A COMPARISON OF EGOCENTRIC AND ALLOCENTRIC SPATIAL MEMORY IN MEDIAL TEMPORAL LOBE AND KORSAKOFF AMNESICS

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Abstract

Two patients with medial temporal lobe damage, seven Korsakoff amnesics and fourteen healthy control subjects were tested on three conditions of a spatial memory test ('short delay', 'allocentric' and 'egocentric'). The task required subjects to recall the position of a single spot of light presented on a board after various delays. The 'short delay' condition tested memory over very short, unfilled intervals. The other two conditions used longer, filled delays. The allocentric condition required subjects to move to a different place around the board before recalling the position of the light. In the egocentric condition stimuli were presented in darkness, which eliminated allocentric cues. The Korsakoff amnesics were impaired at all delays of the short delay tasks, suggesting poor encoding. On the allocentric and egocentric tasks the Korsakoff amnesics showed a comparable impairment in the two conditions, which worsened with delay. This accelerated forgetting suggested that the Korsakoff amnesics also had impaired memory for allocentric and egocentric information. The patients with medial temporal lobe damage were unimpaired in the 'short delay' condition suggesting intact encoding and short-term memory of spatial information. However, they were impaired in the allocentric condition and showed accelerated loss of allocentric spatial information. In the egocentric condition, while the performance of one patient was impaired, the performance of the other was as good as controls. This result suggests that, in contrast to allocentric spatial memory, which is sensitive to medial temporal lobe damage, an intact medial temporal lobe need not be necessary for successful performance on an egocentric spatial memory task.

Key words: amnesia, egocentric, allocentric, spatial, memory

Introduction

Patients with anterograde amnesia suffer impairments of spatial memory. In real life, amnesics can readily find themselves lost (Corkin, 1984), while laboratory studies have revealed a failure to remember a variety of spatial stimuli (Cave and Squire, 1991; Holdstock, Shaw and Aggleton, 1995; Joyce and Robbins, 1991; MacAndrew and Jones, 1993; Mayes, Meudell and MacDonald, 1991; Pigott and Milner, 1993; Shoqeirat and Mayes, 1991; Smith and Milner, 1981, 1989; Warrington and Baddeley, 1974). Although all of these examples assessed 'spatial memory' it may well have been that their varying tasks tapped different cognitive processes. For example, Maguire, Burke, Phillips et al. (1996a) cite studies which show a dissociation between topographical orientation and other aspects of spatial memory. As a consequence, it is important to determine

whether all aspects of spatial memory are equally disrupted in amnesia. Further, the nature of any spatial memory loss, e.g. disruption of encoding or storage, needs to be investigated. These issues were addressed in the present study.

The best known distinction in the spatial memory literature is between allocentric and egocentric memory (O'Keefe and Nadel, 1978; Semmes, Weinstein, Ghent et al., 1963). The former refers to the identification of a place by reference to the relative position of an array of external stimuli or landmarks. This contrasts with egocentric memory, in which a location is identified by its relative position to the observer. Single cell recording studies of rats (Muller, Kubie and Ranke, 1987; O'Keefe, 1991; O'Keefe and Conway, 1978, O'Keefe and Dostrovsky, 1971; O'Keefe and Nadel, 1978; O'Keefe and Speakman, 1987) and monkeys (Feigenbaum and Rolls, 1991) have implicated the hippocampus in spatial memory particularly when an allocentric spatial frame of reference is used (but see Rolls and O'Mara, 1995). This has been supported by lesion studies in rats of the hippocampus (Jarrard, 1993; Morris, Garrud, Rawlins et al., 1982) and fornix (O'Keefe and Conway, 1980). In the study of human amnesia, only a few studies have attempted to develop tests which selectively require just allocentric or egocentric frames of reference. The majority of these studies have investigated spatial memory in patients who have undergone unilateral temporal lobectomy or who have unilateral hippocampal sclerosis rather than amnesia (Abrahams, Pickering, Polkey et al., 1997; Feigenbaum, Polkey and Morris, 1996; Goldstein, Canavan and Polkey, 1989; Maguire, Burke, Phillips et al., 1996; Morris, Pickering, Abrahams et al., 1996). The results of these studies have implicated the temporal lobe, particularly that on the right, in allocentric spatial memory. Further, the results of the study of Goldstein et al. (1989) suggested that right and left unilateral temporal lobectomy, in which the anterior 5.5-6.5 cm of the temporal lobe was removed, did not impair egocentric spatial memory. Consistent with these behavioural results, three positron emission tomography (PET) studies have shown activation of the right hippocampus whilst learning to navigate and orient oneself in an environment (topographical learning) (Maguire, Burgess, Donnett et al., 1998; Maguire, Frackowiak and Frith, 1996, 1997). These studies support the role of the hippocampus in allocentric spatial memory in humans but as appropriate nonallocentric comparison tasks have rarely been used, the question of whether the spatial memory impairment found after temporal lobe damage is exclusively allocentric has not been sufficiently investigated.

Recently, Holdstock, Mayes, Cezayirli et al. (submitted) studied the spatial memory of a patient, YR, with relatively selective damage to the hippocampus using a new procedure which distinguished allocentric from egocentric spatial memory. YR was impaired both at recalling and recognising the position of a previously illuminated light-emitting diode (LED) on a tabletop board after a one minute delay when the use of an allocentric reference frame was encouraged. However, YR was not significantly impaired on an egocentric version of the recall task at the same delay. The results of this study indicate a greater role for the hippocampus in allocentric than egocentric spatial memory and are consistent with the published findings described above.

The parahippocampal gyrus has also been implicated in spatial memory. Deficits

in topographical memory have been reported following medial temporal lobe lesions in humans (De Renzi, 1985; Habib and Sirigu, 1987; Landis, Cummings, Benson et al., 1986; Maguire, Burke, Phillips et al., 1996) including selective lesions of the right parahippocampal cortex (Bohbot, Kalina, Stepankova et al., 1997). In addition, a recent study (Bohbot, Kalina, Stepankova et al., 1998) found that a lesion to the right parahippocampal cortex impaired performance on a task which was analogous to the Morris Water Maze when memory was tested after a 30 minute delay. In contrast, a lesion to the right hippocampus did not impair performance on this task (Bohbot et al., 1998). The results of the study provided further support for the view that the parahippocampal cortex plays a critical role in some types of spatial memory. Further evidence for the role of the parahippocampal gyrus in spatial memory has come from functional magnetic resonance imaging (Aguirre, Detre, Alsop et al., 1996; Aguirre, Zarahn and D'Esposito, 1998) and PET studies (Maguire, Frackowiak and Frith, 1996) which have shown bilateral activation of the parahippocampal gyrus during topographical memory tasks. More specifically, Maguire, Frith, Burgess et al. (1998) suggested that the role of the parahippocampal gyrus is to encode object position e.g. the position of landmarks (but see Epstein and Kanwisher, 1998). Two other PET studies, in which the right parahippocampal gyrus was activated during retrieval of the location of an array of pictures presented on a computer screen (Milner, Johnsrude and Crane, 1997; Owen, Milner, Petrides et al., 1996), indicate that the memory processing of the parahippocampal gyrus may not necessarily be restricted to the allocentric frame used in topographical memory. In these studies, either allocentric or egocentric frameworks could have been used to remember picture locations.

The present study investigated the contribution of regions adjacent to the hippocampus to egocentric and allocentric spatial memory. As it is very unusual to find patients with selective damage to the parahippocampal gyrus, we studied two patients who were shown, by magnetic resonance imaging (MRI), to have bilateral medial temporal lobe damage which included both the hippocampus and the parahippocampal gyrus. In the light of the recent imaging data concerning spatial memory and the parahippocampal gyrus, we were interested to see whether having damage to the parahippocampal gyrus as well as the hippocampus, would accentuate the allocentric spatial memory deficit found after selective hippocampal lesions. Of equal interest was whether additional parahippocampal damage would affect egocentric spatial memory for single light positions. As discussed above, recent PET studies suggest that some aspects of egocentric spatial memory may be affected by parahippocampal gyrus damage (Milner et al., 1997; Owen et al., 1996) and we wanted to see whether egocentric memory for the information presented in our task (the position of the most recently presented LED) was dependent on the parahippocampal gyrus. Given the widespread view that damage to medial temporal lobe and diencephalic structures within Papez circuit result in equivalent memory deficits (Aggleton and Saunders, 1997; Delay and Brion, 1969; Parker and Gaffan, 1997; although see Parkin, 1992; Hunkin, Parkin and Longmore, 1994, for an alternative view), it is also of interest to investigate the role of diencephalic structures in allocentric and egocentric spatial memory. Therefore, a group of a patients with diencephalic damage due to Korsakoff's syndrome were also tested. Korsakoff amnesic patients are interesting

because they all display bilateral pathology in the mamillary bodies (Victor et al., 1971) which appear to be important for allocentric spatial memory (Neave, Nagle and Aggleton, 1997; Sutherland and Rodriguez, 1989). It is, of course, the case that people with Korsakoff's syndrome typically display pathology in a range of sites in addition to the mamillary bodies which may cause more widespread spatial memory deficits. The performance of these two sets of patients was compared with a group of healthy control subjects.

Materials and Methods

Subjects

Two subjects with medial temporal lobe damage (RS and NM), seven subjects suffering from alcoholic Korsakoff's syndrome and fourteen healthy control subjects (CON) were tested in this study. The Korsakoff group (two female, five male) had a mean age of 61 years (range 54-68).

Amnesic subjects were assessed with the WAIS-R and NART-R (Nelson and Willison, 1991) to obtain estimates of present and premorbid IQ, respectively. Memory was assessed with the revised version of the Wechsler Memory Scale (WMS-R) and the Recognition Memory Test (RMT) (Warrington, 1984). The scores of the individual patients and the mean scores of the group on these tests are shown in Table I. Measures of frontal lobe function were obtained for the Korsakoff patients using the Wisconsin Card Sorting Test (WCST) (Heaton, 1981), verbal fluency (Benton, 1968), design fluency (Jones-Gottman and Milner, 1977) and cognitive estimates (Shallice and Evans, 1978). The results of the patients on these tests are shown in Table II. On the WCST the performance of the group was a little low but not abnormal for this age group. Also shown in Table II are scores for the picture arrangement (Mcfie and Thompson, 1972) and block design (Benton, 1968; Goodglass and Kaplan, 1979) subtests of the WAIS-R which are also thought to tax frontal lobe functions.

Patient NM is a 46 year old man who became amnesic after suffering meningitis in 1970. Patient RS is a 49 year old man who became amnesic as a result of herpes simplex encephalitis in September of 1984. A magnetic resonance imaging (MRI) scan was obtained for both patients using a 3D T1-weighted radio-frequency spoiled gradient echo (SPGR)

TABLE I

Details of the Amnesic Subjects in Terms of Aetiology, Age, NART-R Full-scale IQ, WAIS-R Full-scale, Verbal and Performance IQ, Three Index Scores of the WMS-R and Number Correct on the Recognition Memory Test (RMT)

			NART-R	WAIS-R			WMS-R			RMT	
Subject	Aet.	Age	FIQ	FIQ	VIQ	PIQ	GM	ATT/C	DR	W	F
HKK	K	64	90	85	82	91	62	90	55	28	29
RST	K	58	102	105	104	105	51	81	50	30	38
SM	K	68	91	96	98	95	72	107	61	30	38
BP	K	58	94	95	94	99	72	111	51	38	38
BJ	K	66	91	101	103	97	64	101	64	28	29
HK	K	54	118	101	100	88	74	87	51	24	33
TC	K	58	98	80	80	80	50	60	58	30	24
Mean		61	97.7	94.7	94.4	93.6	63.6	91	55.7	29.7	32.7
S.D.		5.1	9.9	9.1	9.8	8.1	10	17.5	5.5	4.2	5.6
NM	M	46	84	84	82	87	77	88	50	26	32
RS	HSE	49	111	107	110	104	74	92	55	33	33

FIQ = full-scale IQ, VIQ = verbal IQ, PIQ = performance IQ, GM = general memory, ATT/C = attention/concentration, DR = delayed recall, W = words, F = faces. Aetiology key: K = alcoholic Korsakoff syndrome, HSE = post herpes simplex encephalitis, M = post meningitis.

TABLE II

Performance of the Amnesic Subjects on Tests of Frontal Lobe Function

	WCST No. Cat.	WCST No. P.R.	VF (FAS)	DF ppr	DF no	WAIS-R picture arrang.	WAIS-R block design	Cog. Est.
HKK	2	28	31	6.7	5	6	7	6
RST	5	30	26	28.6	5	5	9	9
SM	2	57	34	37.5	1	6	9	12
BP	6	20	16	0	4	9	8	3
BJ	3	66	32	15.8	11	7	8	6
HK	0	85	41	16.7	10	6	9	4
TC	2	23	23	61.1	0	2	6	2
NM	6	20	14					11
RS	6	23	26					4

The scores are number of categories (No. cat.) and number of perseverative responses (No. P.R.) on the WCST, corrected score on the verbal fluency test (VF), percent perseverative response on the design fluency test (DF ppr), novel output on the design fluency test (DF no), scaled score on the picture arrangement subtest of the WAIS-R (WAIS-R picture arrang.), scaled score on the block design subtest of the WAIS-R, raw score on the Cognitive Estimates Test (Shallice and Evans, 1978) (Cog. Est.). Scores for patients NM and RS are given for WCST, verbal fluency and cognitive estimates.

sequence (TE = 9 ms, TR = 34 ms, flip angle = 45 degrees, matrix size = 256 × 192, 2 NEX, field of view = 20 cm, acquisition time = 27 minutes and 52 seconds) available on a 1.5 T SIGNA whole-body magnetic imaging system (General Electric, Milwaukee, WI, U.S.A.). Each image referred to a contiguous section of tissue, 1.6 mm thick. Images for both patients are shown in Figure 1.

Detailed radiological inspection of NM's scans showed partial bilateral damage to the head, body and tail of the hippocampus accompanied by partial damage to the amygdala and to the parahippocampal, perirhinal and entorhinal cortices. In addition, there was slight atrophy of the superior frontal and parietal lobes, mamillary bodies and cerebellum. RS's scan showed the hippocampus to be shrunken throughout its length with more evident volume loss at the head. There was no apparent damage to the amygdala. The parahippocampal, perirhinal and entorhinal cortices appeared to be thin on the left and there was possible damage to the perirhinal cortex on the right. The scan also revealed slight gyral and cerebellar atrophy.

In order to obtain quantitative measures of pathology, the volumes of the whole hemisphere, lateral ventricle, third ventricle, caudate, temporal lobe, hippocampus, parahippocampal gyrus, amygdala and parietal lobe were estimated on the right and the left using stereology (Bartzokis, Mintz, Marx et al., 1993) available in Analyze (Mayo Foundation, Minnesota, U.S.A.). The area of the parahippocampal gyrus, the gyrus adjacent and inferior to the hippocampus, which includes the parahippocampal, the entorhinal and the perirhinal cortices plus white matter was measured on the same sections as the hippocampus. In addition to these measures, volumes of grey and white matter were obtained for the prefrontal cortex, which was defined as that part of the frontal lobe which is rostral to the anterior-most point of the corpus callosum when the images are aligned along the AC-PC line. For grey/white matter segmentation, images were spatially co-registered with a reference template using a 12 point affine transformation in the Talairach-space and were segmented into grey and white matter compartments using a modified clustering algorithm.

For each subject, the area of a single midsagittal T1 weighted image from that subject's scan was used to correct the structure volumes for inter-subject variability in pre-morbid brain volume. The section used was that on which the pineal gland and the whole aquaduct of Sylvius could be seen. The slice also passed between the mamillary bodies so that a minimal amount of these structures was visible. The section area was delineated by the internal table of the skull, the tentorium cerebelli, and an imaginary line joining the most anterior point of the tentorium cerebelli and the most inferior point of the frontal lobe. The

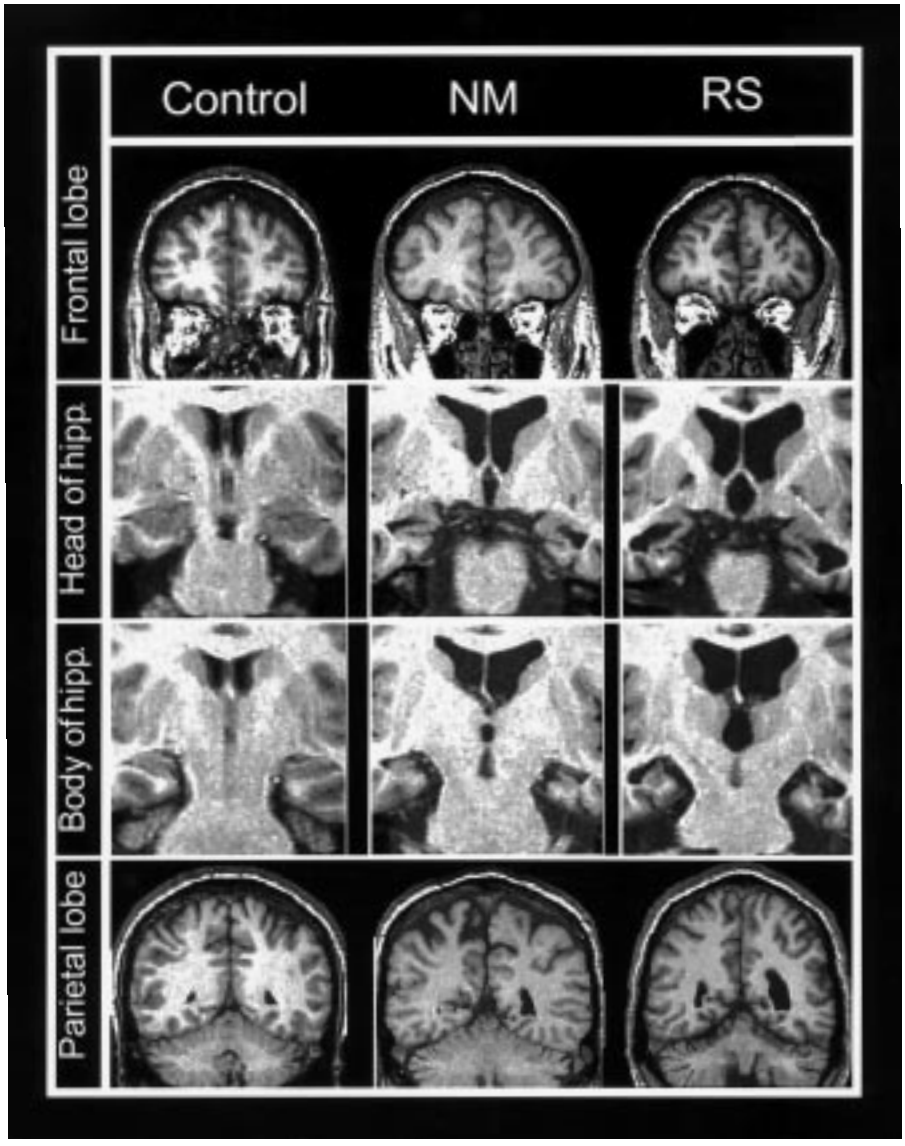

Fig. 1 – *Coronal sections from the patients RS, NM, and an age- and sex-matched control subject. Images on the top and bottom rows show sections through the frontal and parietal lobes, respectively. Images on the second and third row pass through the head and body of the hippocampus, respectively. Heads of the hippocampi of the patients are significantly smaller than controls on both sides. These images also show the parahippocampal gyri.*

section area was calculated by counting the number of pixels within the area of interest. The individual estimated brain structure volumes were then corrected by dividing them by the section area. The estimated structure volumes for the patients were compared with those of a group of eight healthy male controls matched for age.

The estimated volumes of NM's and RS's hippocampi were comparable and significantly smaller than their controls (see Table III). However, more detailed investigation of the hippocampus revealed differences in RS's and NM's pattern of pathology. In this

TABLE III

Volume Measures, before and after Correcting for Sagittal Section Area (see text), of the Hemispheres, Lateral Ventricles, Third Ventricles, Caudate, Temporal Lobe, Hippocampus, Parahippocampal Gyrus, Amygdala and Parietal Lobe for Patients RS and NM, the Mean Volume and Standard Deviation, in brackets, of these Structures for a Group of Age and Sex Matched Controls and the Number of Standard Deviations that RS's and NM's Corrected Volumes were from the Control Mean for each Structure. (Negative Standard Deviations indicate a Smaller Volume than Controls)

	Con		RS			NM		
	raw	corr	raw	corr	sds	raw	corr	sds
R hem	515.7 (41.9)	4.63 (0.139)	457.9	3.821	– 5.82*	557.5	4.34	– 2.07*
L hem	500.8 (40)	4.496 (0.106)	424.2	3.539	– 9.02*	525	4.089	– 3.84*
R wm	232.7 (27.55)	2.088 (0.184)	186.9	1.560	– 2.87*	259.2	2.018	– 0.38
L wm	213.8 (24.07)	1.918 (0.133)	173.5	1.448	– 3.54*	221.8	1.728	– 1.4
R vent	7.47 (3.25)	0.068 (0.029)	18.57	0.155	2.99*	11.25	0.088	0.69
L vent	7.98 (3.41)	0.071 (0.028)	28.8	0.240	5.99*	15.42	0.120	1.73
3rd vent	4.78 (1.58)	0.043 (0.014)	7.36	0.061	1.3	4.01	0.031	– 0.86
R caud	3.79 (0.77)	0.034 (0.007)	3.06	0.025	– 1.29	3.73	0.029	– 0.76
L caud	3.85 (0.92)	0.035 (0.008)	3.51	0.029	– 0.65	4.37	0.034	– 0.07
R temp	77.06 (4.45)	0.693 (0.039)	58.35	0.487	– 5.34*	84.37	0.657	– 0.94
L temp	71.49 (7.49)	0.643 (0.065)	53.71	0.448	– 3*	79.66	0.620	– 0.35
R hipp	2.5 (0.44)	0.022 (0.003)	1.27	0.011	– 4.03*	1.46	0.011	– 3.76*
L hipp	2.37 (0.41)	0.021 (0.003)	0.98	0.008	– 5*	1.16	0.009	– 4.69*
R parahipp	2.66 (0.54)	0.024 (0.004)	2.85	0.024	0	2.08	0.016	– 2*
L parahipp	2.49 (0.58)	0.022 (0.004)	2.46	0.021	– 0.41	1.62	0.013	– 2.2*
R amyg	1.92 (0.29)	0.017 (0.002)	1.6	0.013	– 1.86	0.97	0.008	– 4.57*
L amyg	1.8 (0.32)	0.016 (0.002)	1.68	0.014	– 0.88	0.62	0.005	– 4.71*
R Par	8.75 (0.62)	0.079 (0.003)	8.09	0.068	– 3.36*	6.79	0.053	– 7.79*
L Par	8.53 (0.58)	0.077 (0.004)	8.57	0.072	– 1.33	6.79	0.053	– 6.1*
R Prefron	25.65 (3.8)	0.231 (0.038)	26.59	0.222	– 0.26	30.37	0.237	0.14
L Prefron	26.67 (4.38)	0.241 (0.041)	27.40	0.229	– 0.29	27.34	0.213	– 0.68

* Indicates greater than 1.96 SDs below the control mean.

Con = control mean (sd in brackets); raw = estimated volume of structure; corr = volume structure after correcting for sagital section volume (see text); sds = number of standard deviations that the patient's corrected volume was from the corrected control volume; R hem = Right hemisphere; L hem = Left hemisphere; R vent = Right lateral ventricle; L vent = Left lateral ventricle; 3rd vent = Third ventricle; R caud = Right caudate; L caud = Left caudate; R temp = Right temporal lobe; L temp = Left temporal lobe; R hipp = Right hippocampus; L hipp = Left hippocampus; R parahipp = Right parahippocampal gyrus; L parahipp = Left parahippocampal gyrus; R amyg = Right amygdala; L amyg = Left amygdala; R Par = Right parietal lobe; L Par = Left parietal lobe; R Prefron = Right prefrontal lobe grey matter; L Prefron = Left prefrontal lobe grey matter.

analysis, for NM, RS and their controls, the sectional area of the hippocampus was plotted for 15 slices sampled from the entire length of the structure. As shown in Figure 2, RS had a smaller hippocampus anteriorly than NM on both the right and the left whereas NM had a slightly smaller hippocampus than RS posteriorly. The volume measures of the parahippocampal gyrus and amygdala confirmed that NM had suffered more extensive damage to medial temporal lobe structures beyond the hippocampus than RS.

The estimated volumes of the whole hemispheres suggested that RS had greater cortical atrophy than NM (see Table III). The measures of whole hemisphere white matter volume showed that RS also had significantly less white matter than the controls whereas this was not the case for NM.

Separate volume measures of the prefrontal, temporal and parietal lobes revealed that neither patient showed significant atrophy of the frontal lobe. In the temporal lobe, atrophy was greater bilaterally in RS than NM but the reverse was true for the parietal lobe, where NM had much greater atrophy bilaterally than RS (see Table III). This latter finding is particularly relevant given the proposed role of the parietal cortex in egocentric spatial processing (e.g. Anderson, 1997; Berthoz, 1997; Karnath, 1997; McDaniel, Via, Smith et al., 1995; Save and Moghaddam, 1996). RS also had enlarged lateral and third ventricles whereas NM's ventricle volumes did not differ significantly from the control mean volume (see Table III). Enlarged third ventricle volume may indicate pathology of diencephalic structures such as the thalamus.

Psychometric test scores for patients NM and RS are shown in Tables I and II. In addition, both patients have been tested on the Doors and People test battery (Baddeley, Emslie and Nimmo-Smith, 1994). NM showed poor performance on all subtests of the battery. His performance on the recall subtests, "People" and "Shapes", and the nonverbal recognition test, "Doors", was at less than the first percentile and was at the first percentile level for the verbal recognition, "Names", subtest. RS's performance on the recall subtests, "People" and "Shapes", was at less than the first percentile, which was slightly worse than his performance on the two recognition tests, "Doors" and "Names", where he was between the 10th and 25th percentile level and at the 25th percentile, respectively.

The control (CON) subjects (7 male and 7 female) had a mean age of 56 years (range 47-73). Psychometric testing of these subjects was limited to the NART-R and the mean predicted FSIQ was 103 (range 86-122). A t-test showed that the NART-R FSIQ scores did not differ significantly from those of the Korsakoff patients ($t = 1.05$, d.f. = 19, $p > 0.1$) nor did they differ from the WAIS-R FSIQ scores of the Korsakoff patients ($t = 1.68$, d.f. = 19, $p > 0.05$). The WAIS-R FSIQ scores of patients NM and RS were within one standard deviation of the mean NART-R FSIQ score of the controls.

Apparatus

The study required subjects to view a single light on a large uniform board which consisted of a featureless sheet of translucent Perspex which had a matt finish, so that reflections were not visible in its surface. The sheet was 60.5 cm wide, 91 cm long, and rounded at both ends. Care was taken to ensure that neither the Perspex nor its surrounding edges provided distinctive cues, i.e. it was free of blemishes or joins. Embedded under the Perspex board were twenty-five 20 milliamp standard red LEDs arranged in a random manner. When unlit, the LEDs were not visible from above the test board. When lit, the LEDs were sufficiently dim to avoid causing afterimages.

Design and Procedure

For all task conditions the lightboard containing the LEDs was positioned on a stand in the centre of a room away from obvious local cues. The test apparatus was portable and a variety of rooms were used for the study. The subject was instructed to attend to the lightboard, an LED was lit for approximately two seconds and, after a delay, the subject had to mark the location of that LED. Each of the three task conditions examined consisted of a single session of 45 trials in which three different retention durations (15 trials for each duration) were presented in a mixed sequence. As there were 25 LEDs on the board and 45

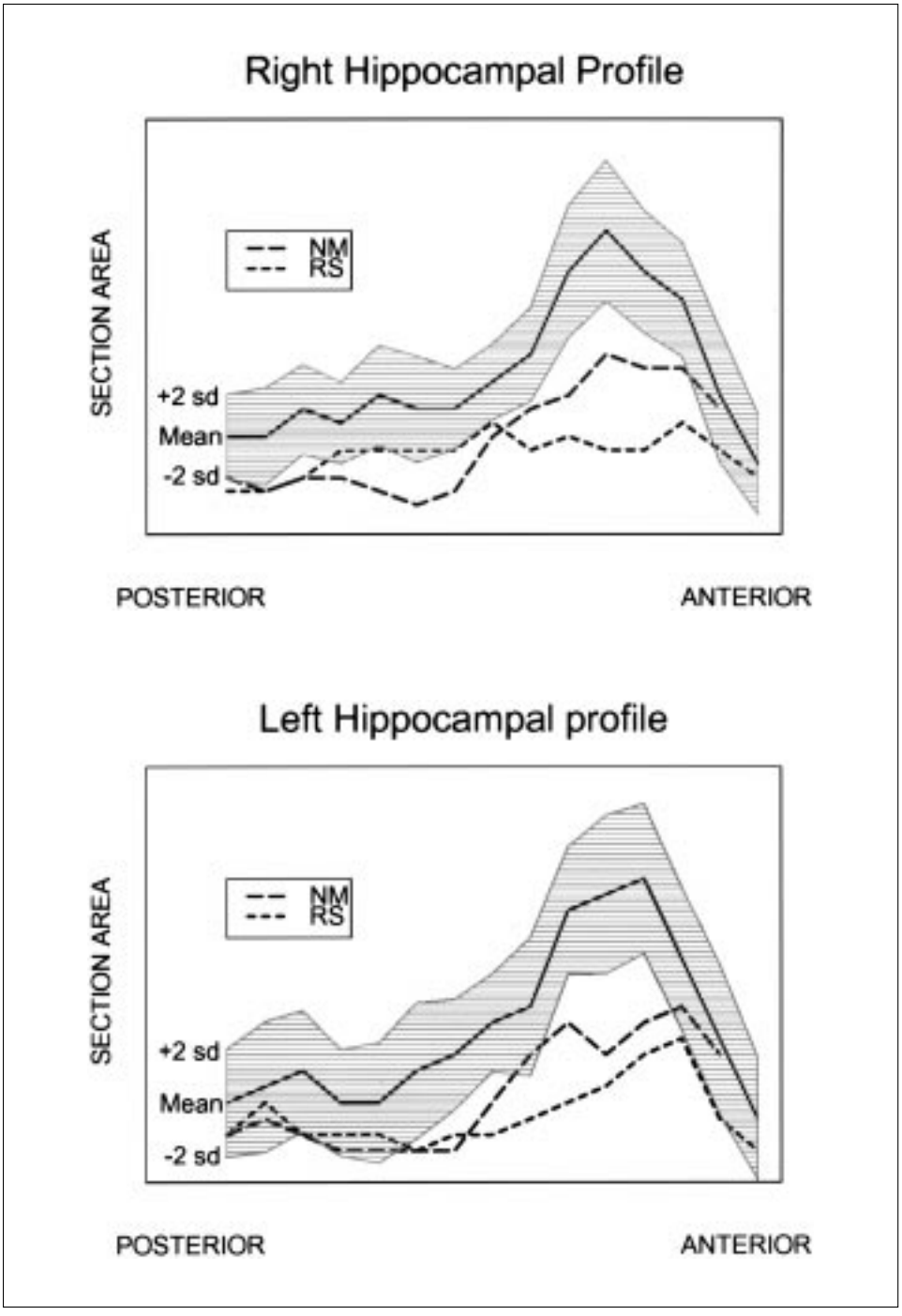

Fig. 2 – *Right and left hippocampal profiles of patients RS and NM plotted against eight age- and sex-matched control subjects. The shaded area is formed by the lines representing two standard deviations above and below the mean section area of the eight controls.*

trials per session, 20 LEDs were presented twice within a session. The repeated LEDs were varied between tasks and between subjects. For two of the three conditions ('allocentric' and 'egocentric') the retention interval was filled as the subjects were required to read a passage of prose. This distractor task was intended to preclude the use of a verbal mediating strategy to help solve the task and to ensure that it was not possible to fixate on the target region through the delay period. A particular advantage of the memory task was that it was demanding even at very short retention delays, which made it possible to avoid ceiling effects.

(1) *'Short Delay'*

In the 'short delay' condition the subject was asked to place a counter (22 mm diameter with a 2 mm hole in the centre) in the exact position of the light immediately after it disappeared (0 s) or after an unfilled delay of 3 s or 8 s during which the subject was instructed to look away from the board. The subjects did not move during the test procedure. Each subject was given one of two test sequences, which differed in the order in which the LED lights were presented and the order of the retention delays. Alternate subjects were given the same sequence. Patients RS and NM were given different sequences to each other.

(2) *Allocentric*

The allocentric condition used a manipulation similar to that used by Morris et al. (1996) and Abrahams et al. (1996). Presentation of the LED sample was followed by a filled delay of 5 s, 20 s or 60 s. Approximately three seconds before the end of the delay, subjects walked to a location indicated by the experimenter which was either the other side of the board or one of the two ends and then, in this new position, placed the counter (described above) as close as they could to the position of the previously illuminated LED. As a consequence of the subject's changed position, the target retained its spatial relationship to all exterior cues except for the subject. Subjects then returned to their original start position before seeing the next target LED.

(3) *Egocentric*

In the egocentric condition testing took place in a blacked out room with the room lights switched off. This manipulation ensured that no allocentric cues were available to contribute to performance. At the end of the filled delay, during which subjects read using a torch, the subject was asked to position a light pointer (Loewe Opta Luxitron 40) to indicate the location of the previous LED. The light pointer was attached to the arm of an angle-poised lamp (with the shade and bulb removed). This arrangement produced a stable circle of light on the board that was approximately the same size as the counter used in the other conditions. The subject was encouraged to stand in the same place throughout each trial. In order to reduce dark adaptation and hence to help ensure that room cues and the edge of the board were not visible to the subject whilst the target light was presented, the room lights were turned on between each trial. Although this meant that subjects were not prevented from forming a mental image of the room it ensured that visible allocentric cues were not available *during* target LED presentation; therefore the position of the LED could not be encoded with reference to these cues, so an egocentric frame of reference had to be used. Debriefing of control subjects confirmed that they had used an egocentric frame of reference.

In all three conditions the position of the centre of the counter (or circle of light in the egocentric task) was marked on a removable Perspex sheet. Each control subject completed just one allocentric and one egocentric sequence. A number of trial sequences were constructed which differed in the order in which target positions and delays were tested and each sequence was used an equal number of times in each condition. The trial sequences which were presented to NM in the allocentric and egocentric conditions were presented in the opposite conditions for RS. All Korsakoff patients were tested on the 'short delay' condition but it was not possible to test patient TC on the allocentric and egocentric conditions.

Finally, a subset of subjects were tested on a control task to determine if the amnesic

subjects were able to place the counter with the same degree of precision as the control subjects when there was no memory load. Six control subjects, six Korsakoff subjects, patient NM and RS were tested on this 'simultaneous condition', each receiving 15 trials which corresponded to 15 different positions. For this, a single LED was illuminated and the subject asked to position the counter as accurately as possible over the light. The light remained visible while the subject positioned the counter. The position of the centre of the counter was recorded on the sheet of Perspex in the same way as for the memory tasks.

RESULTS

The results for all conditions of the spatial task were based on the distance of the recalled target location from the centre of the actual target LED and, for each condition, the mean spatial error was calculated for each delay. The scores of the two medial temporal lobe amnesics were considered to be impaired if they were more than 1.96 SDs worse than the control mean, giving a type 1 error probability of 0.05, 2 tailed. All simple effect analyses used the method of Keppel (1973).

Simultaneous Condition

The mean error distance was 2.4 mm for the six control subjects and an identical mean error was found for the six Korsakoff patients. Patient RS produced a mean error of 2.9 mm and NM produced a mean error of 3.3 mm, which were unimpaired.

'Short Delay' Condition

RS and NM

Both RS and NM were unimpaired in the 'short delay' condition. Table IV shows their mean error scores for each delay of this task which were all within 1.96 SDs of the control mean.

TABLE IV

Mean Error Distance, with Standard Deviation in Parentheses, for the Korsakoff and Control Groups and Mean Score for Patient RS and Patient NM for the Three Delays (0 s, 3 s, 8 s) of the 'Short Delay' Condition of the Spatial Memory Task

	Delays		
Subject type	0 s	3 s	8 s
Korsakoff group	2.1 (0.7)	3.0 (1.1)	3.6 (1.2)
Control group	1.5 (0.5)	2.4 (0.4)	2.8 (0.8)
NM	1.1	2.3	3.9
RS	0.9	1.8	1.7

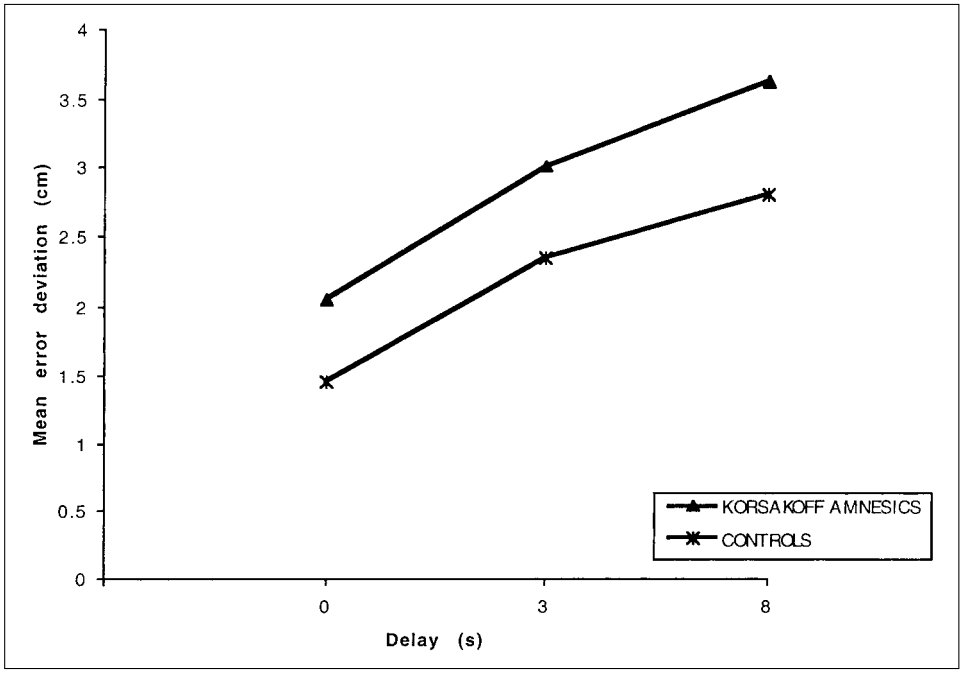

Fig. 3 – *Graph showing the mean error score for the Korsakoff amnesics (N = 7) and the controls (N = 14) for the three delays (0 s, 3 s, 8 s) of the 'short delay' condition of the spatial memory task.*

Korsakoff Group

An ANOVA with a between subject factor of group and within subject factor of delay compared the performance of the control subjects and Korsakoff patients at the three delays (0 s, 3 s, 8 s) of the 'short delay' condition (Figure 3). The Korsakoff patients were significantly worse in this condition than the controls ($F = 6.76$; d.f. = 1, 19; $p < 0.05$). There was no significant group by delay interaction ($F = .24$; d.f. = 2, 38; $p > 0.05$), however, an analysis of simple effects showed that the only significant difference between groups was at the 0 s delay ($p < 0.05$) and not at the 3 s and 8 s delays ($p > 0.05$). Finally, there was a significant effect of delay ($F = 34.0$; d.f. = 2, 38; $p < 0.001$) indicating that, for both groups, accuracy decreased as delay length increased.

Allocentric and Egocentric Conditions

RS and NM

Both patients were impaired in the allocentric condition at the longer delays (see Table V). Neither RS nor NM were impaired at the 5 s delay. In contrast, RS was clearly impaired at 20 s and there was a trend for him to also be impaired at 60 s. Similarly, NM was impaired at the 60 s delay but unlike RS he was not impaired at the 20 s delay.

TABLE V

Mean Error Distance, with Standard Deviation in Parentheses, for the Korsakoff and Control Groups and Mean Score for Patient RS and Patient NM for the Three Delays (5 s, 20 s, 60 s) of the Allocentric Condition and the Egocentric Condition of the Spatial Memory Task

Subject type	Allocentric task delay			Egocentric task delay		
	5 s	20 s	60 s	5 s	20 s	60 s
Korsakoff group	11.3 (6.0)	14.0 (6.2)	17.2 (6.4)	10.0 (4.1)	12.2 (5.0)	19.2 (5.1)
Control group	5.9 (1.6)	6.7 (1.6)	8.3 (2.4)	7.1 (1.7)	7.8 (2.7)	9.7 (2.2)
NM	7.6	6.11	13.4	4.3	8.6	9.4
RS	6.8	11.5	12.1	9.7	14	16.1

In the egocentric condition the two patients showed conflicting patterns of performance. As shown in Table V, RS was impaired in this condition at the 20 s and 60 s delays wheras NM was not impaired at any delay; his performance was actually better than the control mean at two delays.

In order to investigate further the rate of forgetting in each condition we calculated for the controls, NM and RS the difference in mean error distance between successive delays. In the allocentric condition accelerated forgetting (i.e. a difference score 1.96 SDs greater than the control mean difference) was shown by NM between delays of 20 and 60 seconds only (difference score was 2.8 SDs larger than the control mean difference), whereas RS showed accelerated forgetting between the first two delays only (difference score was 3.75 SDs larger than the control mean difference). In the egocentric condition, the difference scores of both patients were within 1.96 standard deviations of the mean control difference for both pairs of delays. However, the difference between error distance at 60 and 5 seconds was significantly larger than the control difference for RS (2.9 SDs larger), but not NM.

Korsakoff Group

An ANOVA was performed with a between subject factor of group and within subject factors of condition and delay in order to compare the performance of the control subjects and the Korsakoff patients on the allocentric and egocentric conditions at delays of 5 s, 20 s and 60 s (Figure 4).

The analysis revealed a highly significant effect of group ($F = 22.2$; d.f. = 1, 18; $p < 0.001$) but this did not interact with condition ($F = 1.72$; d.f. = 1, 18; $p > 0.05$). This indicates that the Korsakoff patients were similarly impaired in both conditions. This significant difference from the control subjects was confirmed in separate analyses of scores from the allocentric condition ($F = 6.09$; d.f. = 1, 18; $p < 0.05$) and the egocentric condition ($F = 16.42$; d.f. = 1, 18; $p < 0.05$). The main effect of condition was not significant ($F = .54$; d.f. = 1, 18; $p > 0.05$), showing that the overall scores in the allocentric and egocentric conditions were similar. Simple effects analysis of the nonsignificant group-by-condition interaction showed that, for each subject group, there was no significant difference between performance in the two conditions (both $p > 0.05$). There was, however, a significant main effect of delay ($F = 62.1$; d.f. = 2, 36; $p < 0.05$) which interacted significantly with group ($F = 15.3$; d.f. = 2, 36;

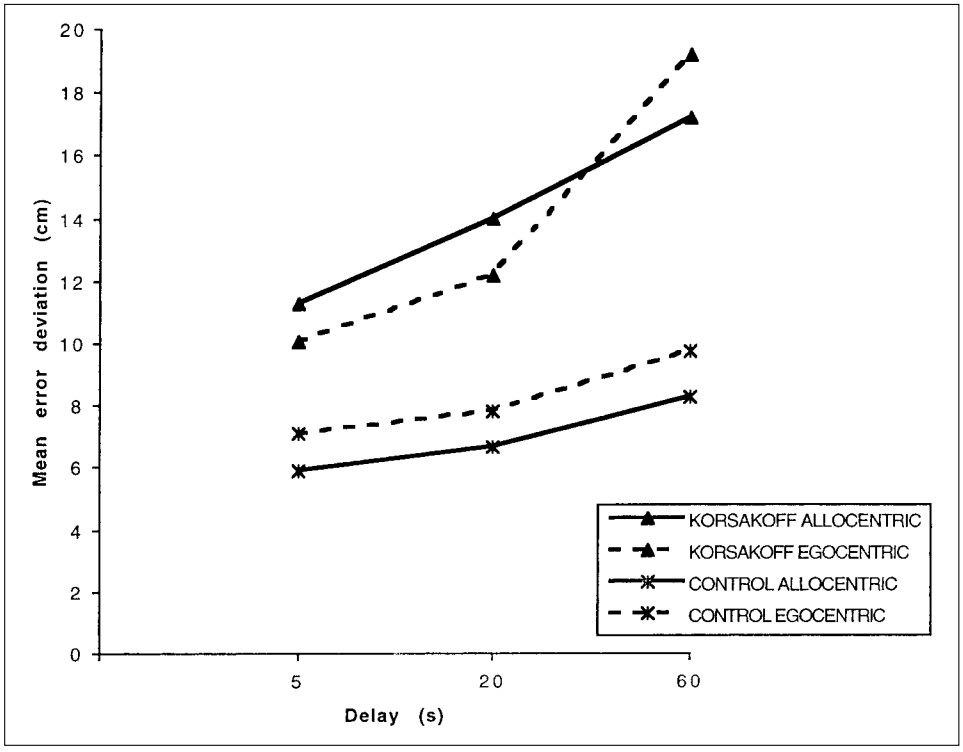

Fig. 4 – *Graph showing the mean error score for the Korsakoff amnesics (N = 6) and the controls (N = 14) for the three delays (5 s, 20 s, 60 s) of the allocentric and egocentric conditions of the spatial memory task.*

$p < 0.001$). An analysis of simple effects revealed a significant effect of delay for both subject groups (both $p < 0.05$), but the decline in performance over delay was greater in the Korsakoff than control group (see Figure 4). There was also a significant interaction between condition and delay ($F = 3.44$; d.f. = 2, 36; $p < 0.05$). Simple effects indicated that the interaction was due to performance on the egocentric and allocentric conditions diverging after the 20 s delay. This condition-by-delay effect was particularly evident in the Korsakoff data but the group-by-condition-by-delay interaction failed to reach significance ($F = 2.5$; d.f. = 2, 36; $p = 0.096$). To control for the effects of large outlying scores that may have resulted from a failure to consistently attend to stimuli, the analyses were repeated using median scores. Identical results were obtained.

Test Correlations

Spearman rank correlations tested whether there was a relationship between performance in the egocentric and allocentric conditions, and whether performance on the spatial task correlated with any of the other psychometric measures.

The first set of correlations showed that the overall performance of the

Korsakoff subjects on the egocentric and allocentric tasks was not significantly correlated (rho = 0.49; $p > 0.05$). The scores of the control subjects did, however, reveal a significant correlation between the two spatial tasks (rho = 0.70, $p < .05$). The second set of correlations tested for a relationship in the Korsakoff patient data between the mean score from the three delays on the 'short delay' condition of the spatial memory task, the allocentric condition or the egocentric condition and scores on the tests of frontal lobe function which are listed in Table II. No correlations were significant (all ps > 0.05). An additional set of tests investigated correlations between the mean score of the Korsakoff patients over the three delays of the 'short delay' spatial memory task and digit span, visual span and the attention/concentration index of the WMS-R. No correlations reached statistical significance (all ps > 0.05). A final set of correlations tested for a relationship between performance on the allocentric and egocentric tasks and scores on the WMS-R general memory index, WMS-R delayed memory index and total score of the WRMT. None of these correlations reached statistical significance (all ps > 0.05).

Discussion

The results suggest that the Korsakoff patients have a possible deficit in both attention and spatial memory. The Korsakoff patients were impaired at remembering spatial positions even at the zero second delay and it was confirmed that this deficit was not due to difficulty placing the counter where intended. These results appear to suggest that the Korsakoff patients could not encode spatial information as well as controls either due to impaired encoding mechanisms or to an attentional deficit. The literature concerning encoding by Korsakoff amnesics is conflicting so it has not been clearly established whether this patient group has widespread cognitive deficits that affect information encoding. Mayes et al. (1993) and Warrington and Baddeley (1974) argue that encoding is intact in Korsakoff amnesics whereas others, such as Cave and Squire (1992), have found an impairment of spatial short-term memory at delays as short as zero seconds. The apparent discrepancy between the results of these studies may not be surprising given that a variety of tasks have been used which may tap slightly different aspects of spatial memory. Thus the encoding success in these patients may depend on the precise nature of the test materials and conditions. The study which has used the most similar task to ours found no evidence of an impairment in a group of amnesics, including patients with Korsakoff's syndrome, in the ability to recall the location of a dot on a lightboard after short delays (Warrington and Baddeley, 1974). Differences with the present task include the use of a considerably smaller test board than ours (31×31 cm) and a shorter sample presentation (one second rather than two second). The impaired performance for our patients was therefore not consistent with these findings. Cave and Squire (1992) proposed that Korsakoff amnesics show an excessive degree of distractibility, caused by frontal lobe damage, which may often cause them to fail to notice the stimulus so that they show impairments even at short delays. Our findings were not consistent with this proposal. The

analysis of median scores suggested that the results obtained in the present study could not be explained by sporadic lapses of attention and a correlational analysis gave no indication of a relationship between spatial short-term memory and frontal lobe function, although the modest group sizes did limit the power of this latter analysis. Other researchers suggest that Korsakoff patients show a generalised impairment in attention or perception due to dysfunction of intralaminar thalamic nuclei or a reduction of brain norepinephrine (Mair et al., 1986; Paller, Acharya, Richardson et al., 1997). The possibility that our Korsakoff group may have had a more diffuse attentional deficit was not addressed by the analysis of median scores and cannot be ruled out.

The results from the allocentric and egocentric conditions showed that the Korsakoff amnesics were equally and severely impaired in the two conditions and, in both cases, they appeared to lose information at an accelerated rate. This latter finding is consistent with related studies of Korsakoff amnesics (Holdstock et al., 1995; Downes, Holdstock, Symons et al., 1998) which have interpreted accelerated forgetting as indicating a deficit in memory consolidation (Downes et al., 1998; Mayes, 1995). In the present case scaling problems, due to unmatched control and amnesic performance at the initial delay, make it difficult to determine whether the Korsakoff forgetting rate is truly accelerated. However, if this accelerated forgetting is genuine, and evidence from previous studies (Holdstock et al., 1995; Downes et al., 1998) indicates that this is likely, then this patient group has a memory impairment for allocentric and egocentric spatial information in addition to a possible encoding deficit suggested by their performance on the short delay tasks. Evidence suggests that an attentional deficit does not necessarily lead to accelerated forgetting of verbal and nonverbal material. Cahn and Marotte's (1995) study of children with Attention Deficit Hyperactivity Disorder showed that, although this disorder impairs attention and organisation of information, which impaired nonverbal memory when tested immediately, forgetting of verbal and nonverbal material was not accelerated. We, therefore, consider that, although the Korsakoff patients may have shown poor encoding of position information, their pattern of performance on the allocentric and egocentric spatial memory tasks provides important information concerning these two types of spatial memory in this patient group. Unlike two patients with medial temporal lobe damage, NM (referred to in this paper) and YR (Holdstock et al., submitted), who were only significantly impaired on the allocentric spatial task, the Korsakoff patients were impaired and showed apparent accelerated forgetting on both the allocentric and egocentric spatial memory tasks.

Data from animal studies suggest that the hippocampus, mamillary bodies and anterior thalamic nuclei form a critical system necessary for allocentric spatial memory (Aggleton and Saunders, 1997). This suggests that the Korsakoff amnesics' impairment on the allocentric spatial task may be associated with abnormalities in the mamillary bodies which invariably show pathology in this disease. While it is possible that egocentric memory is also dependent on the mamillary bodies, animal research has not implicated them in this aspect of spatial memory. Korsakoff's syndrome has been recently found to result in dysfunction to a number of brain structures (Kopelman, 1995; Paller et al., 1997)

beyond those observed in the classic neuropathology. PET has revealed pathology of the frontal and parietal cortices of Korsakoff patients (Paller et al., 1997) and both of these regions have been implicated in egocentric spatial processing (Fuster, 1980; McDaniel, Via, Smith et al., 1995; Niki, 1975; Pohl, 1973; Save and Moghaddam, 1996; Semmes et al., 1963). This may account for the egocentric memory deficit. Although results of our correlational analysis did not support a frontal explanation, the modest size of the amnesic group did limit the power of such analyses.

The results of the patients with medial temporal lobe damage, RS and NM, will now be considered. These two patients were both unimpaired on the 'short delay' condition of the spatial memory task suggesting that encoding of spatial information and working memory for spatial information remain intact following bilateral medial temporal lobe damage. This finding is consistent with a number of studies reporting normal encoding of spatial information following medial temporal lobe damage (Mayes, Downes, Shoqeirat et al., 1993; Warrington and Baddeley, 1974), bilateral damage restricted to the hippocampus (Cave and Squire, 1992) and right temporal lobectomy, including the hippocampus (Smith and Milner, 1989).

Both patients RS and NM were impaired on the allocentric spatial memory task but only RS was impaired on the egocentric task. The control subjects' performance did not differ significantly on the two tasks but, if anything, they found the allocentric task slightly easier than the egocentric task. Therefore, greater impairments on the allocentric than the egocentric task cannot be attributed to the relative difficulty of the tasks. The impaired performance of patients RS and NM on the allocentric task suggests that medial temporal lobe structures are critical for this aspect of spatial memory. This result is consistent with animal studies (Feigenbaum and Rolls, 1991; Jarrard, 1993; Morris et al., 1982; Muller et al., 1987; O'Keefe, 1991; O'Keefe and Conway, 1978, 1980; O'Keefe and Dostrovsky, 1971; O'Keefe and Nadel, 1978; O'Keefe and Speakman, 1987) and human studies (Abrahams et al., 1997; Goldstein et al., 1989; Holdstock et al., submitted; Maguire, Burgess, Donnett et al., 1998; Maguire, Burke, Phillips et al., 1996; Maguire, Frackowiak and Frith, 1996, 1997; Morris et al., 1996) which have implicated the medial temporal lobe, including the hippocampus, in allocentric spatial memory. The severity of their allocentric spatial memory deficit was no greater than that shown on the same task by patient YR (Holdstock et al., submitted), who had relatively selective hippocampal damage. This result was particularly interesting in the light of a recent study (Bohbot et al., 1998) which found that right parahippocampal lesions, but not right hippocampal lesions, impaired performance on a human analogue of the Morris Water maze which was probably tapping allocentric spatial information. That study suggested that under some circumstances the parahippocampal cortex itself may play a critical role in spatial memory which is not shared by that part of the hippocampus which was damaged in Bohbot et al.'s right hippocampally lesioned patients. However, the results from our study suggested that this was not the case for the allocentric and egocentric spatial memory tapped by our tasks. Our results showed that additional damage to the parahippocampal gyrus did not accentuate the allocentric memory deficit for

place information found after more selective hippocampal damage which affected the entire length of the hippocampus. This finding is consistent with a serial relationship between the parahippocampal gyrus and the hippocampus which is suggested by anatomical evidence (Aggleton and Brown, 1999).

Both patients showed accelerated forgetting of the allocentric spatial information. This pattern of results suggests a deficit in the consolidation of visuo-spatial information which is consistent with the hypothesis that the hippocampus mediates the consolidation and storage of associative information (Mayes, 1995). It might be argued that, given the very short initial delay, the pattern of scores could also be interpreted in terms of the differential contribution to performance of intact short-term memory and impaired long-term memory at different delays. This latter explanation is unlikely, at least for patient NM, whose performance was still matched to the controls at 20 seconds, but who showed accelerated forgetting between this delay and 60 seconds. Although the duration of visual short-term memory has not been established, the duration of verbal short-term memory was estimated by experimental methods to be two seconds or less (Muter, 1980) when rehearsal is not possible and amnesic patients with normal short-term memory (assessed by digit span) were severely impaired at verbal recall after a filled delay of only 15 seconds (Isaac and Mayes, 1999). These two sources of evidence indicate that the duration of verbal short-term memory is less than 15 seconds. Therefore, if similar mechanisms underlie visuo-spatial short-term memory it is unlikely that it is still contributing to performance after 20 seconds (see Downes et al., 1998; Isaac and Mayes, 1999, for a discussion of these issues). If this is the case, the accelerated forgetting shown by NM is most likely to be due to a consolidation deficit.

The discrepancy in the pattern of performance of the two patients with medial temporal lobe damage on the egocentric task may be attributed to their different patterns of brain pathology which have been revealed by detailed investigation of their structural MRI scans. A number of brain regions were found to be damaged to a greater extent in RS than NM and so may account for RS's impairment on the egocentric task. (1) Within the medial temporal lobes, the anterior part of the hippocampus was found to be smaller in RS than NM. (2) RS had more cortical atrophy than NM, particularly of the temporal lobes. (3) He also had less white matter than NM which may suggest poorer transmission of information between brain regions in RS than NM. (4) RS's lateral and third ventricles were enlarged which may suggest damage to diencephalic structures such as the thalamus. Surprisingly, there was no evidence that RS had more atrophy than NM to the brain regions which have been previously associated with egocentric spatial processing in the animal literature, that is, the frontal cortex and parietal cortex (Fuster, 1980; McDaniel, Via, Smith et al., 1995; Niki, 1975; Pohl, 1973; Semmes et al., 1963). In fact, RS had much less atrophy to the parietal lobe than NM who was unimpaired on the egocentric task. RS's egocentric spatial memory deficit may therefore be due to damage to the anterior hippocampus, atrophy of the temporal lobes, reduction of white matter or possibly damaged diencephalic structures.

Patient NM's unimpaired performance on the egocentric condition was consistent with previous animal studies which have indicated a much larger

involvement of the hippocampus in allocentric than egocentric spatial memory (Feigenbaum and Rolls, 1991; Jarrard, 1993; Morris et al., 1982; Muller et al., 1987; O'Keefe, 1991; O'Keefe and Conway, 1978, 1980; O'Keefe and Dostrovsky, 1971; O'Keefe and Nadel, 1978; O'Keefe and Speakman, 1987) and studies of patients which have also implicated the right temporal lobe (Goldstein et al., 1989) and the hippocampus bilaterally (Holdstock et al., submitted) in allocentric but not egocentric spatial memory. Previous work suggests that the parahippocampal gyrus may be necessary when the positions of particular objects are encoded within an egocentric framework (Milner et al., 1997; Owen et al., 1995). However, NM's results suggest that the hippocampus, the amygdala and the parahippocampal gyrus including the perirhinal cortex and entorhinal cortex are not necessary for normal egocentric spatial memory when position alone has to be remembered. An interesting finding to arise from NM's data was that considerable atrophy to the parietal lobes was not sufficient to impair egocentric spatial memory. This finding appears inconsistent with previous studies which have implicated the parietal lobe in egocentric spatial memory (e.g. Berthoz, 1997; McDaniel, Via, Smith et al., 1995; Save and Moghaddam, 1996). However, focal damage is much more likely to cause serious disruption of neural circuits than atrophy and our results do not exclude the possibility that more focal lesions to the right areas of the parietal cortex would cause such a memory impairment.

The results from the allocentric and egocentric spatial memory tasks indicate a possible dissociation between amnesia due to Korsakoff's syndrome and medial temporal lobe damage. A number of studies have reported no significant difference between the disproportionately impaired performance of Korsakoff and medial temporal lobe amnesic patients on spatial memory tests (Kopelman, Stanhope and Kingsley, 1997; Mayes et al., 1991; Shoqeirat and Mayes, 1991). However, Chalfonte, Verfaellie, Johnson et al. (1996) report that when object recognition performance was matched to that of the controls, memory for object position was not disproportionately impaired in Korsakoff patients but was disproportionately impaired after medial temporal lobe damage when tested incidentally. Kopelman et al. (1997) also report a trend for their Korsakoff patients to be less impaired at object-position memory than medial temporal lobe amnesics when matched to controls for object recognition. The difference between Korsakoff and medial temporal lobe patient performance in the present study was in the opposite direction to this. The Korsakoff patients showed a considerable impairment of both allocentric and egocentric spatial memory whereas medial temporal lobe damage only consistently impaired allocentric spatial memory. These results suggest that Korsakoff patients are not less impaired than medial temporal lobe amnesics on all spatial memory tasks and that this patient group may actually have additional impairments affecting performance on the egocentric spatial memory task and encoding.

To summarise, allocentric and egocentric spatial memory were assessed separately in amnesic patients with either medial temporal lobe or diencephalic damage. The main findings of the study suggested that medial temporal lobe damage does not affect short-term spatial memory, but it does impair memory for allocentric spatial information. However, this impairment appeared to be no greater than that found following more selective hippocampal damage

(Holdstock et al., submitted). The effect of medial temporal lobe damage on egocentric spatial information needs to be clarified by testing further patients in whom we can be confident that dysfunction is restricted to the medial temporal lobes, but the present findings suggest that these structures, other than possibly the anterior hippocampus, may not be critical for egocentric spatial memory. The role of the diencephalic structures in allocentric and egocentric spatial memory is still to be fully determined. Our results suggest that Korsakoff amnesics are equally impaired under allocentric and egocentric spatial memory task conditions, but interpretation of the results is complicated by the possibility of dysfunction to brain areas other than the diencephalic region (Paller et al., 1997). It will, therefore, be critical to test patients with selective damage to diencephalic structures such as the mamillary bodies and anterior thalamic nuclei in order to further illuminate the role of these structures in allocentric and egocentric spatial memory.

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