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The right parietal lobe is critical for visual working memory

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Abstract

Visual working memory (VWM) permits the maintenance of object identities and their locations across brief delays such as those accompanying eye movements. Recent neuroimaging studies have emphasized the role of the posterior parietal lobe in this process although the specific nature of this involvement in VWM remains controversial. Neuroimaging findings suggest that the parietal lobe may have a general role in remembering various types of visual information whereas neuropsychological findings suggest that parietal involvement is primarily related to motor spatial attention and spatial memory. In the present study, patients with unilateral right parietal lobe damage, lacking symptoms of neglect, were tested in several VWM old/new recognition tasks. Parietal damage lead to impaired performance on all VWM tasks, including spatial, object, and object/spatial conjunction tasks. Deficits were found across several stimulus categories. These results provide neuropsychological support for neuroimaging results, and more generally indicate that the parietal lobe serves a general role in diverse forms of VWM.

Keywords: Parietal lobe; Spatial memory; Visual working memory; Short-term memory

Visual working memory (VWM) permits the maintenance of object identities and their locations across brief delays such as those accompanying eye movements. Psychophysical studies show that VWM capacity is generally limited by two variables: the number of items to be remembered (Luck & Vogel, 1997; Pashler, 1988; Vogel & Machizawa, 2004; see also Cowan, 2001) and stimulus complexity (Alvarez & Cavanagh, 2004; Olson & Jiang, 2002) or stimulus similarity (Awh, Barton, & Vogel, 2007).

The role of the posterior parietal cortex (PPC) in VWM is at the center of a debate in which recent neuroimaging findings conflict with existing neuropsychological evidence. Numerous neuroimaging studies report bilateral PPC activity during diverse VWM tasks, including both spatial and object VWM (for a meta-analysis see Wager & Smith, 2003). Activity in the intraparietal sulcus (IPS) has been shown to titrate with VWM capacity for shapes and colors, suggesting that this region has some role in governing VWM capacity (Macoveanu, Klingberg, & Tegner, 2006; Todd & Marois, 2004, 2005; Xu & Chun, 2006). This activity is not due to the number of items viewed at encoding, but rather to the number of items maintained in VWM, lending support to the idea that the function of the IPS is mnemonic, not perceptual (Todd & Marois, 2004). Suggestions as to the specific mnemonic role of the IPS in VWM include the manipulation of items (Champod & Petrides, 2007), encoding and/or storage of visually complex items (Xu & Chun, 2006; see also Song & Jiang, 2006), or total information accumulation (Xu, 2007).

These findings suggest that portions of the PPC are involved in some aspect of VWM performance. Interestingly, although descriptions of the behavioral sequelae of parietal damage date to the seminal work of Hughlings Jackson (reviewed in Paterson & Zangwill, 1944), memory deficits are not mentioned. Instead, left parietal damage can lead to visual-motor (apraxia), calculation (acalculia), and language problems (aphasia and dyslexia), while right parietal damage is typically associated with problems of visual–spatial representation and attention (Critchley, 1953; Husain & Nachev, 2007; Vallar, 2007). Because we are specifically interested in visual memory, and several lines of evidence (reviewed in Critchley, 1953; Pisella & Mattingley, 2004) suggest that the right PPC, but not the left, is critically involved in visual functions, the remainder of this paper is devoted to the right PPC.

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There is now a growing body of work showing that early studies of parietal lobe patients overlooked one memory deficit: spatial memory problems associated with right PPC damage. Right PPC damage can cause spatial memory impairments across a range of tasks. Deficits are evident in patients with right PPC damage and the clinical syndrome of hemispatial neglect (De Renzi, Faglioni, & Previdi, 1977; Husain et al., 2001; Malhotra et al., 2005; Pisella, Berberovic, & Mattingley, 2004; Ravizza, Behrmann, & Fiez, 2005) and in patients with right PPC damage in the absence of neglect (van Asselen et al., 2006).

Whether the PPC has a role in *object* VWM, rather than exclusive visuospatial involvement, has not been thoroughly examined in neuropsychology. One of the few studies on this topic reported a dissociation between spatial and object VWM performance: right PPC damage diminished spatial VWM performance but not object VWM performance (Pisella et al., 2004). In contrast, Finke, Bublak, and Zihl (2006) found that right PPC patients were impaired on object and spatial delay-match-to-sample tasks, but only when the probed dimension (object or spatial) was unpredictable (Finke et al., 2006), hinting that the right PPC's role in VWM may be attentional rather than mnemonic.

Consequently, two views of right PPC function in VWM emerge. According to the view from neuroimaging, the right PPC has a role in both object and spatial VWM. According to the view of neuropsychology, the right PPC has a role restricted to spatial VWM or object VWM under conditions of encoding/decision uncertainty. It may also be the case that the different views stemming from neuroimaging and neuropsychology may be due to a lack of sufficient neuropsychological evidence. There are few neuropsychological studies investigating VWM and these studies suffer from low power due to small patient populations.

In this manuscript, we investigate whether the right PPC is critical for VWM by comparing the performance of patients with right PPC damage to age- and education-matched control participants. In Experiment 1, PPC patients were tested on a spatial memory task. Deficits on this task would extend prior findings of impaired spatial VWM associated with right PPC damage (Colombo, De Renzi, & Faglioni, 1976; De Renzi et al., 1977; Husain et al., 2001; Malhotra et al., 2005; Pisella et al., 2004; Ravizza et al., 2005). In Experiment 2, PPC patients were tested on an object VWM task. In Experiment 3, PPC patients were tested on a conjunction (spatial + object) VWM task.

1. General methods

1.1. Subjects

Seven right PPC patients without symptoms of neglect (see Table 1 for demographic information and Fig. 1 for lesion drawings) from the Hospital of the University of Pennsylvania patient database participated. Patients were screened for perceptual symptoms of spatial neglect using a modified Albert's line cancellation task (Albert, 1973), the clock drawing task (Agrell & Dehlin, 1998; Van der Horst, 1934), and the greyscales task (Mattingley, Bradshaw, Nettleton, & Bradshaw, 1994); no patient showed signs of spatial neglect or stimulus extinction. Five patients participated in Experiment 1 (mean age = 57.2, range 39–83; mean years of education = 13.2; 1 male) while all seven of them participated in Experiments 2 and 3 (mean age = 62.0, range 39–83; mean years of education = 12.6; 1 male).

Twelve age- and education-matched control subjects (mean age = 59.0, range 38–72; mean years of education = 13.8; 6 males) participated in Experiment 1; an additional two subjects participated in Experiments 2 and 3 (mean age = 59.7, range 38–72; mean years of education = 13.6; 6 males). All participants received $15/h. Testing sessions lasted no more than 2 h. Informed consent was obtained from all participants and the University of Pennsylvania Institutional Review Board approved all experimental protocols.

1.2. Stimuli

Three stimulus categories were used in all experiments reported in this paper: colors, shapes and common objects. The color category consisted of 20 circular color patches selected from the full color spectrum. The shape category consisted of 36 black, bilaterally symmetrical abstract shapes generated by an algorithm that has been used previously to generate objects for VWM studies (Jiang, Olson, & Chun, 2000). The common objects or 'tool' category consisted of 36 grayscale photographs and was limited to the subordinate category of tools in order to be consistent with the other stimulus categories. In Experiments 1 and 3, these three stimulus categories were presented in a visible black 4 × 4 grid on a white background, of approximately 21 cm × 21 cm in size. All physical (i.e. column and row) and temporal (i.e. 1st to 4th) positions were counterbalanced, sampled equally, and associated with an identical number of correct and incorrect responses. In Experiment 2, the same stimuli were used but their size was approximately 6 cm × 6 cm and they were presented centrally, without a grid, on a white background. All stimuli were presented using ePrime software on a Dell laptop monitor.

1.3. Task

Task designs are shown in Fig. 2 ADG. Prior to each task, subjects were shown a trial example on paper, and they performed computerized practice trials to familiarize them with the trial design. The order of task performance was counterbalanced across subjects. In all cases the probe was equally likely to be "old" or "new" and blocks were pseudorandomly counterbalanced.

1.4. Experiment 1: spatial task

Subjects were instructed to remember item location without regard to item identity. Trials began with a fixation cross (1000 ms), followed by the sequential presentation of four items in different grid locations (1000 ms/item). After a checkerboard mask (1000 ms), a single probe item appeared. The task was to decide whether that location had been previously occupied. One block of each stimulus category was tested for a total of 60 trials.

1.5. Experiment 2: object task

Subjects were instructed to remember the identity of items. Trials began with a central fixation cross (1000 ms), followed by the central, sequential presentation of four stimuli (1000 ms/stimulus). After a checkerboard mask (1000 ms), a single probe stimulus appeared at central fixation. The task was to decide whether the probe stimulus had appeared during the initial encoding period, or whether it was a new item. There were 180 trials total, 60 trials per stimulus category.

1.6. Experiment 3: conjunction task

Subjects were instructed to remember the identity and location of items. Trials began with a fixation cross (1000 ms), followed by the memory image (2000 ms). After a checkerboard mask (1000 ms), a single probe item appeared in the matrix. The task was to decide whether that particular probe stimulus had been located in that particular position. On non-match trials, items from the memory image were repositioned to previously occupied locations. There were 180 trials total, 60 per stimulus category.

Table 1
Patient characteristics. Edu = education in years; MCA = middle cerebral artery; Moya–Moya = a rare cerebrovascular disorder; AVM = arteriovenous malformation, I = inferior, S = superior

Subject	Age	Sex	Edu	Injury date	Etiology	Lesion location	Frontal involvement
592	39	F	13	2001	MCA infarct, Moya–Moya	S frontoparietal	+
474	47	F	12	1995	MCA infarct	I parietal, L caudate	–
312	57	F	17	1981	AVM resection	S parietal, cerebellar atrophy	–
560	60	M	12	2003	MCA infarct	I parietal	–
564*	73	F	10	2001	Infarct	S parietal frontal 2 lesions	+
444*	75	F	12	2002	MCA infarct	I temporoparietal	–
316	83	F	12	1999	MCA infarct	S temporoparietal	–
Mean	62.0	6 F/1 M	12.6				

A single '*' demarks patients that were not tested in Experiment 1.

1.7. Analysis

Hit rates (responding "yes" on a match trial) and false alarm rates (responding "yes" on a non-match trial) were used to calculate corrected recognition (CR = hits − false alarms) as the dependent measure. Trials were excluded if no response was registered within two standard deviations of the mean reaction time. Statistical analyses were conducted using SPSS (SPSS, Chicago, IL). Data were subjected to repeated measures analysis of variance (ANOVA) and significance was determined using an α level of .05.

2. Experiment 1: VWM for spatial locations

2.1. Results

Performance was collapsed across the three stimulus categories because this dimension was irrelevant to the task. An independent groups *t*-test with group (right PPC, control) found that right PPC patients had impaired memory for location ($t_{15} = 3.39$, $p = .004$); see Fig. 2B and C and Table 2.

Because right PPC damage is associated with a rightward attentional bias we assessed whether performance followed a spatial gradient. If patients' spatial VWM impairment was due to residual neglect, they would be expected to perform more poorly for items appearing on the left-most portions of the grid. This hypothesis was assessed by repeated measures ANOVA with group and lateral position (outer left, inner left, inner right, and outer right) of the probe stimulus as factors. Patients exhibited worse spatial VWM than controls ($F_{1,15} = 5.59$, $p = .03$). There was a main effect of the lateral position of the probe item ($F_{3,45} = 3.49$, $p = .02$) following a significant quadratic trend ($F_{1,15} = 6.97$, $p = .02$) such that performance on the outer columns was better than performance on the inner columns, across both groups. However, there was no significant interaction between lateral position and group ($F_{3,45} = 1.16$, $p = .34$) suggesting that the spatial memory deficits were not due to subtle neglect.

Table 2
Performance by task and stimulus category

Experiment	Group	Location	Stimulus category		
			Color	Shape	Tool
1	Controls	.75 (.17)	–	–	–
1	Patients	.44 (.14)	–	–	–
2	Controls	–	.53 (.18)	.47 (.14)	.77 (.13)
2	Patients	–	.40 (.20)	.32 (.16)	.62 (.22)
3	Controls	–	.52 (.26)	.42 (.23)	.58 (.18)
3	Patients	–	.33 (.23)	.12 (.09)	.35 (.18)

Means are followed by standard deviations in parentheses.

3. Experiment 2: VWM for objects

Experiment 1 established that right PPC damage impairs spatial VWM. In Experiment 2, we asked whether right PPC damage impairs object VWM by testing patients and controls on an object VWM task that minimized spatial memory demands. Poor performance on the object task would suggest that the right PPC has a general role in VWM whereas unimpaired performance would suggest that the right PPC has a role in VWM limited to spatial VWM.

3.1. Results

A repeated measures ANOVA compared corrected recognition scores for group (right PPC, control) and stimulus category (colors, shapes, tools). Right PPC performance was significantly poorer than that of controls ($F_{1,19} = 4.51$, $p = .05$). The main effect of stimulus category was significant ($F_{2,38} = 42.61$, $p < .001$) due to overall better memory for tools than for colors ($p < .001$) or shapes ($p < .001$). The interaction between group and stimulus category was not statistically reliable ($F_{2,38} < 1$, n.s.) suggesting that the patients were similarly impaired across stimulus categories (see Fig. 2E and F, Table 2).

Because there is some evidence of PPC involvement in temporal as well as spatial attention (Husain, Shapiro, Martin, & Kennard, 1997; Malcolm & Barton, 2007) we assessed whether VWM impairments in the right PPC group would be clearer when temporal order of stimuli was taken into account. Performance was analyzed by temporal position (1st to 4th) of the target item, collapsing across stimulus category to increase power. Corrected recognition was calculated by first subtracting the false alarm rate from the hit rate of each of the four temporal positions.

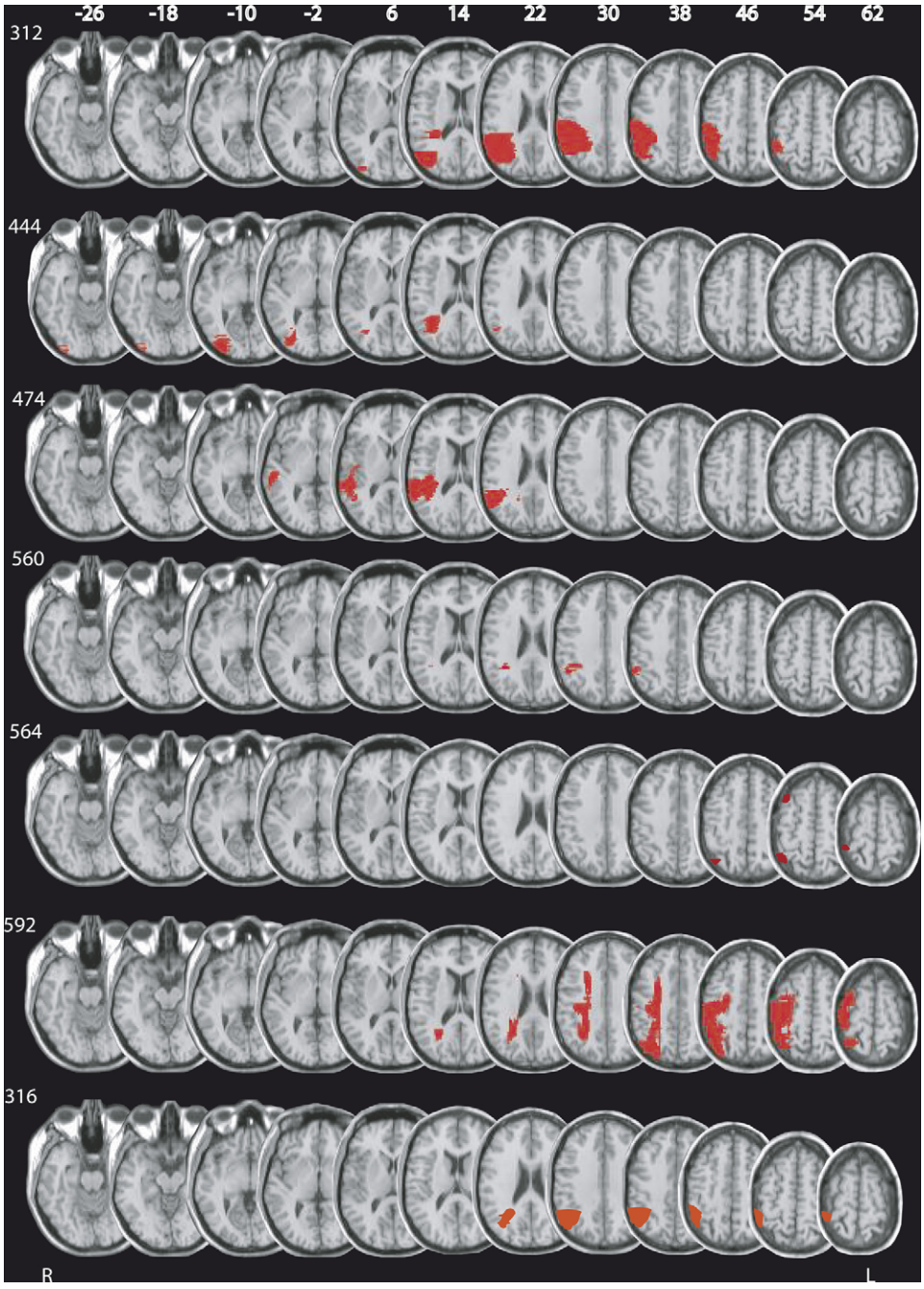

Fig. 1. Patient lesions. Lesions were traced from either standard or flair MRI images on a standardized brain by a neurologist using MRIcro software. Talairach-z coordinates are indicated at the top of the figure Images follow radiological convention (right on the left).

A repeated measures ANOVA compared performance accuracy for group (patient, control) and temporal position (1st, 2nd, 3rd, and 4th) collapsing across stimulus category. Right PPC patients performed worse than controls ($F_{1,19} = 5.21$, $p = .03$) and the effect of temporal position was highly significant ($F_{3,57} = 8.69$, $p < .001$), due to a recency effect across groups. Of interest, the interaction between group and temporal position did not reach significance ($F_{3,57} = 1.51$, $p = .22$).

4. Experiment 3: VWM for object/spatial conjunctions

The previous results suggest that right PPC damage leads to both spatial and object VWM deficits. Does VWM performance worsen when both spatial and object feature information are probed? Patients with medial temporal lobe damage are disproportionately impaired at remembering conjunction stimuli, even at short delays (Olson, Moore, Stark, & Chatterjee, 2006). In Experiment 3, we asked whether VWM deficits would be

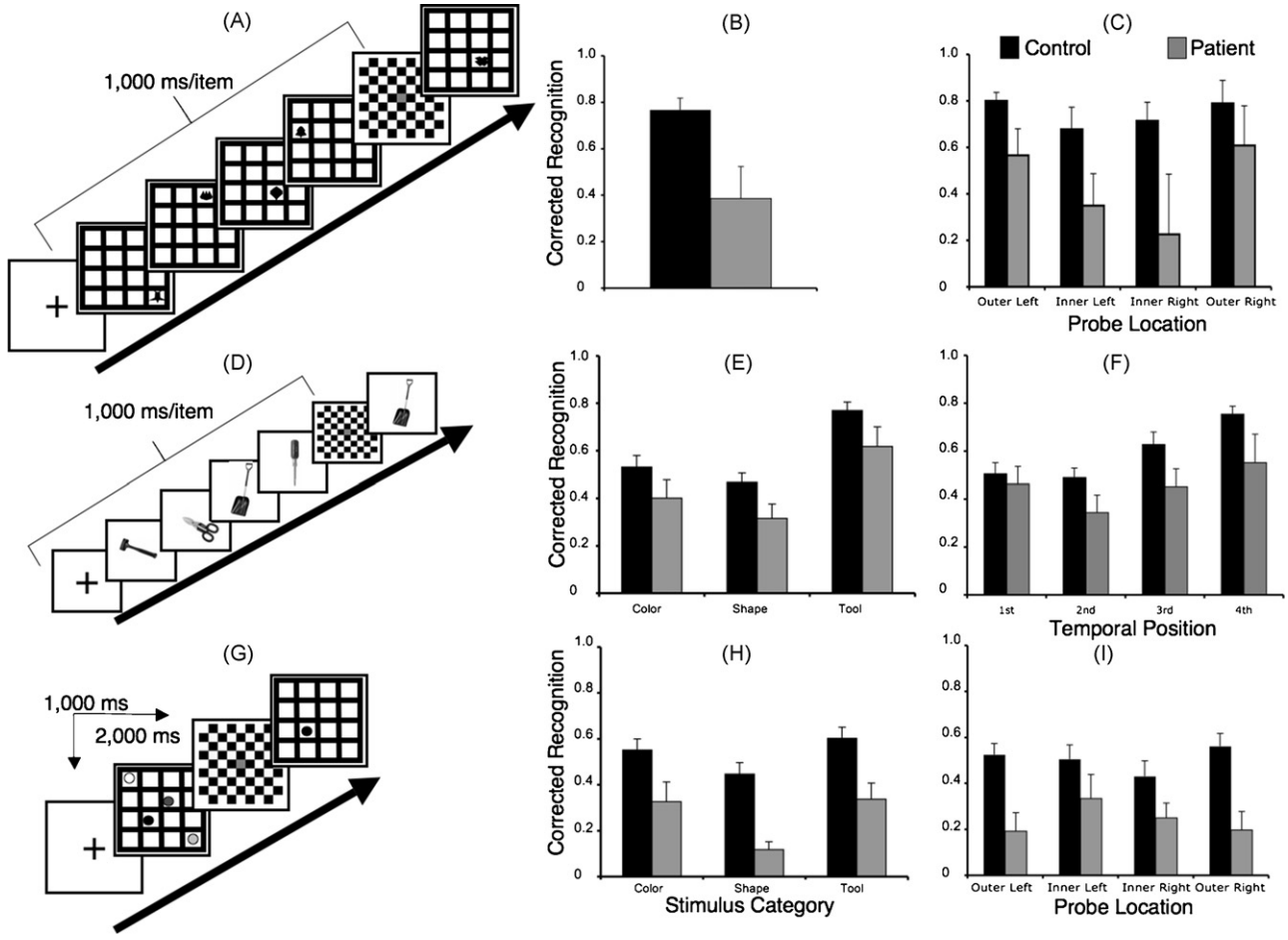

Fig. 2. Task diagrams and performance. (A) Schematic depiction of the spatial VWM task (Experiment 1). Four individual items appeared sequentially in different locations in a visible matrix. Following a masked delay, the probe stimulus appeared and subjects responded whether that location had been previously occupied or whether it was a new location. (B) Spatial VWM task performance of controls and right PPC patients. Corrected recognition scores collapsed across stimulus category by group. (C) Spatial VWM task performance as a function of column location of the probe item. (D) Schematic depiction of the object VWM task (Experiment 2). Following a fixation cross, four objects were presented sequentially at fixation. Following mask delay, a probe image appeared until an old/new response was made. (E) Corrected recognition by group (controls and right PPC patients) and stimulus category (color, shape, and tool). (F) Object VWM performance by the temporal position (1st to 4th item) of the target in the trial sequence. (G) Schematic depiction of the object/spatial conjunction VWM task (Experiment 3). Following a fixation cross, four items were presented simultaneously within a visible matrix. After a masked delay, a probe image appeared. Subjects responded whether the probe image contained a previously viewed item in the same position as in the test display. (H) Corrected recognition by group (controls and right PPC patients) and stimulus category (color, shape, and tool). (I) Conjunction memory performance as a function of the spatial location of the target. All error bars represent the standard error of the mean.

modulated by the requirement to remember the conjunction of object location and object identity.

4.1. Results

A repeated measures ANOVA with group (right PPC, control) and stimulus type (color, shape, and tool) as factors found that patients exhibited worse VWM than controls ($F_{1,19} = 8.24$, $p = .01$). There was also a main effect of stimulus category ($F_{2,38} = 11.93$, $p < .001$) due to generally worse memory for shapes than tools ($p = .001$) or colors ($p = .02$). Although patients showed a larger numerical deficit for the shape stimuli, the interaction of group and stimulus type failed to reach statistical significance ($F_{2,38} < 1$, $p =$ n.s.; see Fig. 2H and I, Table 2).

As in Experiment 1, we assessed whether performance followed a spatial gradient by evaluating performance based on the column position in an ANOVA with group and target position as factors. Patients exhibited worse VWM performance than controls ($F_{1,19} = 10.58$, $p = .004$; control $M = .50$, patient $M = .24$). However, target position had no overall affect on performance ($F_{3,57} < 1$, n.s.) nor was there any specific effect of target position on the performance of patients ($F_{3,57} = 1.62$, $p = .20$). Patients exhibited a slight trend towards rightward impairment, the opposite of what would be expected if there were residual symptoms of contralesional neglect.

5. Additional analyses

5.1. Effects of stimulus complexity/similarity

Stimulus complexity/similarity strongly affects VWM performance and capacity measures. When required to remember

items that are drawn from a stimulus set with a high degree of feature overlap, such as abstract shapes, Chinese characters, or faces, fewer items can be remembered (Alvarez & Cavanagh, 2004; Awh et al., 2007; Eng et al., 2005; Olson & Jiang, 2002; Olsson & Poom, 2005). Behavioral findings have been linked to changes in neural activity in portions of the parietal lobe. These findings predict that parietal lobe damage should differentially impair VWM for highly similar stimuli as compared to dissimilar stimuli (Xu & Chun, 2006; Yago & Ishai, 2006).

Our results provide no evidence in support of this prediction since there were no significant interactions of stimulus type and group. However, it is possible that more careful scrutiny would reveal such an association. We performed two additional analyses to evaluate this possibility. First, we calculated difference scores for the right PPC group by subtracting individual scores from Experiments 2 and 3 from the mean of the control group. These values were subjected to repeated measures ANOVA examining each stimulus category (color, shape, and tool), group (right PPC, control) and task (object, conjunction). Although the main effect of group reached significance ($F_{1,19} = 5.99$, $p = .02$), there was no significant main effect of task ($F_{2,38} < 1$, $p =$ n.s.), stimulus category ($F_{2,38} = 1.24$, $p = .30$) or interaction of stimulus category and group ($F_{2,38} < 1$, $p =$ n.s.) or stimulus category and task ($F_{2,38} = 2.23$, $p = .12$).

One explanation for this null result is that we collapsed across patients, whereas the effects of stimulus similarity in VWM would only be apparent in patients with superior parietal lobe damage. This explanation rests on the results of Xu and Chun (2006) who showed that BOLD activity titrates with stimulus similarity in portions of the superior parietal lobe, but not inferior parietal lobe, and that there was no difference between left and right hemispheres of the parietal lobe. To assess this, we split the patients into an inferior lesion group ($N = 3$) and a superior lesion group ($N = 4$; see Table 1).[1] Then we reanalyzed the results of the object and conjunction memory tasks. An ANOVA with lesion location and stimulus category as factors was conducted for each experiment. In Experiment 2, there was no effect of lesion location for any stimulus category (p's $> .10$). In Experiment 3, this comparison showed that patients with inferior damage were more impaired at remembering shapes than were patients with superior damage ($p = .03$). There was no difference between inferior and superior patients for colors or tools (p's $> .62$). These results fail to support the hypothesis that PPC damage disproportionately impairs VWM for complex/highly similar items. However, it is possible that with increased power, differential deficits will be observed.

6. General discussion

Neuroimaging studies predict that parietal damage should impair VWM for both object and spatial information. In the present study we tested this hypothesis by assessing VWM performance across three tasks and three stimulus categories in patients with right PPC damage, but without neglect. In Experiment 1, subjects were asked where items appeared, in Experiment 2, subjects were asked what items appeared, and in Experiment 3, subjects were asked where a particular item appeared. The VWM performance of right PPC patients was universally impaired.

Additional analyses show that patient performance was not modulated by stimulus similarity/complexity. This analysis was predicated on findings showing a performance cost for remembering items drawn from a stimulus set with high levels of inter-item similarity compared to items drawn from a stimulus set with low levels of inter-item similarity (Alvarez & Cavanagh, 2004; Awh et al., 2007; Olson & Jiang, 2002). This performance cost has been associated with BOLD activity in superior portions of the parietal lobe (Xu & Chun, 2006). Our results suggest that the PPC is not necessary for this function, and that patients with superior PPC damage perform no differently than patients with inferior PPC damage. Alternatively, it is possible that the parietal lobe does have some critical role in this process, but our statistical power was too weak to detect an effect. Even if this is true, it must be the case that such effects are of a small magnitude.

6.1. Alternative explanations

We conducted several analyses to rule out alternative explanations for the findings presented here. The most worrisome explanations – that perceptual or attentional problems account for the observed VWM deficits – can be ruled out. The patients performed normally in a perceptual matching task that required reading the name of an object and matching it to a color photograph of the correct object from a set of four pictures. This task ensured that all subjects could perceive and match color and objects. Furthermore, these patients are unimpaired at mapping a word or picture to a diagrammatic representation, for example mapping a picture of a spoon inside a mug to a schematic line drawing representing the preposition 'in' (Amorapanth et al., 2007). This suggests that these patients do not have a global cognitive deficit.

It is also clear that the observed VWM deficits are not due to an uneven distribution of spatial attention. The patients' performed normally on neuropsychological evaluations of neglect and extinction. Their performance on VWM tasks with a spatial component was no worse when items appeared on the left as compared to the right (Experiments 1 and 3). In Experiment 2 stimuli appeared at fixation, thereby eliminating any spatial component, and performance remained impaired. This pattern of data is difficult to reconcile with a spatial attention explanation.

Although temporal attention did not receive the same degree of scrutiny, it provides little explanatory power. When items were presented sequentially in Experiment 2, there was no interaction of group and temporal position. At longer timescales, patient performance did not decrease across the duration of the testing session.

There are several concerns regarding the specific PPC patient population. First, this group varied widely in age and age predicts memory performance. We do not believe that this explains our findings, because we compared performance with an age-

[1] This analysis was not conducted for Experiment 1 because fewer patients were tested.

matched control group. A second concern relates to the inclusion of two patients with additional frontal pathology. These two patients did not demonstrate significantly different performance when compared to the other patients, however it is possible that more power would reveal that frontal pathology exacerbates the effects of parietal damage on VWM performance.

6.2. Laterality differences

Hemispheric differences in parietal activations have been observed in working memory tasks. For example, a number of behavioral and PET studies employing *n*-back tasks report left-lateralized activity for verbal working memory and right-lateralized activity for spatial working memory (reviewed in Smith & Jonides, 1998). In contrast, more recent MRI studies of VWM observe bilateral parietal activity (see Todd & Marois, 2004; Todd, Fougnie, & Marois, 2005; reviewed in Naghavi & Nyberg, 2005; Owen, McMillan, Laird, & Bullmore, 2005) or have examined bilateral regions of interest (Song & Jiang, 2006; Xu & Chun, 2006). Prior neuropsychological studies have also shown that left parietal damage can impair verbal short-term memory (Warrington, Logue, & Pratt, 1971; for a meta-analysis see Vallar & Papagno, 2002) while our results and those of others (De Renzi et al., 1977; Husain et al., 2001; Malhotra et al., 2005; Pisella et al., 2004; Ravizza et al., 2005; van Asselen et al., 2006) show right PPC involvement in spatial short-term memory. Our study additionally shows that the right PPC is necessary for accurate object short-term memory.

We acknowledge that a weakness of the present study is that left PPC patients were not tested. We speculate that the left PPC does not have an important role in VWM. Our review of the literature shows that left PPC damage is rarely associated with visual perceptual, attentional, or mnemonic deficits. For instance, Haramati et al. (2008) conducted a careful study of patients with unilateral left or right PPC damage and found that the left PPC patients exhibited intact visual long-term recognition memory. In addition, we have preliminary data showing that left PPC damage does not cause VWM deficits. Three left PPC patients without neglect or aphasia were tested in the VWM tasks described here. Two of the patients performed as well as control subjects on all tasks across all stimulus categories. One patient performed normally with the color and tool stimuli, but had impoverished memory for novel shapes. Although these data are preliminary, they suggest that left parietal damage does not produce the same general VWM deficits as those observed in the right PPC patients. A more conclusive statement awaits additional data collection. Data describing the effects of bilateral PPC damage on VWM performance can be found in an accompanying article (Berryhill & Olson, 2008).

6.3. Memory retrieval

In the present study we identified VWM deficits in a series of tasks. However, we did not vary the recall task, raising the question of whether parietal lobe damage impairs VWM regardless of how memory is probed. There is evidence that some neural areas, for instance the hippocampus, are more heavily recruited for recall as compared to recognition (Aggleton & Shaw, 1996; Yonelinas, 2002, but see Wixted & Squire, 2004). Although it is tempting to assume that the parietal lobe is generally required for accurate VWM, and is not sensitive to processes engaged by different recollection tasks, there is little evidence that directly addresses this question given that neuroimaging (Song & Jiang, 2006; Todd & Marois, 2004, 2005; Xu & Chun, 2006) and neuropsychological (Finke et al., 2006; Pisella et al., 2004) studies of VWM overwhelmingly test memory with old/new recognition. In a forthcoming Neuropsychologia paper (Berryhill & Olson, 2008), we present evidence that PPC damage differentially affects old/new recognition as compared to recall, using tasks and stimuli similar to those tested here. These findings suggest that the role of the PPC in VWM is associated with retrieval processes.

7. Conclusions

Our results demonstrate that damage to the right PPC leads to a generalized deficit in VWM across a range of stimuli and encoding tasks. This finding provides neuropsychological evidence that supports neuroimaging reports. While neuropsychological studies are challenging to conduct due to difficulties in identifying suitable patients, they provide causal corroboration for the correlational neuroimaging data. The importance of this corroboration cannot be overstated. Future neuropsychological studies should aim to determine more precisely which portions of right parietal cortex are involved in VWM processing, the level involvement of left parietal cortex, and the degree to which parietal damage is sensitive to retrieval demands.

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Appendix A. Supplementary data

Supplementary data associated with this article can be found, in the online version, at doi:10.1016/j.neuropsychologia.2008.01.009.

References

Aggleton, J. P., & Shaw, C. (1996). Amnesia and recognition memory: A re-analysis of psychometric data. *Neuropsychologia*, *34*(1), 51–62.

Agrell, B., & Dehlin, O. (1998). The clock-drawing test. *Age and Ageing*, *27*, 399–403.

Albert, M. L. (1973). A simple test of visual neglect. *Neurology*, *23*(6), 658–664.

Alvarez, G. A., & Cavanagh, P. (2004). The capacity of visual short-term memory is set by visual information load and by number of objects. *Psychological Science*, *15*, 106–111.

Amorapanth, P. X., Widick, P., & Chatterjee, A. (2007). Dissociations in the representation of spatial relationships. In *Proceedings of the Cognitive Neuroscience Society*.

Awh, E., Barton, B., & Vogel, E. K. (2007). Visual working memory represents a fixed number of items regardless of complexity. *Psychological Science*, *18*, 622–628.

Berryhill, M. E., & Olson, I. R. (2008). Is the posterior parietal lobe involved in working memory retrieval? Evidence from patients with bilateral parietal lobe damage. *Neuropsychologia*, *46*(7), 1775–1786.

Champod, A. S., & Petrides, M. (2007). Dissociable roles of the posterior parietal and the prefrontal cortex in manipulation and monitoring processes. *Proceedings of the National Academy of Sciences: USA*, *104*(37), 14837–14842.

Colombo, A., De Renzi, E., & Faglioni, P. (1976). The occurrence of visual neglect in patients with unilateral cerebral disease. *Cortex*, *12*(3), 221–231.

Cowan, N. (2001). The magical number 4 in short-term memory: A reconsideration of mental storage capacity. *Behavioral & Brain Sciences*, *24*(1), 87–114.

Critchley, M. (1953). *The parietal lobes*. London: Edward Arnold.

De Renzi, E., Faglioni, P., & Previdi, P. (1977). Spatial memory and hemispheric locus of lesion. *Cortex*, *13*(4), 424–433.

Eng, H. Y., Chen, D., & Jiang, Y. (2005). Visual working memory for simple and complex visual stimuli. *Psychonomic Bulletin & Reivew*.

Finke, K., Bublak, P., & Zihl, J. (2006). Visual spatial and visual pattern working memory: Neuropsychological evidence for a differential role of left and right dorsal visual brain. *Neuropsychologia*, *44*(4), 649–661.

Haramati, S., Soroker, N., Dudai, Y., & Levy, D. A. (2008). The posterior parietal cortex in recognition memory: A neuropsychological study. *Neuropsychologia*, *46*(7), 1756–1766.

Husain, M., & Nachev, P. (2007). Space and the parietal cortex. *Trends in Cognitive Science*, *11*(1), 30–36.

Husain, M., Mannan, S., Hodgson, T., Wojciulik, E., Driver, J., & Kennard, C. (2001). Impaired spatial working memory across saccades contributes to abnormal search in parietal neglect. *Brain*, *124*, 941–952.

Husain, M., Shapiro, K., Martin, J., & Kennard, C. (1997). Abnormal temporal dynamics of visual attention in spatial neglect patients. *Nature*, *385*(6612), 154–156.

Jiang, Y., Olson, I. R., & Chun, M. M. (2000). Organization of visual short-term memory. *Journal of Experimental Psychology: Learning, Memory, and Cognition*, *26*, 683–702.

Luck, S. J., & Vogel, E. K. (1997). The capacity of visual working memory for features and conjunctions. *Nature*, *390*, 279–281.

Macoveanu, J., Klingberg, T., & Tegner, J. (2006). A biophysical model of multiple-item working memory: A computational and neuroimaging study. *Neuroscience*, *141*(3), 1611–1618.

Malcolm, G. L., & Barton, J. J. (2007). Sequence Agnosia" in Balint's syndrome: Defects in visuotemporal processing after bilateral parietal damage. *Journal of Cognitive Neuroscience*, *19*(1), 102–108.

Malhotra, P., Jager, H. R., Parton, A., Greenwood, R., Playford, E. D., Brown, M. M., et al. (2005). Spatial working memory capacity in unilateral neglect. *Brain*, *128*, 424–435.

Mattingley, J. B., Bradshaw, J. L., Nettleton, N. C., & Bradshaw, J. A. (1994). Can task specific perceptual bias be distinguished from unilateral neglect? *Neuropsychologia*, *32*(7), 805–817.

Naghavi, H. R., & Nyberg, L. (2005). Common fronto-parietal activity in attention, memory, and consciousness: Shared demands on integration? *Consciousness and Cognition*, *14*(2), 390–425.

Olson, I. R., & Jiang, Y. (2002). Is visual short-term memory object based? *Perception & Psychophysics*, *64*, 1055–1067.

Olson, I. R., Moore, K. S., Stark, M., & Chatterjee, A. (2006). Visual working memory is impaired when the medial temporal lobe is damaged. *Journal of Cognitive Neuroscience*, *18*(7), 1087–1097.

Olsson, H., & Poom, L. (2005). Visual memory needs categories. *Proceedings of the National Academy of Sciences: USA*, *102*(24), 8776–8780.

Owen, A. M., McMillan, K. M., Laird, A. R., & Bullmore, E. (2005). N-back working memory paradigm: A meta-analysis of normative functional neuroimaging studies. *Human Brain Mapping*, *25*(1), 46–59.

Pashler, H. (1988). Familiarity and visual change detection. *Perception & Psychophysics*, *44*, 369–378.

Paterson, A., & Zangwill, O. L. (1944). Disorders of visual space perception associated with lesions of the right cerebral hemisphere. *Brain*, *67*, 331–358.

Pisella, L., & Mattingley, J. B. (2004). The contribution of spatial remapping impairments to unilateral visual neglect. *Neuroscience & Biobehavioral Reviews*, *28*(2), 181–200.

Pisella, L., Berberovic, N., & Mattingley, J. B. (2004). Impaired working memory for location but not for colour or shape in visual neglect: A comparison of parietal and non-parietal lesions. *Cortex*, *40*, 379–390.

Ravizza, S. M., Behrmann, M., & Fiez, J. A. (2005). Right parietal contributions to verbal working memory: Spatial or executive? *Neuropsychologia*, *43*(14), 2057–2067.

Smith, E. E., & Jonides, J. (1998). Neuroimaging analyses of human working memory. *Proceedings of the National Academy of Sciences: USA*, *95*(20), 12061–12068.

Song, J. H., & Jiang, Y. (2006). Visual working memory for simple and complex features: An fMRI study. *Neuroimage*, *30*(3), 963–972.

Todd, J. J., & Marois, R. (2004). Capacity limit of visual short-term memory in human posterior parietal cortex. *Nature*, *428*(6984), 751–754.

Todd, J. J., & Marois, R. (2005). Posterior parietal cortex activity predicts individual differences in visual short-term memory capacity. *Cognitive, Affective, & Behavioral Neuroscience*, *5*(2), 144–155.

Todd, J. J., Fougnie, D., & Marois, R. (2005). Visual short-term memory load suppresses temporo-parietal junction activity and induces inattentional blindness. *Psychological Science*, *16*(12), 965–972.

Vallar, G. (2007). Spatial neglect, Balint-Homes' and Gerstmann's syndrome, and other spatial disorders. *CNS Spectrums*, *12*(7), 527–536.

Vallar, G., & Papagno, C. (2002). Neuropsychological impairments of verbal short-term memory. In A. Baddeley, B. Wilson, & M. D. Kopelman (Eds.), *Handbook of memory disorders* (2nd ed., pp. 249–270). Chichester, UK: John Wiley.

van Asselen, M., Kessels, R. P., Neggers, S. F., Kappelle, L. J., Frijns, C. J., & Postma, A. (2006). Brain areas involved in spatial working memory. *Neuropsychologia*, *44*(7), 1185–1194.

Van der Horst, L. (1934). Constructive apraxia: Psychological views on the conception of space. *Journal of Nervous and Mental Disease*, *80*, 645–650.

Vogel, E. K., & Machizawa, M. G. (2004). Neural activity predicts individual differences in visual working memory capacity. *Nature*, *428*(6984), 748–751.

Wager, T. D., & Smith, E. E. (2003). Neuroimaging studies of working memory: A meta-analysis. *Cognitive, Affective, & Behavioral Neuroscience*, *3*(4), 255–274.

Warrington, E. K., Logue, V., & Pratt, R. T. (1971). The anatomical localisation of selective impairment of auditory verbal short-term memory. *Neuropsychologia*, *9*(4), 377–387.

Wixted, J. T., & Squire, L. R. (2004). Recall and recognition are equally impaired in patients with selective hippocampal damage. *Cognitive, Affective, & Behavioral Neuroscience*, *4*(1), 58–66.

Xu, Y. (2007). The role of the superior intraparietal sulcus in supporting visual short-term memory for multifeature objects. *Journal of Neuroscience*, *27*(43), 11676–11686.

Xu, Y., & Chun, M. M. (2006). Dissociable neural mechanisms supporting visual short-term memory for objects. *Nature*, *440*(7080), 91–95.

Yago, E., & Ishai, A. (2006). Recognition memory is modulated by visual similarity. *Neuroimage*, *31*(2), 807–817.

Yonelinas, A. P. (2002). The nature of recollection and familiarity: A review of 30 years of research. *Journal of Memory and Language*, *46*, 441–517.