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A comparison of egocentric and allocentric spatial memory in a patient with selective hippocampal damage

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Abstract

The spatial memory of a single patient (YR) was investigated. This patient, who had relatively selective bilateral hippocampal damage, showed the pattern of impaired recall but preserved item recognition on standardised memory tests that has been suggested by Aggleton and Shaw [Aggleton JP, Shaw C. Amnesia and recognition memory: a reanalysis of psychometric data. Neuropsychologia 1996;34:51–62] to be a consequence of Papez circuit lesions. YR was tested on three recall tests and one recognition test for visuospatial information. The initial recall test assessed visuospatial memory over very short unfilled delays and YR was not significantly impaired. This test was then modified to test recall of allocentric and egocentric spatial information separately after filled delays of between 5 and 60 s. YR was found to be more impaired at recalling allocentric than egocentric information after a 60 s interval with a tendency for the impairment to increase up to this delay. Recognition of allocentric spatial information was also assessed after delays of 5 and 60 s. YR was impaired after the 60 s delay. The results suggest that the human hippocampus has a greater involvement in allocentric than egocentric spatial memory, and that this most likely concerns the consolidation of allocentric information into long-term memory rather than the initial encoding of allocentric spatial information. The findings also suggest that YR's item recognition/free recall deficit pattern reflects a problem retrieving or storing certain kinds of associative information.

Keywords: Amnesia; Hippocampus; Recall; Recognition; Encoding; Forgetting

1. Introduction

Patients with medial temporal lobe damage suffer anterograde amnesia which includes impairments of spatial memory [8,10,24,27,31,39,58,68–70,74]. Animal studies indicate that within this region the most critical structure for spatial memory is the hippocampus. Indeed, one of the major theoretical models of the role of the hippocampus in animals is the cognitive mapping theory [50,55]. This theory largely arose from single cell recording studies in rats that identified 'place cells' [49,53]. These cells respond when the animal is in a particular place within an environment and, in some cases, they are insensitive to the direction in which the animal is facing. The cognitive mapping theory proposes that the hippocampus is critical for forming a representation of a place in the environment when this is identified by the relative position of an array of external stimuli or landmarks (i.e., using an allocentric frame of reference), but not when a location is identified by its relative position to the observer (i.e., using an egocentric frame of reference).

Subsequent single cell recording studies in rats have confirmed the presence of place cells in the hippo-

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campus [45,50,51,55] and evidence from lesions of the hippocampus [26,43] and fornix [52] of rats has provided further support for the cognitive mapping theory. The maintenance of place cell firing following removal of cues which define the environment has emphasised the mnemonic role of the rat hippocampus in processing allocentric spatial information [55]. In contrast to the rat studies, place cells have rarely been identified in monkeys as few studies have made recordings while the monkey has been moving freely within the environment. One such study [56] did identify place cells in the monkey hippocampus. However, a similar study which used a less complex environment and involved passive movement of the monkey failed to find such cells [64]. Instead cells were found within the hippocampus that responded to views and one cell which responded to a combination of view and place. Similar cells were identified in an earlier study [14] in which the monkey viewed stimuli presented in different positions on a screen. In that study the majority of these view cells were reported to code in allocentric coordinates. A further study of monkeys identified cells within the hippocampus whose response was dependent on the direction of auditory and visual stimuli [71]. Some of these cells were found to code in allocentric coordinates but others coded in egocentric coordinates. The evidence from monkey studies that the hippocampus plays a critical role in allocentric but not egocentric spatial memory is, therefore, less clear-cut than that from rat studies.

It is clearly of theoretical importance to determine the role of the human hippocampus in allocentric and egocentric spatial memory. Within the human spatial memory literature there have been relatively few attempts to develop spatial memory tests which emphasise the use of either an allocentric or an egocentric spatial frame of reference. Support for the role of the human hippocampus in allocentric spatial memory has come from a small number of studies which have found deficits in this aspect of memory following unilateral temporal lobectomy or unilateral hippocampal sclerosis [18,34] particularly on the right [1,13,44]. Further, one of these studies [18] 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) [33,35,36], a task which is thought to rely on allocentric spatial memory. These studies, therefore, support a role for the human hippocampus in allocentric spatial memory. However, because the majority of studies have not used an appropriate egocentric comparison task, the studies provide limited evidence as to whether the human hippocampus also plays an equivalent role in egocentric spatial memory.

The present study was designed to obtain measures of allocentric and egocentric spatial memory. Spatial recall was tested by requiring the subject to view a single light on a uniform board and then mark the position of the light, following a delay, after it had disappeared. Initially, memory was tested in a condition which allowed the use of both allocentric and egocentric frames of reference after short unfilled delays ('short delay' condition) in order to assess whether spatial information could be successfully encoded. The task was then modified to strongly encourage subjects to use an allocentric spatial reference frame to encode position. The subject viewed the light from one position around the board and then moved to another position before indicating its location. This manipulation, in which the relationship between the observer and environmental cues is changed but the relationship between these cues and the target location is maintained, is based on the manipulations which have been used to assess allocentric spatial memory in animal studies employing the '+' maze [51,55] and the Morris water maze [29,43,65]. Our manipulation is effectively the same as that used by Goldstein et al. [18] and by Abrahams et al. [1] to assess allocentric spatial memory in unilateral temporal lobectomy and hippocampal sclerosis patients. The task was also modified, in a novel way, to strongly encourage subjects to use egocentric spatial memory. In this condition the subject viewed the target light and indicated its location in the dark, a situation in which allocentric cues were eliminated. These allocentric and egocentric conditions used longer filled delays. Finally, a version of the allocentric task was developed to assess yes/no recognition memory.

The present case study reports the results of an individual (YR) who, consistent with [2], showed impaired recall but intact item recognition on standardised memory tests following bilateral damage to the hippocampus. YR was tested on the battery of spatial memory tests described above to compare her allocentric and egocentric spatial memory. The allocentric spatial recognition condition was included to test two possible explanations for YR's pattern of impaired recall but normal item recognition on standardised tests. One possible explanation is that there is an impairment in the processes which underlie recall but which are not involved in recognition. If this explanation is correct YR would be unimpaired on the recognition version of the allocentric condition and on any recognition task. The second explanation is that recall and item recognition differ in the type of information which is retrieved and it is possible that recall depends to a much greater extent than

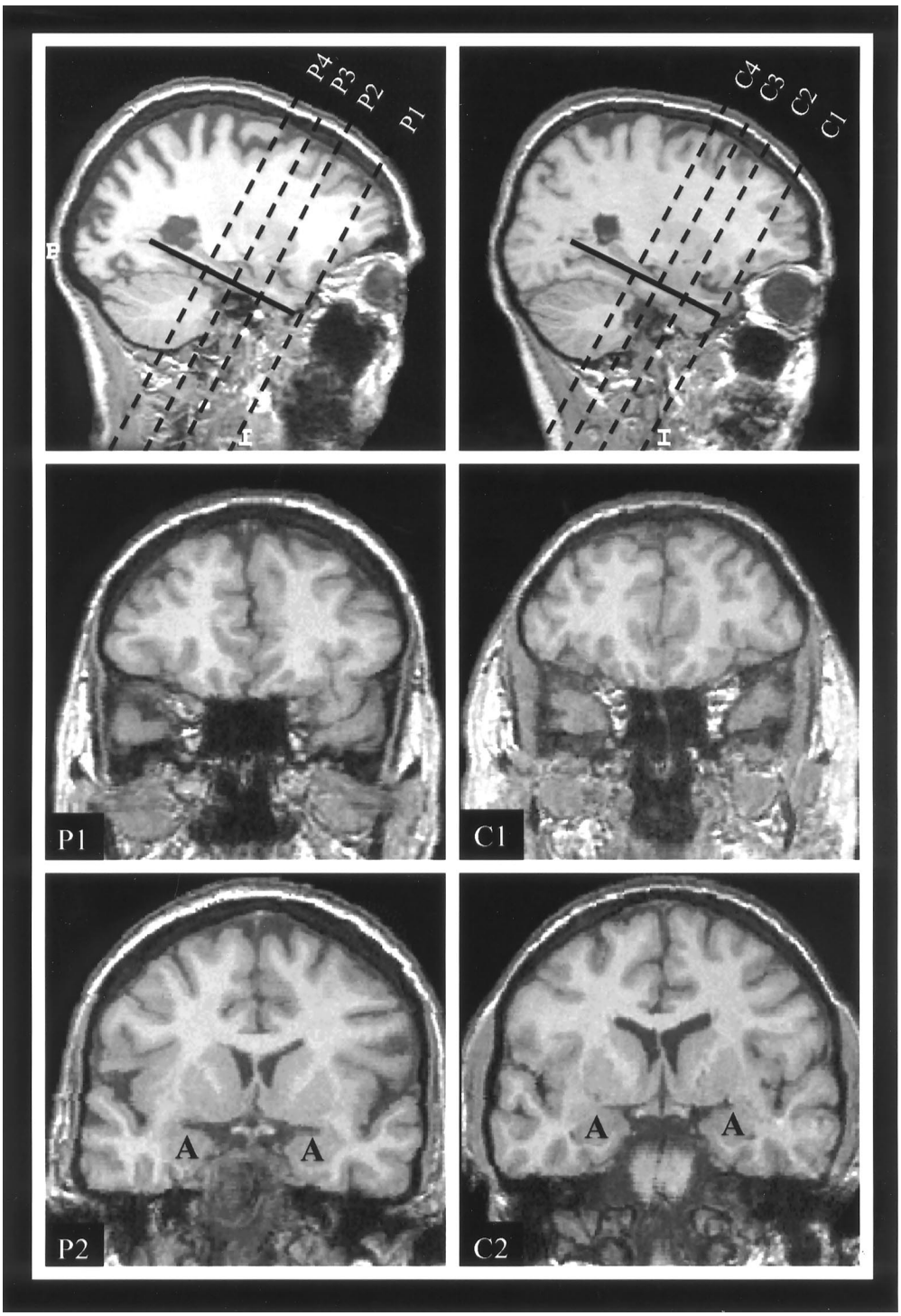

Fig. 1. T1 weighted images of patient YR and one of eight age- and sex-matched control subjects. The images on the left and the right of the figure refer to the patient (P) and the control subject (C), respectively. For each subject the sagittal image shown in the upper panel is marked so as to indicate the location and orientation of the coronal images shown in the lower panels and in Fig. 2. The coronal oblique images show sections through the frontal lobes and in the lower panel the section includes the amygdala (labelled A). YR shows no pathology to these brain regions.

item recognition on retrieving certain types of associative information. As allocentric spatial memory involves the formation and later retrieval of a number of associations between target location and the relative positions of environmental markers in order to create a cognitive map, the second explanation predicts that YR would be impaired on the allocentric recognition task.

2. Method

2.1. Subjects

Patient YR is a female who was 58 years old and working in a clerical position at the time of the main part of the study. She was 60 years old when spatial recognition was tested. In 1986 she had received an opiate drug to relieve a severe back pain and may have then suffered an ischaemic infarct. Immediately following this incident, she suffered a memory impairment which has persisted for 12 years.

2.2. Patient YR: neuropathology

A Magnetic Resonance Imaging (MRI) scan was obtained for patient YR using a 3D T1-weighted radio-frequency spoiled gradient echo (SPGR) sequence [TE = 9 ms, TR = 34 ms, flip angle = 45°, matrix size = 256 × 192, 2 NEX, field of view = 20 cm, acquisition time = 27 min and 52 s] available on a 1.5 T SIGNA whole-body magnetic imaging system (General Electric, Milwaukee, WI). Each image referred to a contiguous section of tissue, 1.6 mm thick. As shown in Fig. 1, there was no evidence of visible frontal lobe damage. There was also no evidence of damage to medial temporal lobe structures other than the hippocampus which was damaged bilaterally (Fig. 1, images P2 and C2; Fig. 2, images P3, P4, C3 and C4). The amygdala was small, but showed no sign of pathology. There was an indication of a small degree of general parietal lobe atrophy, shown in Fig. 3.

In order to obtain quantitative measures of the volume of the hippocampus, and the parahippocampal

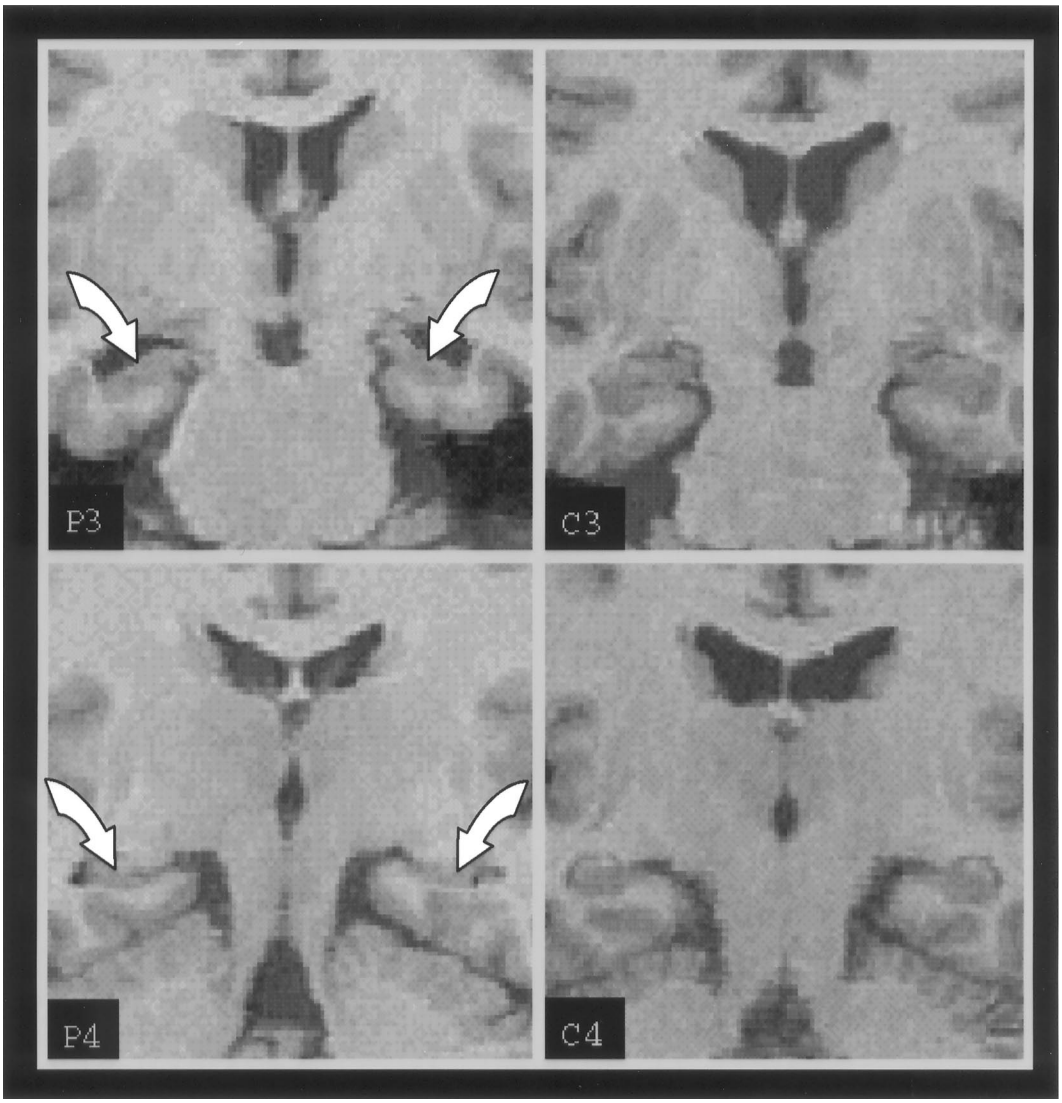

Fig. 2. T1 weighted images of patient YR and one of eight age- and sex-matched control subjects. The left hand images refer to the patient and those on the right to the control. The location and orientation of the images is illustrated in Fig. 1. The upper and lower panels show atrophy of the head and the body of the hippocampus (indicated by arrows), respectively, relative to the control.

gyrus we used the procedure described by Mackay and colleagues [32]. The parahippocampal gyrus measure included the parahippocampal, the entorhinal and the perirhinal cortices plus white matter. Given the proposed role of the frontal and the parietal lobes in egocentric spatial processing [16,41,48,59,66], volume measures of these regions were also obtained. The frontal measure was of the prefrontal lobe which was defined as that part of the frontal lobe which is anterior to the anterior-most point of the corpus callosum when the images are aligned along the AC-PC line. Separate volumes were obtained for grey and white matter in the prefrontal lobe. 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. Structure volumes were estimated using the Cavalieri method of modern design stereology [19,61–63] via stereology menus within Analyze (Mayo Foundation, Minnesota, USA) software running on a SPARC 10 workstation (SUN Microsystems, CA, USA). The estimated structure volumes for patient YR were compared with those of a group of eight healthy female control subjects matched for age and IQ. For each subject the volume of each brain structure was divided by their intracranial volume in order to correct for differences in premorbid brain size. Uncorrected and corrected brain structure volumes are reported in Table 1 for YR and her controls.

The corrected volume of the hippocampus was 2.25 standard deviations and 3 standard deviations smaller than the control mean on the right and left, respectively. In contrast, the corrected volume of the parahippocampal gyrus, which included the perirhinal, entorhinal and parahippocampal cortices, was at least one standard deviation larger than the mean volume of this region in the control group. YR's parietal lobe volume was found to be smaller than, but well within two standard deviations of, the control mean (1.27 and 1.4 standard deviations below the control mean on the

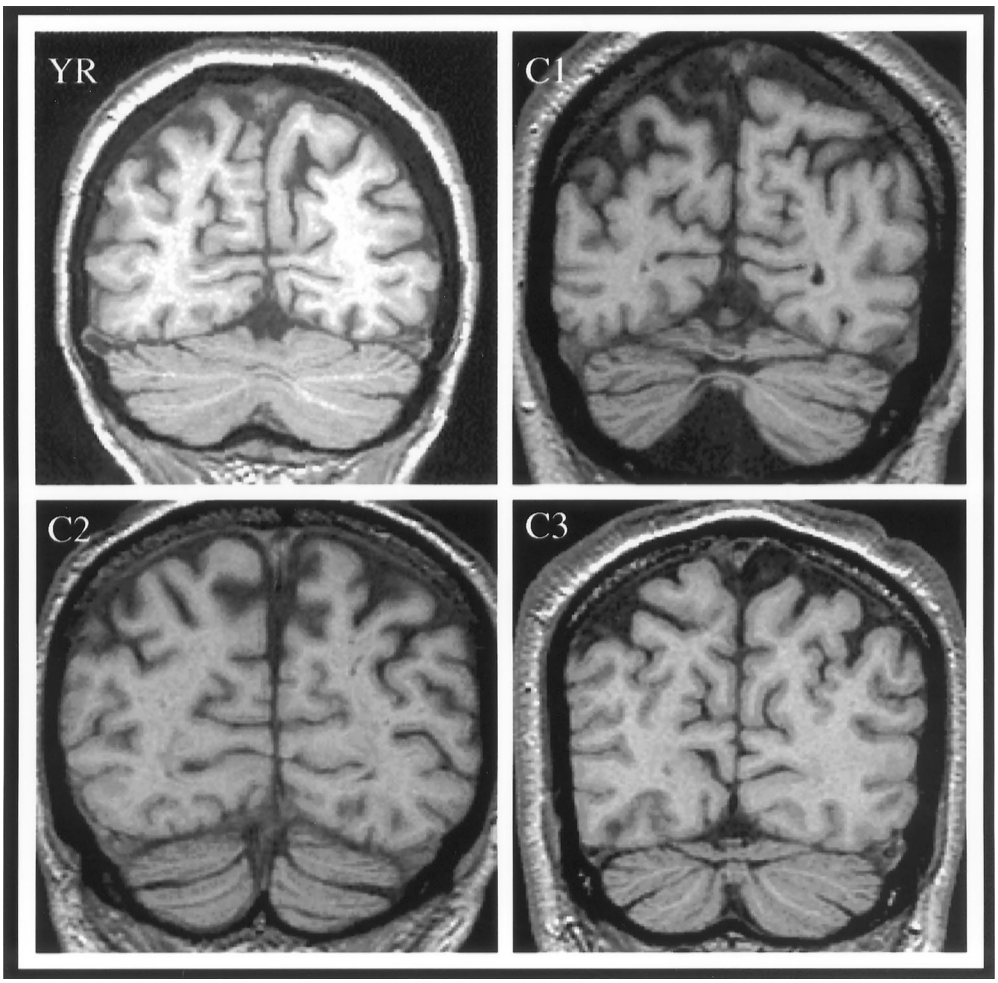

Fig. 3. T1 weighted images of patient YR (labelled YR) and three of eight age- and sex-matched control subjects (labelled C1–C3). The coronal images show a section through the parietal lobe.

Table 1
Volumes of the hippocampus, parahippocampal gyrus, parietal cortex and the grey and white matter of the prefrontal cortex (before and after correcting for intracranial volume) on the right and the left for YR and the mean volumes (standard deviation in brackets) of these regions for a group of eight healthy female control subjects matched for age. Corrected volumes which were two or more standard deviations below the control mean are indicated by an asterisk[a]

	Uncorrected		Corrected	
	Control	YR	Control	YR
R Hipp	2.32 (0.28)	1.10	0.0020 (0.0004)	0.0011*
L Hipp	2.18 (0.24)	0.98	0.0019 (0.0003)	0.0010*
R Parahipp	2.42 (0.42)	2.75	0.0021 (0.001)	0.0027
L Parahipp	2.30 (0.38)	2.86	0.0020 (0.001)	0.0028
R PF GM	34.5 (3.4)	29.05	0.0297 (0.005)	0.0288
L PF GM	34.96 (3.15)	33.1	0.030 (0.0039)	0.0328
R PF WM	20.68 (2.67)	16.07	0.0178 (0.0034)	0.0159
L PF WM	17.64 (3.01)	16.18	0.0152 (0.0029)	0.0160
R Par			2.368 (0.249)	2.050
L Par			2.161 (0.215)	1.859

[a] R Hipp = right hippocampus, L Hipp = left hippocampus, R Parahipp = right parahippocampal gyrus, L Parahipp = left parahippocampal gyrus, R PF GM = right prefrontal grey matter, L PF GM = left prefrontal grey matter, R PF WM = right prefrontal white matter, L PF WM = left prefrontal white matter, R Par = right parietal, L Par = left parietal.

right and left, respectively). Her corrected parietal lobe volume on the right was within the control range and was just below the control range on the left (control range 1.91–2.46). The corrected volume of white and grey matter in the frontal lobe was normal. On the left, the volume of white and grey matter was slightly larger than the control mean. On the right, the volume was 0.19 and 0.56 standard deviations smaller than the control mean for white and grey matter, respectively.

2.3. Patient YR: psychometric assessment

YR was tested on the National Adult Reading Test (NART-R) [47], which requires the pronunciation of irregular words, and the Weschler Adult Intelligence Scale-revised (WAIS-R) in order to assess premorbid and postmorbid IQ, respectively. Memory functions were assessed by: the Recognition Memory Test [73] which tests forced-choice recognition of words and unfamous faces; the revised Weschler Memory Scale which provides measures of immediate and delayed memory for visual and verbal material; and the Doors and People Test [4] which provides separate measures of visual and verbal recall and recognition. Her results on these tests are shown in Table 2. To assess frontal functions YR was also tested on: the Wisconsin Card Sorting Test (WCST) [21] which requires appropriate categorization rules to be determined and changed throughout the test; the Verbal Fluency test (FAS) [5] which requires as many words as possible, beginning with a specified letter, to be generated within a minute; and the Cognitive Estimates Test (CET) [67] in which unknown quantities are estimated, such as the weight of a full pint bottle of milk. The results from these tests are also shown in Table 2.

YR has an IQ which is a little above average and is slightly higher for verbal than performance tests. The difference between her premorbid and present IQ was 13 points which indicates a fall of less than one standard deviation in IQ (one standard deviation on the WAIS-R is 15 points). There was, therefore, no clear indication of a significant decline in IQ in this patient. Her pattern of performance on the three memory tests, which were administered, suggests that her recognition of visual and verbal items is relatively intact, but that recall is impaired. As shown in Table 2, YR's performance was at the fifth percentile or below on the two recall subtests of the Doors and People test and on the general and delayed memory indices of the WMS-R. On the other memory tests, which all tapped item recognition, YR showed no indication of an impairment.

There was no evidence of impairment in YR's

Table 2
YR's performance (score) on psychometric tests measuring premorbid IQ (NART-R) and present IQ (WAIS-R), memory (raw scores on the Recognition Memory Test, index scores on the Wechsler Memory Scale — Revised and raw scores on the Doors and People Test) and executive functions [number of categories and number of perseverative errors on the Wisconsin Card Sorting Test, total word score on the FAS test and score on the Cognitive Estimates Test (CET)]. Percentile scores (perc) are provided for all tests apart from FAS and CET for which YR's performance was compared with a group of matched controls and is reported in the text[a]

	NART	WAIS-R			RMT		WMS-R			D&P				WCST		FAS	CET
	FSIQ	FSIQ	VIQ	PIQ	W	F	GEN*	A/C	DEL*	P**	D	S*	N	CAT	P.ERR		
Score	115	102	108	97	45	48	66	122	73	9	18	22	22	3	6	42	8
Perc.	84	55	70	42	75	> 95	1	93	4	< 1	50	1–5	99	6–10	88		

[a] KEY: FSIQ = full scale IQ, VIQ = verbal IQ, PIQ = performance IQ, RMT = Recognition Memory Test, W = Words, F = Faces, WMS-R = Weschler Memory Scale-Revised, GEN = general memory, A/C = attention/concentration, DEL = delayed memory, D&P = Doors and People, P = People (recall), D = Doors (recognition), S = Shapes (recall), N = Names (recognition), WCST = Wisconsin Card Sorting Test, CAT = number of categories correct, P.ERR = number of perseverative errors. (* = below 5th percentile, ** = below 1st percentile.).

performance on tests of executive function. Performance on the FAS and CET was compared with the performance of a group of 10 healthy female controls matched for age and IQ [mean age 59.6 years (SD = 3.6); mean NART-R 104.8 (SD = 7.15)]. On the FAS test YR's score was 0.11 standard deviations below the control mean of 43.9 and on the CET she was 0.69 standard deviations below the control mean of 6. So there was no indication of an impairment on either of these tests. In comparison to the test norms, the number of categories which YR correctly sorted on the WCST fell between the 6th and the 10th percentile. YR correctly sorted the cards according to the first three categories (colour, form and number) without difficulty but did not achieve any further correct categories because, instead of re-using these three categorisation rules, she attempted to find new, more complex rules, to categorise the remaining cards. This led to her achieving only three correct categories. YR made only six perseverative errors in 128 trials (performance was at the 88th percentile) which gave no suggestion of frontal dysfunction. The overall pattern of YR's executive test performance is consistent with the MRI evidence indicating that the white and grey matter in the prefrontal cortex was intact.

YR's perception of objects and space was assessed with the Visual Object and Space Perception Battery (VOSP) [75], the Judgement of Line Orientation test [6] and the 'Little Men' Test of mental rotation [60], which is discussed in more detail later in the paper. Spatial reasoning was also assessed using the Verbal and Spatial Reasoning Test (VESPAR) [30]. On the VOSP, YR was within one standard deviation of the mean of the normative sample for all subtests and performed better than the controls on one of the four object perception subtests (Silhouettes: determining whether a silhouette represented a real object) and three of the four spatial subtests (Dot Counting: counting the number of dots in a presented array, Position Discrimination: determining whether a dot was positioned centrally or off-centre and Cube Analysis: counting the number of cubes contributing to a 3-D construction which was presented as a line drawing) (see Table 3). On another visuo-spatial perceptual test, the Judgement of Line Orientation test, YR obtained a score of 28 which was better than the control mean score of 22.2 indicating that she also had no impairment on this task. Also, as shown later in the Results section, YR performed better than the control mean on those conditions of the Little Men test which require mental rotation and are sensitive to parietal lobe dysfunction. In addition, as shown in Table 3, YR's spatial reasoning, measured by the VESPAR, was unimpaired. On this test battery YR scored at between the 51st and 75th percentile on the 'spatial odd one' subtest in which the odd one of four shapes had to be selected and scored at above the 75th percentile for all other spatial subtests (selecting the same visuo-spatial manipulation which has been applied to another shape; completing a series formed by three shapes).

Table 3
YR's raw scores (score) and percentile scores (perc.) on the eight subtests of the Visual Object and Space Perception Battery (VOSP) and the three spatial reasoning tests from the Verbal and Spatial Reasoning Test (VESPAR)[a]

	VOSP								VESPAR		
	Object perception				Space perception						
	IL	S	OD	PS	DC	PD	NL	CA	SOO	SA	SS
Score	18	24	16	11	10	20	9	10	16	21	23
Perc.	27	69	18	46	69	66	34	76	51–75	76–90	76–90

[a] VOSP = Visual Object and Space Perception Battery. Object perception tests: IL = Incomplete letters, S = Silhouettes, OD = Object Decision, PS = Progressive silhouettes. Space perception tests: DC = Dot counting, PD = Position Discrimination, NL = Number location, CA = Cube analysis. SOO = spatial odd one, SA = spatial analogy, SS = spatial series.

2.4. Control subjects

YR's spatial memory performance was compared with that of three control groups, one for the recall tasks and one for each of the delays of the recognition task. The control groups overlapped but, because of the duration of the tasks, not all of the subjects were able to attend all experimental sessions. The control subjects for the three recall conditions consisted of 10 females who had a mean age of 57 years (SD = 6.47). YR's age was 0.15 standard deviations above the control mean. Psychometric testing of the control subjects was limited to the NART-R and the mean predicted FSIQ was 97.7 (SD = 10.15). YR's FSIQ from the WAIS-R was 102 which was 0.42 standard deviations above the control mean NART-R FSIQ.

For the 5 s delay of the recognition task the control group consisted of 10 healthy female volunteers who had a mean age of 57 years (SD = 4.72) and a predicted NART-R FSIQ of 104.4. YR's age was 0.55 standard deviations higher than the control group and her FSIQ from the WAIS-R was 0.41 standard deviations below the control mean NART-R predicted FSIQ. For the 60 s delay the group consisted of 10 healthy females who had a mean age of 58 years (SD = 3.69) and a predicted NART-R FSIQ of 109 (SD = 10.85). YR's age was 0.65 standard deviations above the control mean and her FSIQ from the WAIS-R was 0.62 standard deviations below the control mean NART-R predicted FSIQ.

2.5. Apparatus

The test board consisted of a large, 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 25 20 mA standard red light emitting diodes (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.

2.6. Design and procedure

Three different recall tasks and one recognition task were examined. The main design features of each task are summarised in Table 4. In all the 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.

For the recall tasks the subject was instructed to attend to the lightboard, an LED was lit for approximately 2 s and, after a delay, the subject had to mark the location of that LED. Each task consisted of 45 trials in which three different retention durations were presented in a mixed sequence. As there were 25 LEDs on the board and 45 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 was intended to preclude the use of verbal rehearsal and to ensure that it was not possible to fixate on the target region through the delay period.

2.7. Simultaneous

YR and six of the control subjects were tested on a control task to determine whether YR was able to place the counter with the same degree of precision as the control subjects when there was no memory load. For this 'simultaneous condition', the subject viewed the board from the same position as in the 'short delay' condition, 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. Each subject completed 15 trials which used 15 different positions.

2.8. Recall

2.8.1. Short delay recall

In this condition the subject was asked to look at the board and place a counter (22 mm diameter with 2 mm diameter hole in the centre) in the exact position of the light immediately after it disappeared (0 s) or after an unfilled delay of 3 or 8 s during which the subject was instructed to look away from the board. Use of these delays allowed encoding and short-term/working memory for the light positions to be assessed. The subjects did not move during the test procedure. Each control 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. YR completed both of the test sequences with each one being given in a different testing session.

2.8.2. Allocentric recall

The allocentric manipulation was similar to that used by Abrahams and colleagues [1]. Presentation of the LED sample was followed by a filled delay of 5, 20 or 60 s. Approximately 3 s 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. Whilst moving to this new location subjects were not allowed to look at the test board. Once in this new position, subjects placed the

Table 4
Design features of the control task, the three spatial recall tasks and the spatial recognition task on which YR and her matched controls were assessed

Task	View/test position	Test conditions	No. of trials	Delays assessed	No. of runs completed by YR
Control					
Simultaneous	same	daylight	15	No delay	1 (+2 after sight corr.)
Recall					
Short delays	same	daylight	15 per delay	0, 3, 8 s (intermixed)	2 (+1 after sight corr.)
Egocentric	same	dark	15 per delay	5, 20, 60 s (intermixed)	4 (+1 (60 s) after sight corr.)
Allocentric	different	daylight	15 per delay	5, 20, 60 s (intermixed)	4 (+1 (60 s) after sight corr.)
Recognition					
Allocentric	different	daylight	30 per delay	5, 60 s (blocked)	1 (after sight corr.)

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.

2.8.3. Egocentric recall

In the egocentric condition testing took place in a blacked out room with the room lights off. This manipulation ensured that no allocentric cues were visible when the target light was presented. This condition would therefore strongly encourage subjects to remember the position of the light relative to their own position, and by recalling head and eye movements required to bring the light to the centre of vision. Although this information would also be available to subjects in the allocentric task, the design of that task encouraged the use of allocentric cues over this information. 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 produced a 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 *during* a trial, the room lights were turned on between each trial. Although this may have allowed subjects to form an allocentric representation of the room which could have been maintained while the room lights were off, alone, this would have been of limited help to them in performing the egocentric task. This is because, since no allocentric cues were visible during presentation of the LED, the subjects could not have determined the location of the LED within this putative allocentric representation without knowing how the array of room cues was positioned relative to themselves. This would have required memory for the egocentric locations of at least three cues in the allocentric room representation (to anchor the room representation in the three dimensions of egocentric space). Although we cannot rule out the possibility that subjects would use such a strategy, remembering the egocentric location of each single light would be the easier task as it involves remembering less information. Debriefing of control subjects confirmed that they had used an egocentric frame of reference, not an allocentric frame of reference.

For both the allocentric and egocentric tasks, memory was tested after delays of 5, 20, and 60 s. These delays provided a spread of retention intervals from short to long within practical constraints. The shortest delay was determined by the time required to move to a new position round the lightboard in the allocentric task and the longest by the limitations on the length of the test sessions determined by the duration after which it was considered subjects would become bored and no longer concentrate properly. Inclusion of a number of delays allowed forgetting rate for allocentric and egocentric information to be investigated. Each control subject completed just one allocentric and one egocentric sequence. Four trial sequences were constructed which differed in the order in which target positions and delays were tested. Sequences were assigned to subjects such that each sequence was used approximately the same number of times in each condition and each subject received a different sequence for the allocentric and egocentric conditions. YR completed four allocentric sequences and four egocentric sequences to obtain more stable measures of these two aspects of memory. As we were testing a single patient it was possible that, if our findings were based on the results of a single session, they could be biased by factors such as fatigue and lack of attention or concentration due to the subject having an 'off-day' but we would not know that this was the case. By testing YR on multiple sessions, we hoped to ensure that the pattern of performance on the allocentric and egocentric tasks which we report reflects an accurate indication of the effect of her hippocampal damage on these tasks and was affected minimally by other incidental factors. The four different orders of target positions and delays that were used for the control subjects were also used for YR. When testing YR each order was used once in the allocentric condition and once in the egocentric condition but was never used twice within the same testing session. Testing took place over four sessions in each of which one allocentric and one egocentric sequence was completed. The order in which the allocentric and egocentric conditions were completed alternated over sessions so that condition order would not influence the results. For the simultaneous and all recall conditions the position of the centre of the counter (or circle of light in the egocentric task) was marked on a piece of removable Perspex so that the distance of the recalled position of the target LED from its actual position could be measured.

Two years after testing on the recall tasks had been completed YR was found to have myopia. Testing on the simultaneous condition, short delay condition and the 60 s delay of the allocentric and egocentric tasks was therefore repeated after her sight had been corrected to determine whether impaired vision had contributed to her original performance on these tests.

In addition to the recall tasks, YR and matched controls were tested on a yes/no recognition version of the allocentric condition. In this condition, subjects viewed the presentation of the target light from one

position around the board and then, following a filled delay, were instructed to move to a different specified position. Once subjects were in the new position, a test light was presented which was either in the same or a different position to the one which had been presented at study. Subjects responded 'yes' if the light was in the same position and 'no' if it was in a different position. Subjects completed 30 trials with a 5 s delay and 30 trials with a 60 s delay in separate test sessions. The same filler task was used as for the recall conditions. YR completed this task after eyesight correction.

Finally, as it could be argued that mental rotation may contribute to performance on the allocentric spatial recall and recognition tasks, YR and a group of 10 healthy age and IQ matched female controls (mean age 57 years, SD = 2.99; mean NART-R FSIQ 108, SD = 8.9) were tested on the 'Little Men' Test of mental rotation [60]. This test requires subjects to indicate whether the left or right hand of a line drawn figure is marked when the figure is upright and facing towards (A) or away (B) from the viewer or inverted and facing towards (C) or away (D) from the viewer. Patients with right parietal lobe damage have been shown to make more errors than controls when the figure is inverted [60], and therefore requires mental rotation. YR and the control subjects completed eight trials from each condition using the procedure described by Ratcliff [60].

3. Results

The results from the recall tasks were based on the distance of the recalled target location from the actual target location, and for each condition the mean spatial error was calculated for each delay. For each delay, YR's score on single tests was considered to be impaired if it was more than 1.96 standard deviations (SDs) worse than the control mean giving a type 1 error probability of 0.05, two tailed.

3.1. Simultaneous condition

When first tested in the 'simultaneous condition', YR was impaired and was on average 2 mm less accurate than the control mean at placing the counter over the target LED (see Table 5. Following sight correction for myopia, this deficit in fine discrimination was no longer present. YR's performance, with corrected vision, was within 2 SDs (1.6 SDs and 1.8 SDs) of the control mean. As shown in Table 5, her error scores on these two retests were 0.35 cm and 0.36 cm which were under 1 mm larger than the control mean (0.27 cm).

Table 5
Mean scores of the control subjects, with standard deviations in parentheses, and patient YR on the simultaneous condition and the four spatial memory tests. For the simultaneous condition and the three recall tests the presented score is the mean distance from the correct location. For all conditions control performance is based on one run through the task. YR's performance on the short delay test was based on two runs through the task and her scores on the allocentric and egocentric tasks is based on four runs through the task. For each task the scores reported for YR are the mean distance from the correct location when originally tested, and her performance on a retest which took place after her sight had been corrected for myopia (labelled as 'retest' in the table). For the recognition task d′ scores are presented

Control			
Simultaneous			
Control group	0.27 (0.05)		
YR			
Run 1	0.44		
Run 2 retest	0.35		
Run 3 retest	0.36		
Recall			
Short delays	0 s	3 s	8 s
Control group	1.42 (0.55)	2.5 (0.47)	2.84 (0.36)
YR	2	3.1	3.4
YR retest	1.93	2.47	4.26
Egocentric	5 s	20 s	60 s
Control group	7.34 (1.96)	8.3 (3.04)	10.37 (2.15)
YR	7.9	12.82	13.3
YR retest	No retest	No retest	14.1
Allocentric	5 s	20 s	60 s
Control group	6.69 (1.26)	7.55 (1.17)	9.44 (2.13)
YR	7.36	11.4	17
YR retest	No retest	No retest	22.3
Recognition			
Allocentric	5 s	60 s	
Control group			
d′	2.15 (0.89)	2.25 (0.68)	
YR			
d′	1.05	0	

3.2. 'Short delay' condition

YR was not significantly impaired in the 'short delay' condition before her eyesight had been corrected. Her scores were 1.05, 1.28 and 1.56 SDs below the control mean at the delays of 0, 3 and 8 s, respectively (see Table 5). Her performance improved slightly following sight correction for the 0 and 3 s delays, for which her scores were 0.93 SDs worse and 0.06 SDs better than the control mean, respectively; however she was worse than on original testing after the 8 s delay, as her score was 3.9 SDs worse than the control mean (see Table 5).

3.3. Allocentric and egocentric conditions

Table 5 shows the means, for each delay, of YR's scores from four runs through the allocentric and egocentric tasks (prior to eyesight correction) along with

the mean scores of the control group. Table 5 also shows YR's performance at the 60 s delay after eyesight correction. Her pattern of performance on the allocentric and egocentric tasks was identical on this retest to that found in the earlier test sessions and so will not be discussed further here. The following analyses all refer to data collected in the four main test sessions. A 3 × 2 (delay × task) ANOVA comparing the control subject performance on the two tasks showed that this did not differ significantly. The main effect of task ($F(1,18) = 1.13$, $p > 0.05$) and the task by delay interaction ($F(2,36) = 0.04$, $p > 0.05$) were not statistically significant. Fig. 4 shows, in standard deviation units, YR's mean performance over the four runs of the allocentric and egocentric conditions. The pattern of her results was very clear. YR's performance was within 2 SDs of the control mean for the egocentric task at all delays (0.29 SDs below the control mean at 5 s, 1.49 SDs below the control mean at 20 s, 1.36 SDs below control mean at 60 s) whereas for the allocentric task her performance was unimpaired at the 5 s delay (0.53 SDs worse than the control mean), but impaired at delays of 20 s (3.29 SDs worse than the control mean) and 60 s (3.55 SDs worse than the control mean). On the allocentric task, YR showed differences in performance between the 5 and 20 s delays and between the 20 and 60 s delays which were 1.96 SDs and 1.64 SDs greater than the control mean difference, respectively. YR showed some variation in performance on both the allocentric and egocentric tasks between test sessions but this was quite small. For example, for the 60 s delay the standard deviation of YR's accuracy scores from the four sessions was 1.6 cm in the allocentric and 2 cm in the egocentric task. This variation was most likely to be due to variations in fatigue, attention or concentration. Despite some variation in absolute performance, the pattern of YR's performance was consistent across test sessions, i.e. in each session, after a 60 s delay, YR was less accurate in the allocentric than the egocentric condition.

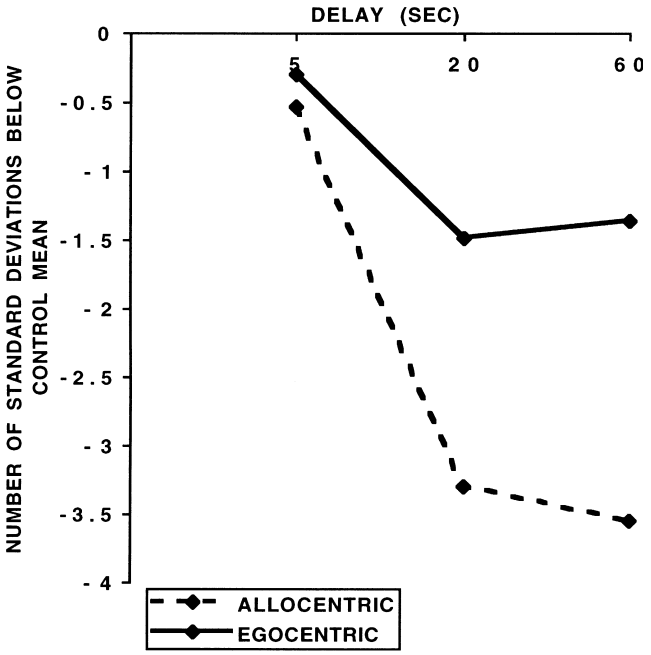

Fig. 4. Graph showing the number of standard deviations that YR's mean error score (based on four runs of each condition) was below the control mean error score for each of the three delays (5, 20, 60 s) of the allocentric and egocentric conditions of the spatial memory recall task.

3.4. Allocentric recognition condition

From the proportion of hits and false alarms, d′ [17] was calculated for each control subject and for YR. Using this d′ measure of performance, YR was not significantly impaired when tested after a delay of 5 s; YR's d′ was 1.23 SDs below that of the controls. In contrast, when tested after a delay of 60 s YR's d′ was 3.31 SDs below the mean d′ of the control group. Three control subjects completed both the 5 s and 60 s delay conditions. The mean difference between the d′ score obtained at the two delays was −0.06 (SD = 0.59) for these three subjects. YR's performance drop from 5 to 60 s was 1.78 SDs greater than that of the controls. Although this score does not exceed our criterion for impairment, at 60 s YR had reached chance performance and it was, therefore, not possible for her performance to fall any further. Due to this, it is highly likely that YR's pattern of performance indicates accelerated forgetting on this allocentric spatial recognition memory task.

3.5. Mental rotation

YR's performance on the mental rotation task was not impaired compared with controls in any of the four conditions. In condition A (upright and towards) YR scored 1 SD worse than the control mean. However, in condition B (upright and away), condition C (inverted and towards) and condition D (inverted and away) she scored 0.5 SDs, 0.4 SDs and 0.05 SDs *better* than the control mean, respectively.

4. Discussion

The results from the present study showed that a patient (YR) with bilateral, relatively selective hippocampal damage was more impaired at recalling the position of a single light at a 60 s delay under conditions which strongly encouraged the use of an allocentric frame of reference than under conditions which forced the use of an egocentric frame of reference.

Taking 1.96 SDs below the control mean as our criterion for impairment, her recall of egocentric spatial information was not significantly impaired. In contrast, YR was significantly impaired on the allocentric spatial recall task at delays of 20 and 60 s and this impairment became greater with increasing delay, indicating that allocentric spatial recall declined abnormally quickly. These results clearly indicate that the hippocampus plays a greater role in allocentric than egocentric spatial memory.

Our results also suggest that the hippocampus is necessary for *recognition* of allocentric information, at least after a 60 s delay. YR was impaired, and in fact performing at a chance level, on the recognition version of the allocentric task after a filled delay of 60 s. In contrast, her performance was not significantly impaired and within the normal range after a retention delay of 5 s. This latter finding indicates that YR's chance performance after the 60 s delay could not be due to a misunderstanding of the task instructions and instead, consistent with all other data we have collected, was due to a memory deficit. In addition, this pattern of data demonstrated that with recognition, as with recall, YR forgets allocentric spatial information at an accelerated rate.

The difference in the extent of YR's impairment on the allocentric and egocentric tasks could not be explained by her very small placing error (0.2 cm greater than the controls) which was obtained on initial testing in the simultaneous condition. Subsequent retesting, after YR's sight had been corrected for myopia, suggested that this impairment was due largely to her slightly compromised vision. Following sight correction, she was no longer significantly impaired in the simultaneous condition and yet she showed the same pattern of very impaired allocentric spatial memory, but not significantly impaired egocentric spatial memory, that she had shown on original testing.

It might be argued that one strategy which may be adopted in the allocentric spatial memory task is to mentally rotate the board or the subject to the presentation position so that the task becomes one involving egocentric spatial memory. YR's greater impairment in the allocentric condition could then be interpreted as resulting from a deficit in her ability to mentally rotate stimuli, perhaps related to her slight parietal lobe atrophy. This argument predicts that the allocentric condition should be harder than the egocentric condition for healthy control subjects because successful performance on it requires both mental rotation and egocentric memory. Our data suggest that it is unlikely that this strategy was used, at least by the control subjects in the present study, as their performance was slightly better on the allocentric task. The mental rotation explanation of YR's results is also highly unlikely. YR's scores were better than the control mean on the critical conditions of the 'Little Men' Test [60] which has been shown to be sensitive to the mental rotation impairments caused by right parietal lobe damage [60]. This result shows that YR is at least as good as the controls at mental rotation and so her allocentric spatial memory scores cannot be attributed to mental rotation problems. We are, therefore, confident that YR's deficit in performance on the allocentric task is due to an allocentric spatial memory deficit.

Developing tasks which exclusively tap either allocentric or egocentric spatial memory is difficult, as subjects probably retrieve a combination of allocentric and egocentric information in order to remember spatial positions in most situations. Many published tasks in the literature which are reported to be allocentric, such as navigation in a real or virtual world environment [33–36], could be solved using egocentric cues such as remembering that a particular sequence of turns from a particular landmark will result in arriving at another particular location or remembering that, for example, when facing the church the chemist was to your left. However, in complex environments allocentric strategies may predominate because using egocentric information may become very difficult (e.g. remembering a long series of directions). Most tasks which have tried to tap allocentric or egocentric spatial memory (including our own) should, therefore, be considered as strongly encouraging the use of one of these spatial reference frames over the other rather than providing pure measures of allocentric or egocentric memory.

Although our results clearly demonstrate that the hippocampus has a much larger involvement in allocentric than egocentric spatial memory, they do not allow us to draw conclusions as to whether the hippocampus plays *any* role in egocentric spatial memory. On the egocentric task YR's performance was consistently below the control mean but, as the performance of about half the control subjects was also below average, this does not of itself indicate an impairment. What is critical is whether her performance was sufficiently below the control mean to allow one to reject the hypothesis that her scores came from the same population of scores as the controls. By convention this hypothesis is rejected if, when the hypothesis is true, the probability of obtaining a score that extreme is less than 0.05, two-tailed. This corresponds to a z-score of greater than 1.96. Using this criterion, YR's mean performance over four runs through the egocentric condition was not significantly impaired at any delay. Therefore it is entirely possible that YR's below average performance on this task may reflect a below average premorbid egocentric spatial memory ability. However, we cannot rule out the possibility

that YR has a mild postmorbid impairment in this aspect of spatial memory. This may be due to hippocampal damage but, as is always the case when studying patients with brain lesions, there may be damage to additional regions which structural MRI has not detected [e.g. 57].

If YR's below average performance on the egocentric task *is* reflecting a mild impairment of egocentric spatial memory, this could indicate a role for the hippocampus in this aspect of memory and would be consistent with some studies [14,64,71] which have identified cells in the primate hippocampus that appear to use egocentric spatial coordinates. However, there is some evidence from human studies which suggests that medial temporal lobe lesions may leave egocentric spatial memory intact at least at short delays. First, we have studied another patient (NM) who has more extensive medial temporal lobe damage which includes the parahippocampal, perirhinal and entorhinal cortices, in addition to the hippocampus, and found that his egocentric spatial memory was normal (0.14 SDs better than control performance) after a 60 s delay [23]. Second, Warrington and Baddeley [74], using a lightboard task similar to ours, found that patients, some of whom almost certainly had large medial temporal lobe lesions comparable to our patient NM, showed normal spatial memory over filled delays of up to 60 s. Warrington and Baddeleys' task [74], like our 'short delay' condition, would have allowed the use of both allocentric and egocentric spatial memory but, given that the viewer's position was constant, it is possible that primarily egocentric spatial memory was used. These data suggest that egocentric spatial memory is not necessarily impaired following medial temporal lobe lesions even when these include not just the hippocampus, but also the parahippocampal, entorhinal and perirhinal cortices.

MRI and behavioural evidence suggests that any mild impairment of egocentric spatial memory cannot be attributed, in patient YR, to mild parietal lobe atrophy. The parietal lobe has been associated with the processing of egocentric spatial information in the animal literature [3,7,28,41,65]. However, the volumes of YR's parietal lobes, corrected for intracranial volume, were not significantly smaller than the volumes of age and sex matched controls. In fact, the volume of her parietal lobe on the right, which is the side which has been related to spatial processing [e.g. 15,60] and some aspects of spatial memory [11], was within the range of the volumes of the eight control subjects. Psychometric testing also revealed no evidence of a spatial processing deficit. Further, patient NM, who is described above, had considerably more parietal lobe atrophy than YR and yet was unimpaired on the egocentric task. NM's parietal lobe volume, after correcting for individual differences in premorbid brain volume, was 7.8 SDs smaller than his matched controls on the right and 6.1 SDs smaller than his matched controls on the left. In comparison, YR's corrected parietal lobe volumes were 1.3 and 1.4 SDs smaller than controls on the right and left, respectively. Despite his parietal lobe atrophy, NM performed better than his control group mean at delays of 5 and 60 s on the egocentric task and was 0.3 SDs below the mean at the 20 s delay. NM's data show that atrophy of the parietal lobe is not sufficient to impair egocentric spatial memory as measured by this task and so it is unlikely that YR's very mild atrophy would have affected her performance on the egocentric task.

The resolution of the question of whether the hippocampus plays any role in egocentric spatial memory will depend on testing more patients like YR who have selective bilateral damage to the hippocampus or the fornix. If damage to these structures causes only a mild egocentric memory deficit, it may not be detected as a significant impairment in individual patients, but would be shown by consistent below control level performance in a group of such individuals. Although more work is needed to determine whether the human hippocampus plays any role in egocentric spatial memory, the results from patient YR clearly indicate that it is much more necessary for allocentric spatial memory.

YR's pattern of performance on these spatial memory tasks raises a number of points for discussion. First, consistent with previous literature [24,39,58,68–70], the results suggest that the hippocampus plays a crucial role in spatial long-term memory. Further, the results suggest that the hippocampus is much more critically involved in the recall and recognition of allocentric spatial information than the recall of egocentric spatial information. The impairment of allocentric recall and recognition is consistent with other human studies [1,13,18,34,44] and with animal studies [14,51,54,55] which have implicated the hippocampus or medial temporal lobe in allocentric spatial memory. It is also consistent with the results of human PET studies which have reported activation of the hippocampus during allocentric (topographical) memory tasks [33,35,36]. The present study adds to these previous findings by showing that the hippocampus is much less, if at all, necessary for normal egocentric spatial memory and by demonstrating the involvement of the hippocampus in the *recognition* of allocentric spatial information as well as recall.

YR's impaired performance on allocentric spatial recognition shows that damage to the hippocampus does not leave recognition for all types of information unimpaired. This means that her pattern of impaired recall but intact item recognition, which has been found on standardised tests, cannot be explained by the differences in the processes underlying recall and recognition. Instead, the results suggest that her mem-

ory performance depends on the type of information which has to be retrieved. YR's data indicate that for some types of information, including the allocentric spatial information used in the present study, hippocampal damage will impair recognition. This finding is consistent with the study of Vargha-Khadem and her associates [72] which found an impairment in recognising the positions of objects following fairly selective hippocampal damage in three young subjects. The critical factor causing the recognition deficit in these two situations may be the spatial nature of the task. However, both YR and the three young hippocampal patients also showed deficits on nonspatial associative tasks in which associations between different types of information had to be remembered [40,72], but performed normally on tasks in which associations between the same kinds of information had to be remembered [22,72]. For example, YR is impaired at recognising picture-sound and face-voice associations, but not face-face associations. The pattern of performance of YR and Vargha-Khadem et al.'s patients on these other associative tests suggests that hippocampal damage disrupts memory for associations between different kinds of information which may be represented in different cortical regions. If this is correct, then spatial memory deficits are merely a special case of the more general failure in associating different kinds of information in memory.

Egocentric spatial memory, like allocentric, involves linking different kinds of information in memory (e.g. self, an object and the egocentric spatial relationship). If egocentric spatial memory is unimpaired following hippocampal damage in humans, then the view that the hippocampus is vital for linking different information of all kinds in memory cannot be completely correct. If the hippocampus is involved in storing associations between different kinds of information, egocentric spatial memory should also be impaired. Our results do not exclude this possibility, however. This is because, allocentric spatial memory typically involves forming more spatial associations than does egocentric. This certainly applied in our task. So one possible reason why a clear egocentric spatial memory deficit was not obtained could simply be that few associations were involved and a deficit would have become clearly apparent if the number of egocentric spatial memory associations that needed to be linked in memory had been markedly increased. Indeed, if it were possible to match complexity, allocentric and egocentric spatial memory might be similarly disrupted by hippocampal lesions. It is therefore important to test whether hippocampal damage clearly disrupts more complex forms of egocentric spatial memory.

Another key question is whether YR has a problem encoding allocentric spatial information or a problem storing this information. YR's performance after the five second filled delay of the allocentric condition was not significantly impaired which suggests that her encoding of allocentric spatial information was intact. This is consistent with a number of studies which report that medial temporal lobe damage can leave the encoding of spatial information and spatial short-term memory intact [9,20,38,42,58,70,74]. Further, YR's results make an important addition to these findings by indicating that hippocampal damage spares encoding of spatial information even when this is encoded within an allocentric framework. The proposal that YR's allocentric spatial memory deficit is caused by inadequate storage (consolidation) in long-term memory [37] predicts accelerated forgetting of allocentric spatial information. YR's data shows that her significant impairment on the allocentric recall and recognition conditions compared with controls was greater at later than at earlier delays indicating accelerated forgetting of allocentric spatial information which, in the recall condition, was particularly striking between the 5 and 20 s delays. This pattern could be interpreted as supporting the hypothesis of a deficit in the consolidation of allocentric visuospatial information. It might be argued that the results can be explained by an alternative account. This explanation rests on an assumption concerning the duration of visuospatial short-term memory. If visuospatial short-term memory is considered to still be active following a filled 5 s delay it could be argued that our pattern of results was produced by differential contribution to performance of intact short-term memory and impaired long-term memory for allocentric spatial information at the three delays. For this explanation to account for the pattern of YR's scores, however, it is necessary to postulate that visuospatial short-term memory is still contributing to YR's recall performance after 20 s because there was a strong trend indicating accelerated forgetting after this delay (YR's difference in performance between the 60 and 20 s delays was 1.64 SDs greater than the control mean difference). Although the duration of visual short-term memory has not been established, the duration of verbal short-term memory has been estimated by experimental methods to be 2 s or less [46] 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 s [25]. These two sources of evidence indicate that the duration of verbal short-term memory is probably less than 15 s. Therefore, if similar mechanisms underlie visuospatial short-term memory it is unlikely that it is still contributing to performance after 20 s [see 12,25, for a discussion of these issues].

In summary, the data from patient YR provide support for the view that hippocampal lesions impair memory for allocentric spatial information and that

this impairment is likely to be one of consolidation of information into long-term memory. This is consistent with previous studies that have indicated an impairment in the consolidation of other types of associative information following such lesions [72]. In contrast, the results suggest that hippocampal lesions have much less, if any, effect on the egocentric spatial memory tapped by our task.

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