures (e.g. recognition, spatial discrimination and spatial assembly). The major findings were: (i) immediate recall in amnesics was improved by providing an organizational strategy; (ii) following the organization trials, amnesics and non-amnesics retained information to a comparable extent over a 30-min delay; (iii) two subgroups of amnesics emerged, those subjects impaired in acquisition and a second group with impaired retrieval; (iv) all subjects showed preserved memory on non-recall measures. These findings have important implications with respect to using organizational strategies in cognitive treatments and in using non-recall measures in improving the validity and reliability of patient assessment.

Keywords: anterior communicating artery; amnesia; executive; complex figure; basal forebrain

Abbreviations: ACoA = anterior communicating artery; ANOVA = analysis of variance; CVLT = California verbal learning test; ROCFT = Rey–Osterrieth complex figure test; R-OEM = Rey-organizational and extended memory test; WAIS-R = Wechsler adult intelligence scale—revised; WCST = Wisconsin card sort test

Introduction

Neurobehavioural deficits commonly observed following anterior communicating artery (ACoA) aneurysm include impaired memory (Alexander and Freedman, 1984), confabulation (DeLuca and Cicerone, 1991), personality change (Steinman and Bigler, 1986) and impaired executive function (DeLuca and Diamond, 1995). While brain imaging data and surgical reports have often confirmed the presence of basal forebrain damage following ACoA aneurysms, it should be noted that lesion location and size can be quite variable following ACoA aneurysm rupture (Irle *et al*., 1992; DeLuca and Diamond, 1995). Interestingly, the traditional cerebral areas implicated in amnesia (i.e. mesial temporal and diencephalic structures), are not damaged, yet amnesia can still be manifested (for a review, *see* DeLuca and Diamond, 1995). Damage to the basal forebrain is thought to underlie the amnesia observed in a subset of ACoA patients (Damasio *et al*., 1985; DeLuca, 1993).

The Rey–Osterrieth complex figure test (ROCFT) (Rey,

1941; Osterreith, 1944) which has been used as a measure of visual memory, planning and organizational skills, and visual–spatial constructional performance in a wide spectrum of brain-damaged and elderly populations (Loring *et al*., 1988; Berry *et al*., 1991; Grossman *et al*., 1993; Janowsky *et al*., 1993), has also been used with ACoA patients (Phillips *et al*., 1987; DeLuca and Cicerone, 1991; Diamond and DeLuca, 1996). Most of the studies with ACoA patients using the ROCFT show performance on the copy trial to be within normal limits. However, recall is often impaired. For instance, Phillips *et al*. (1987) reported a patient who had no recollection of the design after a 30 min delay. Damasio *et al*. (1985) reported that three out of four of their amnesic patients with basal forebrain lesions showed impaired performance on the 3 min delayed recall condition. However, not all ACoA patients show impaired recall on the ROCFT (e.g. Teissier du Cros *et al*., 1984). More recently, Diamond and DeLuca (1996) found that despite copy scores that were within normal limits, amnesic ACoA subjects displayed a profound loss of information between the copy and immediate recall conditions on the ROCFT while displaying no significant loss of information between immediate and delayed recall.

Importantly, what is not clear from these studies of visual memory using the ROCFT is whether impaired recall is attributable to inadequate encoding, accelerated rates of forgetting, retrieval failure or a combination of these impairments. For instance, the dramatic drop in immediate recall on the ROCFT may be due to a rapid rate of forgetting (Diamond and DeLuca, 1996). However, it is also possible that ACoA subjects fail to encode the information adequately during the copy trial, perhaps because of poor integration and organization of the complex figure, secondary to executive dysfunction (a common deficit in ACoA patients; *see* DeLuca and Diamond, 1995). Relationships between deficits in patients' organizational and strategic processing and frontal impairments, particularly with respect to performance on tests of free recall, have been documented (Incisa della Rocchetta and Milner, 1986; Jetter *et al*., 1986; Janowsky *et al*., 1989; Incisa della Rocchetta and Milner, 1993; Stuss and Alexander, 1994). Lastly, it is possible that ACoA patients may achieve adequate encoding and consolidation of the figure during the copy trial, but may be unable to retrieve the stored information. In fact, frontal impairments may also cause organizational problems at retrieval and thus by providing cues at testing, recall performance may be enhanced (Jetter *et al*., 1986; Incisa della Rocchetta and Milner, 1993; Gershberg and Shimamura, 1995).

A major goal of the present study was to employ a procedure which allowed us to address whether impaired visual memory in ACoA subjects on the ROCFT is due to inadequate encoding, accelerated rates of forgetting or retrieval failure. Because of methodological limitations, the traditional administration of the ROCFT does not allow for the determination of the specific mechanism(s) responsible for impaired memory. That is, the use of recall measures alone may not adequately reflect the total amount of information that has been encoded (Hanley *et al*., 1994; Meyers and Lange, 1994). Moreover, the standard ROCFT procedures are not sensitive in evaluating rates of forgetting (between immediate and delayed recall) because the ROCFT protocol contains no mechanism for equating subjects on immediate recall. The purpose of the present study was to employ a paradigm to address specifically whether impairments on the ROCFT are due to problems in encoding, consolidation or retrieval. This was accomplished by using the Rey-organizational and extended memory (R-OEM) procedure.

Specifically, the R-OEM protocol first assesses visual–spatial and motor performance (i.e. normal ROCFT copy condition) and is then followed by an immediate recall condition. If criterion is not achieved on immediate recall, the R-OEM uses an organizational procedure for enhancing encoding thereby providing a mechanism for attempting to equate subjects on immediate recall. This is an important prerequisite for accurately measuring rates of forgetting and for addressing the issue of scaling effects (i.e. the inability to measure rates of forgetting if little information has been acquired; *see* Mayes, 1988). If amnesic patients exhibit improved immediate recall after the R-OEM procedure, this would suggest that the procedure was effective in improving encoding. Moreover, if patients show little to no loss of acquired information over a 30 min delay, this would argue against an accelerated rate of forgetting and would suggest adequate consolidation of information. On the other hand, if impaired recall in subsets of ACoA patients is due to a rapid rate of forgetting, delayed recall in the ACoA group should be impaired relative to controls, even after matching for immediate recall. However, impaired delayed recall (after matching for encoding) may suggest either rapid forgetting or impaired retrieval. In order to disentangle these mechanisms a series of complex recognition, discrimination and spatial assembly trials were administered. Preserved performance on these measures lend additional support for the idea of impaired retrieval.

Methods

Subjects

Subjects consisted of six amnesic ACoA patients; three non-amnesic ACoA patients, and one non-amnesic patient with an anterior cerebral artery aneurysm. All of the patients had ruptured and repaired cerebral aneurysms. The time (mean ± SD) between surgery and testing was 28.6 ± 23 months. (Unless otherwise stated, all data are given as mean ± SD.) The time between surgery and testing in the amnesic (30 ± 25 months) and non-amnesic (23 ± 20 months) groups did not differ statistically [$t(8) = 0.47$, $P = 0.64$]. The amnesic and non-amnesic groups did not differ with respect to age, intelligence (verbal and performance IQs) or education. The non-amnesic group

Table 1 *Lesion sites and demographic data*

Subject	Age (years)	Group	Aetiology	CT Scan
B.C.	42	Non-amnesic	ACoA	Bilateral frontal infarcts (R > L)
I.L.	75	Non-facilitated Amnesic	ACoA	L frontal contusion, slight effacement of L lateral ventricle, focal lucency in supracellar region
O.J.	47	Facilitated Amnesic	ACoA	L frontal encephalomalacia
S.R.	62	Non-amnesic	ACoA	R frontal infarct
M.C.	32	Facilitated Amnesic	ACoA	L frontal and L anterior basal ganglia infarcts, with mild L to R midline shift
W.A.	57	Facilitated Amnesic	ACoA	R frontal infarct and L basal ganglia
A.M.	45	Non-amnesic	ACA	Encephalomalacia in R inferior frontal area, infarct in distribution of L ACA
M.J.	61	Non-facilitated Amnesic	ACoA	Zone of focal lucency adjacent, and superior, to the suprasellar region consistent with R frontal infarct.
H.G.	30	Non-facilitated Amnesic	ACoA	Radiolucency over the medial aspect of the right frontal lobe and the left basal ganglia, most likely representing a small infarct.
P.K.	40	Non-amnesic	ACoA	CT not available

ACoA = anterior communicating artery; ACA = anterior cerebral artery; R = right; L = left.

had a mean age of 47.2 ± 10 years, and attained mean verbal and performance IQs of 106.6 ± 18.6 and 97 ± 11, respectively, and a mean education level of 14.6 ± 2.3 years. The amnesic group's mean age was 50.3 ± 17.4; they attained mean verbal and performance IQs of 108.6 ± 7.1 and 94 ± 16, respectively, and a mean education level of 15 ± 1.4 years. Table 1 presents CT scan information for the ACoA subjects. All subjects had given informed consent to participate in the study, which was approved by the Kessler Institute Internal Review Board.

In previous studies with amnesics, the degree of memory impairment has been classified on the basis of verbal memory performance in relation to intellectual performance, i.e. MQ–IQ (memory quotient–IQ). This approach has been criticized in terms of measurement sensitivity and for its failure to take into account the heterogeneity of patient populations (*see* Mayes, 1988). Thus, for the purposes of this study, amnesia was defined as: (i) showing preserved intelligence and normal digit span; (ii) performing at ⩾3 SD below the mean on the short-delay free recall measure on the California verbal learning test (CVLT) and (iii) not reaching criterion (50th percentile performance, according to Lezak, 1995) on immediate recall of the ROCFT (i.e. scores of ⩽21). Performance on Digit Span was within normal limits in all ACoA subjects (mean, 13.7 ± 3.4). Table 2 presents the data for the ACoA subjects on the standardized neuropsychological instruments.

Materials and procedure

All subjects were administered the following standardized neuropsychological tests: the Wechsler adult intelligence scale-revised (WAIS-R; Wechsler, 1981) as a measure of intelligence, the CVLT (Delis *et al*., 1987) to assess verbal memory; the Wisconsin card sort test (WCST; Heaton, 1981) as a measure of executive function and the ROCFT as a measure of non-verbal memory.

The R-OEM was administered in four stages. In the first stage of the assessment, subjects were administered the ROCFT in the standard manner (Lezak, 1995). That is, following the copy trial, subjects were asked to reproduce the figure from memory (i.e. immediate recall condition). If performance on immediate recall fell below the criterion score (i.e. mean ⩽ 21, *see* Lezak, 1995) the 'organization procedure' was initiated (i.e. stage 2). The purpose of the organizational procedure was to try to improve immediate recall by providing the subjects with an organizational structure in order to enhance learning of the figure and, therefore, to help subjects to match the immediate recall performance of healthy controls.

Organization procedure

The organization procedure consisted of dividing the figure into five subunits, each of which was displayed sequentially on transparent acetate sheets (*see* Fig. 1 for Subunits 1–5).

Table 2 *Attention, executive function and verbal memory scores*

	Digit span	WCST-C	WCST-P	CVLT-SDF
Amnesic	16.5 ± 6.2	2.6 ± 1.6	54.8 ± 18.8	−4.0 ± 0.8
Non–amnesic	15.5 ± 4.4	3.5 ± 1.7	59.0 ± 30.3	−2.2 ± 2.2
Facilitated	19.0 ± 8.5	4.0 ± 1.0	54.6 ± 28.0	−4.3 ± 0.5
Non-facilitated	14.0 ± 2.6	1.3 ± 0.50	55.0 ± 10.1	−3.6 ± 1.1

WCST = Wisconsin card sort test; C = categories; P = perseverative responses; CVLT-SDF = California verbal learning test-short delayed free recall. All the measures represent raw score mean values (±SD) with the exception of the CVLT measures which represent standard scores.

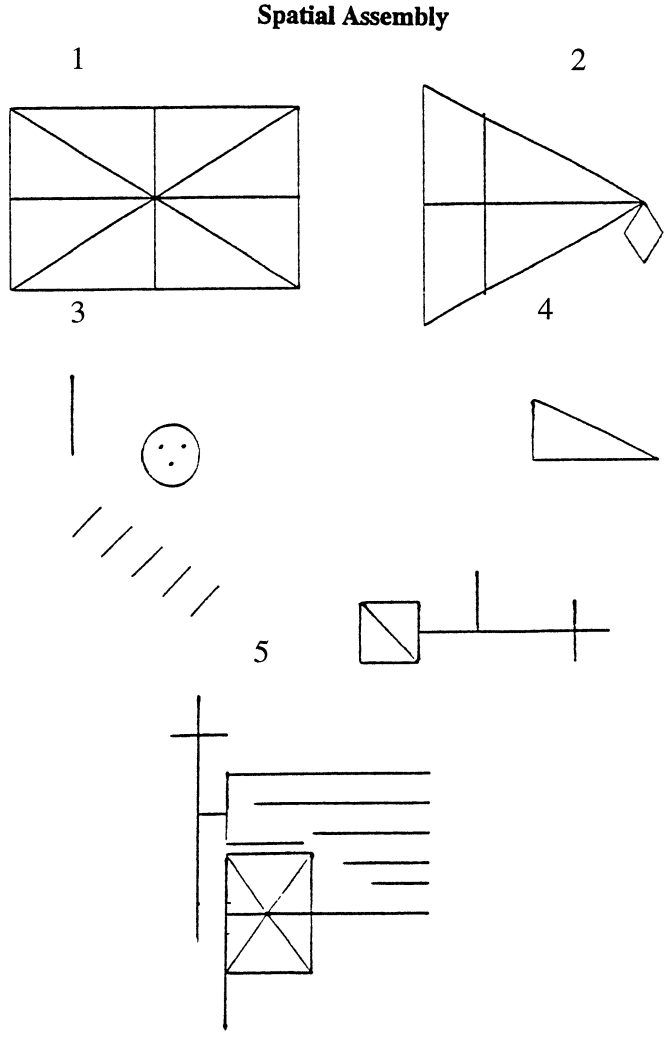

Fig. 1 Sketches of the subunits of the R-OEM protocol used for the organization trials (stage 2). Each subunit was on a clear acetate sheet and the subject was asked to arrange the subunits into their correct position.

Each subunit was presented successively, and was overlayed in its correct spatial position onto the previous subunit such that by the fifth subunit the complete figure was visible. Upon presentation of each subunit, subjects were asked to trace the subunit with their finger. The next subunit was then overlayed on the previous subunit and the subject was again asked to trace the subunit. This process was repeated until all five subunits were displayed and traced. Immediately following the fifth tracing, subjects were once again asked to reproduce the figure (i.e. immediate recall 2). This entire cycle was repeated until criterion was reached or until a maximum of five presentations. In the third stage of the assessment, subjects were asked to reproduce the figure following a 30-min delay (i.e. delayed recall). The 30-min delay was filled with non-visual tasks (e.g. digit-span, verbal memory assessment). Following the delayed free recall condition, all subjects were administered the extended memory measures (i.e. stage four) which consisted of the following (*see* Fig. 2): (i) a two alternative, forced choice subunit recognition task, consisting of five trials, in which one of the subunits was presented along with a foil showing a previously unseen figure; (ii) a spatial assembly task, in which subjects were given the five subunits presented above and asked to reassemble the subunits of the figure into their proper spatial position; (iii) a spatial discrimination task in which subjects were presented with five copies of the ROCFT and were required to discriminate which part of each figure was incorrectly positioned; (iv) a five alternative, figure recognition task in which subject's were asked to select the correct complete ROCFT. The maximal attainable score on the extended measures was 20 points [i.e. subunit recognition = 5; spatial assembly = 10 (maximum); spatial discrimination = 4; whole-figure recognition = 1].

Data analysis

The performance of two groups was examined in this study: amnesic versus non-amnesic ACoA's. Analysis of variance (ANOVA) procedures were used to assess these contrasts. However, when the assumptions of homogeneity of variance were violated, the non-parametric, Mann–Whitney Test was used. In addition, when evaluating relationships between memory and WCST performance (i.e. Spearman's ρ) all memory and WCST results were transformed into standard scores in order to account for scaling differences.

Results

Amnesic versus non-amnesic subgroups

Copy and immediate-recall conditions

The amnesic and non-amnesic subjects did not differ significantly from established norms (50 percentile, mean =

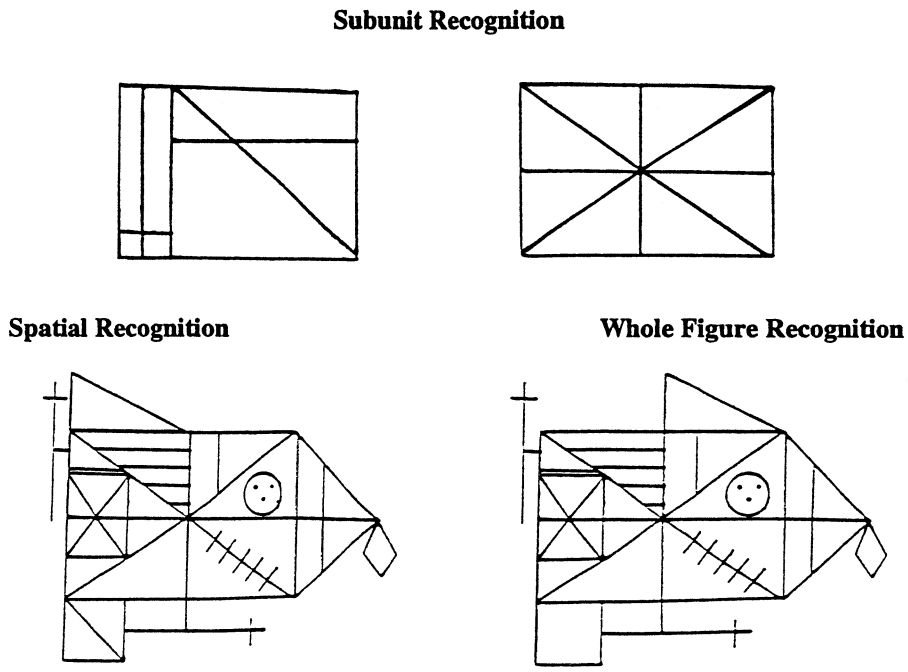

Fig. 2 The subunit recognition task which is a two-alternative, forced-choice recognition task. The spatial recognition task requires the subject to identify that part of the figure that is not in its correct position. The whole-figure discrimination task requires the subject to select the one correct figure among five alternative figures.

32) (*see* Lezak, 1995) or from each other [$F(1,8) = 1.52$, $P = 0.25$] with respect to their copy scores (*see* Table 3). In contrast, the amnesic group recalled significantly less information on immediate recall (mean, 9.5 ± 5.4) compared with the non-amnesic group (mean, 24.7 ± 4.2), [$F(1,8) = 21.8$, $P = 0.001$]. Based on established norms (50 percentile, mean = 22) (*see* Lezak, 1995) and previously published work from our laboratory (mean for healthy controls aged 35.7 ± 0.68 years, 22 ± 6.25; *see* Diamond and DeLuca, 1996), it is clear that the non-amnesic group was not impaired on immediate recall.

Importantly, following the last organizational trial, the amnesic group's mean recall score did not differ from the non-amnesic group's mean immediate recall score [$F(1,8)$, 2.7, $P = 0.13$]. In other words, the organizational strategy appeared to facilitate recall of the ROCFT in the amnesic group (*see* Fig. 3). The mean number of trials required by the amnesic group to reach criterion (or until discontinuation after five trials) was three. By definition, all of the non-amnesic ACoA's achieved criterion on the first trial.

Delay condition

The non-amnesic and amnesic groups did not differ with respect to recall on the delay condition (i.e. 30 min following the last recall trial) (Mann–Whitney: $Z = -1.7$, $P = 0.08$), [$F(1,8) = 2.8$, $P = 0.12$] (*see* Table 3 and Fig. 3), i.e. the improvement in learning that accrued over the organization trials was maintained over the delay. Importantly, a savings score (representing a ratio of the delayed recall score over the last immediate organization trial divided by the copy score) showed no significant difference between the amnesic (mean, 0.97 ± 0.23) and non-amnesic groups (mean, 1.02 ± 0.31), [$F(1,8) = 0.67$, $P = 0.43$]. In other words, the two groups did not significantly differ with respect to the amount of information that was lost between the immediate and delayed recall conditions. In addition, the amnesic (mean, 0.80 ± 0.12) and non-amnesic (mean, 0.85 ± 0.04) groups did not differ with respect to the proportion of the total possible points that could be attained on the extended memory total score [$F(1,8) = 0.52$, $P = 0.49$] (*see* Table 3A).

Wisconsin card sort test (WCST)

The WCST was used as a test of executive function. The analysis showed that the mean number of categories achieved and perseverative responses obtained in the amnesic and nonamnesic groups did not differ (*see* Table 2).

Facilitated versus non-facilitated groups

The amnesic ACoA group was not homogeneous with respect to their ability to benefit from an organizational strategy and, therefore, in the effectiveness of the R-OEM procedure in improving recall (for a review, *see* DeLuca and Diamond, 1995). Therefore, in order to account better for this heterogeneity, the amnesic subgroup was further divided into a facilitated group (those who benefited from the organizational procedure and who showed enhanced

Table 3 *Copy, recall and extended measure performance*

(**A**)

Variable	Amnesic		Non-amnesic	
	Mean	SD	Mean	SD
Copy	32.5	4.5	35.5	1.0
Immediate recall	9.5	5.4	24.7	4.2
Last organizational trial	16.5	10.2	24.7	4.2
Delayed recall	16.1	9.2	25.3	6.9
Extended measure score	0.8	0.12	0.85	0.04

(**B**)

Variable	Facilitated		Non-facilitated	
	Mean	SD	Mean	SD
Copy	34.6	2.3	30.3	5.6
Immediate recall	13.0	5.5	5.6	3.2
Last organizational trial	24.3	2.5	8.6	5.2
Delayed recall	23.6	2.8	7.8	6.9

(**C**)

Variable	Facilitated		Non-facilitated	
	Mean	SD	Mean	SD
Sub-unit recognition	5	0	5	0
Whole figure recognition	1	0	1	0
Spatial arrangement	9.6	0.5	8.3	1.5
Spatial recognition	3.6	0.5	3.3	0.7
Extended measure total score	0.71	0.05	0.94	0.19

These scores represent mean values with the exception of the extended measure total score which represents a proportional (normalized) score.

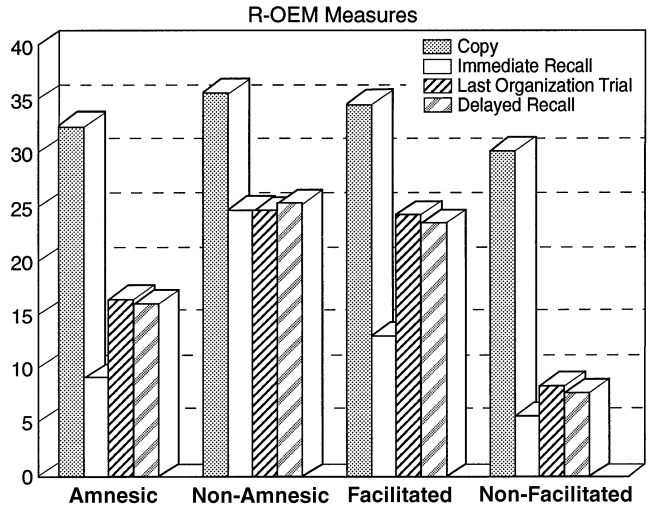

Fig. 3 Scores achieved on the copy and recall conditions by the amnesic, non-amnesic, facilitated and non-facilitated groups.

immediate recall) and a non-facilitated group (those who did not show enhanced recall following administration of the organization procedure). The results are presented in Fig. 3.

Immediate recall, organizational procedure and saving scores

A two-way ANOVA, with group as the between subjects factor, and recall (i.e. immediate recall versus recall on the last organization trial) as the within subjects factors, showed that the facilitated group did not differ from the non-facilitated group on the traditional ROCFT immediate recall condition [$F(1,4) = 3.8$, $P = 0.12$], (although the facilitated group showed a tendency towards better immediate recall). However, by the last organization trial, the facilitated group recalled significantly more information (mean, 24.3 $\pm$ 2.5) than the non-facilitated group (mean, 8.6 $\pm$ 5.2), [$F(1,4) = 26.6$, $P = 0.006$]. A one-way ANOVA was computed in order to assess if there were differences in the amount of information that was lost between the last organization trial and delayed recall (i.e. delayed recall divided by the result of the last organization trial); there were no significant differences between the facilitated (mean, 0.97 $\pm$ 0.04) and non-facilitated (mean, 0.94 $\pm$ 0.09) groups, i.e. virtually no information was lost between the last organization trial and the delayed recall condition. The important finding here is that by the last organization trial, the facilitated group,

unlike the non-facilitated group appeared to benefit from the additional organization trials.

Extended memory measures

Results on the extended memory measures (comprising the sum of the spatial recognition/discrimination, whole-figure recognition and spatial assembly measures minus recall scores) are presented in Table 3C. An analysis of mean extended memory total scores (expressed as proportion of the total possible score) showed that there were no significant differences between the facilitated (mean, 0.71 $\pm$ 0.05) and the non-facilitated (mean, 0.94 $\pm$ 0.19) [$F(1,4) = 3.9$, $P = 0.11$] groups. Furthermore, the mean performance did not differ between the two groups on any of the individual extended memory measures: (i) subunit recognition ($P = 0.37$); (ii) whole figure recognition ($P = 0.99$); (iii) spatial discrimination ($P = 0.57$); (iv) spatial asssembly ($P = 0.23$). In other words, despite the fact that the non-facilitated, amnesic ACoA subgroup exhibited little to no improvement in their recall of the ROCFT, they did not differ from the facilitated group with respect to their mean scores on any of the extended (i.e. non-recall) measures.

Wisconsin card sort test (WCST)

Importantly, there were no significant differences between the facilitated and non-facilitated groups in perseverations emitted. However, the non-facilitated group did achieve significantly fewer categories (mean,1.3 $\pm$ 0.5) than the facilitated group (mean, 4 $\pm$ 1) [$F(1,4) = 16$, $P = 0.01$].

Amnesic facilitated versus non-amnesic groups

Interestingly, a two-factor repeated measures ANOVA showed no main effects or interactions by group, recall on the last organization trial or delayed recall, showing that by providing an organization strategy, the amnesic facilitated group recalled as much information as the non-amnesic ACoA patients. Moreover, the two groups retained comparable amounts of information.

CT scan analysis

All of the ACoA subjects showed evidence of frontal lobe involvement on CT scan. However, only amnesic subjects also had involvement of areas beyond the frontal lobes. That is, three out of six amnesic ACoA subjects had infarcts in the basal ganglia, while two others had involvement in the Suprasellar region (*see* Table 1). None of the four non-amnesic ACoA subjects displayed lesions in regions other than the frontal lobes. Furthermore, two of the three facilitated patients displayed left frontal lesions while two of the three non-facilitated patients displayed right frontal lesions.

Discussion

The present study was designed to examine whether the impaired memory performance observed in ACoA subjects on the ROCFT is due to compromised encoding, consolidation or retrieval. The first major finding of this study is that the immediate recall of non-verbal material by some amnesic ACoA subjects could be significantly improved by providing them with an organizational strategy for encoding details of the ROCFT. That is, by providing a structured, multi-modal learning procedure which organized the figure into subunits and then combined motor input (i.e. tracing) with visual–spatial input over repeated trials, recall in the amnesic group could be improved to the level of the non-amnesic ACoA subjects (Kaplan, 1983, 1989). This finding provides some support for the idea that encoding deficits in the ACoA amnesics (perhaps attributable to executive dysfunction) may mediate their profoundly impaired immediate recall.

In order to measure rates of forgetting accurately, we attempted to raise the levels of immediate recall in the ACoA amnesic group so that their performance would match that of non-amnesic ACoA subjects more closely (Mayes, 1988). Thus, the second major finding is that, after the amnesic and non-amnesic groups were matched on immediate recall, the two groups retained comparable amounts of information and hence displayed similar rates of forgetting when tested at a delay of 30 min. Moreover, the amnesic and non-amnesic groups did not differ on the extended memory scores. These findings may suggest intact consolidation and provide further support for the idea that problems in encoding and organization of complex visual information may account for the impairments observed in the immediate recall of, at least some of, the amnesic ACoA subjects.

Importantly, there was heterogeneity in the performance of ACoA patients. In order to account for this heterogeneity better, amnesic ACoA patients were further categorized into two groups: those who benefited (the facilitated group) from the organization procedure, and those who derived little or no benefit (the non-facilitated group). Consistent with previous reports suggesting that performance on immediate recall closely parallels delayed recall on the ROCFT (Diamond and DeLuca, 1996), improvements in recall accrued over the organization trials by the facilitated group were maintained by this group over the 30-min delay. In other words, there was no evidence of accelerated forgetting which again suggests intact consolidation in amnesics and provides additional support for the encoding theory of ACoA amnesia.

As explained earlier, we also evaluated memory by using measures of recognition, spatial discrimination and spatial assembly in order to enhance the sensitivity of our measures and to allow us to evaluate better whether the ACoA group's memory impairments were attributable to failures of encoding, storage/consolidation or retrieval. The third major finding of this study is that, despite showing only marginal improvements when tested on explicit measures (e.g. recall), the non-facilitated group showed preserved memory when

memory was assessed with measures other than recall. That is, despite showing profoundly impaired immediate recall and failing to show any significant improvement in recall over repeated organizational trials, this amnesic group exhibited preserved memory on the extended measures. This is important, because without the information derived from the extended measures, differentiating whether impairment was due to encoding, consolidation or retrieval would be difficult. This group's performance on the extended measures suggests that information had been encoded, although the adequacy of that encoding may not have been normal for the purpose of recall. Further, preserved performance on the extended measures, in the face of poor recall, suggests relatively intact consolidation, but impaired retrieval, in the non-facilitated group.

Interestingly, much of the material remembered by the non-facilitated group was retrieved in the absence of awareness of the previous learning episode. That is, while performing many of the extended memory tasks perfectly, they expressed a sense of disbelief as to how they were able to perform the task (although this was not formally assessed).

An important issue that could be raised is whether the non-recall measures tapped the same information as the recall measures. In order to address this, we specifically designed the study so that the stimuli presented during recall (i.e. during the R-OEM trials) and the stimuli presented during recognition (i.e. extended measures) were identical. Thus, while one cannot say with absolute certainty that the recall and non-recall tasks involved retrieval of identical information, the present design optimized the conditions for such retrieval. Moreover, if the spatial assembly and discrimination tasks tap the contextual information that is needed for recall, then the fact that both the facilitated and non-facilitated patients could encode this information and retrieve it on the extended memory measures shows that memory for spatial context had been encoded. However, only the facilitated patients could, with effort, retrieve this contextual information for the purpose of recall (e.g. the contextual memory deficit hypothesis, *see* Mayes *et al*., 1985). In other words, performance on the extended measures and recall tasks appears to have been based on identical or similar representations. However, the expression of the underlying information appears to have been differentially affected by the retrieval demand characteristics imposed by the recall and non-recall tasks, rather than based on different informational representations.

The fourth major finding is that ACoA subjects cannot be classified into a single functionally homogeneous group (*see* Stenhouse *et al*., 1991). The present data suggest the existence of at least three subgroups (i) a non-amnesic group; (ii) an amnesic group displaying problems in the organization, encoding and representation of complex visual material in a form that is accessible for explicit retrieval purposes (i.e. facilitated group); (iii) an amnesic group who failed to show improvements in recall, following administration of the organizational procedure (i.e. non-facilitated group) (although performance on the extended measures was intact and remained so following a delay of 30 min) suggesting retrieval failure. Clearly, these results provide evidence against the use of a global descriptor in characterizing the pattern of functional impairments observed in ACoA patients.

The results of the present study have important implications with respect to differentiating the memory impairments in ACoA patients (with presumed basal forebrain damage) from those observed in patients with lesions to sites traditionally implicated in amnesia (e.g. mesial temporal and diencephalic). The current findings suggest the existence of at least two amnesic subgroups of ACoA patients. One group which exhibits impairments in encoding information (similar to diencephalic amnesia, i.e. alcoholic Korsakoff's syndrome; *see* Kopelman, 1995), and a second group that appears to have impairments in retrieval, although this group may have a combination of both retrieval and encoding deficits.

Subjects with right temporal-lobe lesions have shown striking impairments in recognition accuracy for figurative detail. In addition, these patients also have deficiencies in recognizing figural deletions and displacements. Importantly, only subjects with lesions that included a large hippocampal excision were impaired on a measure of object location (*see* Pigott and Milner, 1993). Clearly, in contrast to Pigott and Milner's patients, the ACoA patients in the current study, irrespective of the degree of impairment in encoding or retrieval, showed preserved memory for spatial recognition and object position (i.e. assembly performance and spatial displacement). This finding may further help differentiate ACoA patients from right mesial temporal patients with extensive hippocampal involvement in that spatial recognition accuracy may be impaired in the former and intact in the latter.

Compared with Korsakoff patients, ACoA amnesics may show disproportionately greater improvements in recall when provided with strategic information (e.g. *see* patient J.B. in Parkin *et al*., 1988). That is, patients with frontal lesions may tend to acquire information in a more passive manner, showing greater vulnerability to disturbances in the learning of complex material (i.e. the facilitated group) (Mayes, 1988). Moreover, frontal damage, which may compromise the ability to engage in strategic planning may impair both retrieval and acquisition (Luria, 1973) (i.e. non-facilitated group). Therefore, the current study provides support for the idea that the ACoA amnesics may exhibit a disproportionate ability to benefit from an organizational strategy and strategic cueing. This is in contrast to diencephalic (i.e. Wernicke–Korsakoff) patients, who may exhibit a less dramatic reduction in executive functioning, and mesial temporal amnesics, who presumably do not have frontal damage.

As discussed earlier, the results of the present study suggest that deficits on measures of long-term recall were not attributable to accelerated forgetting. Therefore, ACoA patients, like alcoholic Korsakoff's and unlike mesial temporal amnesics, do not show accelerated rates of forgetting. However, it should be noted that much of the previous literature in which rates of forgetting in patients

with mesial temporal and diencephalic lesions have been examined, has been based on recognition measures. While the results of some studies do suggest that patients with mesial temporal lesions tend to show accelerated rates of forgetting compared with patients with diencephalic lesions, there are discrepancies in the literature (Lhermitte and Signoret, 1972; Parkin, 1984; but *see* McKee and Squire, 1992). If ACoA and alcoholic Korsakoff patients show normal rates of forgetting compared with mesial temporal amnesics, it would argue against the idea that the amnesia observed in these groups is mediated by a single and common memory disturbance. However, until mesial temporal and diencephalic amnesics are examined on the R-OEM, we cannot be sure what pattern of forgetting they will show.

The results of the present study are consistent with previous ACoA studies in that the ACoA group's recognition and spatial discrimination scores were much better than their recall scores and their performance significantly improved with cueing (*see* Parkin *et al*., 1988). Differences in recall and recognition performance have been attributed to either an impairment in encoding and retrieval strategies or impairments in the organization of material to be learned (Risse *et al*., 1984; Smith and Milner, 1984; Janowsky *et al*., 1989; Gershberg and Shimamura, 1995; Eslinger and Grattan, 1992). While the literature supports the idea that amnesics are generally more impaired on recall than recognition (Volpe and Hirst, 1983; Hirst *et al*., 1986; Hirst *et al*., 1988; Mayes, 1988; Parkin, 1988; Moscovitch, 1989; DeLuca, 1992; Parkin *et al*., 1994), there are some reports that suggest that mesial temporal amnesics display both poor recall and recognition (Haist *et al*., 1992). In contrast, ACoA patients consistently show greater impairment in recall versus recognition (Hanley *et al*., 1994; DeLuca and Diamond, 1995) but *see* Delbecq-Derouesne *et al*. (1990). Therefore, a greater discrepancy between recall and recognition may serve to differentiate ACoA amnesics from other aetiologies. Once again, this can only be said with certainty after both ACoA and mesial temporal patients are directly compared on the same tests of recall and recognition. If substantiated, however, such data would tend to support a view of a multiple (Perani *et al*., 1993) versus a single memory system (Corkin, 1985).

If the degree to which the facilitated and non-facilitated groups benefited from the organizational strategy is related to the extent of their frontal lobe damage, do the two groups differ with respect to their performance on a test purportedly sensitive to executive functions (e.g. the WCST). Overall, both the amnesic and non-amnesic patients showed similar impairments in executive functioning. Furthermore, the facilitated and non-facilitated patients did not differ with respect to Perseverative Responses. However, these two groups did differ with respect to categories achieved (i.e. non-facilitated patients achieved fewer categories). While this may suggest that the non-facilitated group may exhibit greater impairment in executive functioning, this potential connection with organizational performance needs replication. On the other hand, since impairments in executive functioning may have adversely affected both organization at encoding and directed search at retrieval (Mayes, 1988; Delbecque-Derousne, 1990), one could hypothesize that the extent of damage to basal forebrain in conjunction with other areas (e.g. basal ganglia; *see* Irle *et al*., 1992) may be the critical determinants of whether the deficit is primarily one of encoding or retrieval. In support of this idea is the finding, in the present study, that only amnesic ACoA patients showed evidence of lesions outside of the frontal lobes on CT scans (i.e. five out of six) compared with non-amnesics (i.e. zero out of four). This is consistent with recent data suggesting that memory is more severely impaired in ACoA subjects when lesions extend beyond the basal forebrain to include other regions such as the basal ganglia (Irle *et al*., 1992; Hanley *et al*., 1994; for a review, *see* DeLuca and Diamond, 1995).

Lastly, in order to assess the implications of the present results it is important to discuss first the type of memory examined in this study. The R-OEM protocol involved copying, tracing, visual input and an organizational component. The copying as well as the tracing procedure, therefore, involved not only visual and organizational but motoric components as well. It could be argued that at least, in part, memory for the figure may have become proceduralized (Schacter, 1987) and may not have been represented in sites traditionally implicated in amnesia (*see* Mayes, 1988). However, these results cannot be entirely ascribed to proceduralized memory. First, recall was improved in some patients. Secondly, in the spatial assembly task, subjects at no time received training in assembling the subunits of the figure into their correct position despite the fact that the subunits in the recognition task were identical to those presented during the R-OEM. Performance on the spatial assembly task may have involved a combination of priming and novel task performance. We are currently assessing what role the organizational procedure plays in mediating performance on the extended versus recall measures.

It could be argued that any procedure that allows for additional trials might have led to an equivalent degree of improvement in performance. Indeed studies have shown that even amnesics can benefit from repeated stimulus exposures (*see* Mayes, 1988). However, in the present study, the non-facilitated patients were also given repeated exposures to the stimuli and they did not show improved recall performance. This suggests that simply providing additional learning opportunities does not necessarily result in improved performance.

It is hypothesized that it is the improved organization of the encoded material, resulting from the R-OEM procedure that produced better recall. This is consistent with some recent work in patients with frontal lesions showing that organizational strategies are effective in improving memory (Eslinger *et al*., 1995). However, because the present design did not include a control group which received only repeated exposures to the ROCFT (without the organization strategy),

one cannot say with absolute assurance that the specific organizational strategy employed in the present study was the primary reason for improved recall performance among the facilitated patients.

With respect to future work, implementation of the R-OEM procedure or similar procedures may allow us to differentiate better the various mechanisms that mediate impairments in performance across a wide spectrum of aetiologies and, importantly, allow us to better differentiate patterns of performance (i.e. encoding versus retrieval deficits) in various clinical groups. Future work will, however, need to address the issue of testing mesial temporal, diencephalic and ACoA amnesics on the same tests of recognition and recall in order to allow us to conclude that ACoA amnesia differs from classical amnesia. The R-OEM protocol and similar procedures may provide added assessment sensitivity, in addition to providing a way to organize and structure information in order to improve cognitive and vocational rehabilitation. Overall, the development and implementation of more sensitive neurobehavioural tests when used in combination with structural and functional neuroimaging may help resolve many of the outstanding issues in memory research.

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