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Similarities and differences between parietal and frontal patients in autobiographical and constructed experience tasks

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ABSTRACT

Recent findings suggest that *constructed experience*, the ability to envision future events, activates the same cortical network as recollection of past events. For example, damage to one key area, the hippocampus, impairs patients' ability to remember the past and to imagine novel experiences (Hassabis, Kumaran, Vann, & Maguire, 2007). Here, we investigated whether damage to two other areas, posterior parietal cortex and prefrontal cortex, also impairs this ability. Patients with bilateral posterior parietal lesions or unilateral prefrontal lesions were tested in their ability to describe imaginary future events. Only parietal patients were impaired at freely describing autobiographical memories, but both patient groups were impaired when elaborating constructed experiences. This dissociation suggests that parietal and prefrontal structures are differentially involved in constructed experience. Current tasks may impose overly broad cognitive demands making it impossible to specify the deficient cognitive component in any patient group. These findings provide additional constraints regarding the mechanistic role of the parietal cortex in memory.

There are words and phrases that shape our anticipation of the future: if I get tenure, after the New Year, when the grant is funded. Recently, considerable attention has focused on the cognitive mechanism underlying this ability, variably termed constructed experience, prospective memory, episodic future thinking, or mental time travel, (see Buckner & Carroll, 2007). A number of investigators have proposed that constructed experience draws upon episodic memory in order to predict or imagine the future (Arzy, Molnar-Szakacs, & Blanke, 2008; Atance & O'Neill, 2001; Botzung, Denkova, & Manning, 2008; Buckner & Carroll, 2007; Hassabis & Maguire, 2007; Schacter & Addis, 2007; Schacter, Addis, & Buckner, 2007; Schacter, Addis, & Buckner, 2008; Spreng, Mar, & Kim, 2009; Suddendorf, Addis, & Corballis, 2009; Suddendorf & Corballis, 2007). This idea rests on neuroimaging findings showing robust and consistent overlap in the network of regions activated during episodic and constructed experience tasks, including the hippocampus, lateral and medial parietal cortex, and medial prefrontal regions (Addis, Wong, & Schacter, 2007; Botzung et al., 2008; Okuda et al., 2003; Szpunar & McDermott, 2008; Szpunar, Watson, & McDermott, 2007; but see Hassabis, Kumaran, & Maguire, 2007). Additional support is found in neuropsychological studies showing that patients with medial temporal lobe (MTL) amnesia are significantly impaired when recollecting past episodic events or constructing future scenarios (Hassabis, Kumaran, Vann, & Maguire, 2007; Jensen, Duff, Adolphs, & Tranel, 2008).

Only recently has the suggestion of posterior parietal (PPC) involvement in episodic memory gained ground. A large number of neuroimaging studies have reported activations in inferior (supramarginal and angular gyri) and medial (precuneus) PPC regions during episodic memory retrieval (Cabeza, Ciaramelli, Olson, & Moscovitch, 2008; Olson & Berryhill, 2009; Wagner, Shannon, Kahn, & Buckner, 2005). Damage to the PPC can impair specific aspects of episodic memory retrieval. For instance, we tested two patients with bilateral PPC damage and matched controls on a previously published autobiographical memory task in which they were required to recollect events from throughout their lifetimes (Levine, Svoboda, Hay, Winocur, & Moscovitch, 2002). In the first phase, participants freely recalled autobiographical events in as much detail as possible. In the second phase, they answered specific probe questions about each memory. The results showed that PPC damage decreased the vividness and amount of freely recalled detail. The observed deficits could not be attributed to spatial memory deficits per se, as the patients also reported fewer perceptual, emotional and referential details. Nor could the observed deficit be attributed to general mental imagery deficits, a problem which could plausibly diminish the ability to "see" the past, as mental imagery for non-spatial visual features was normal. However, when the same autobiographical memories were probed with specific

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questions, the parietal patients' recollection was normal (Berryhill, Phuong, Picasso, Cabeza, & Olson, 2007; Davidson et al., 2008).

It is important to note that patients with PPC damage are not amnesic and do not exhibit the same constellation of memory deficits observed in patients with hippocampal lesions. For instance, PPC lesions do not appear to affect the accuracy of source memory judgments (Simons et al., 2008; Simons, Peers, Mazuz, Berryhill, & Olson, 2010) or the ability to form new associative memories (Berryhill, Drowos, & Olson, 2010) whereas hippocampal damage causes poor performance on these types of tasks (reviewed in Cohen et al., 1999; Johnson, Hashtroudi, & Lindsay, 1993). In contrast, both patient groups perform poorly on the classic false memory tasks, although the magnitude of the impairment is somewhat greater after hippocampal damage (Davidson et al., 2008; Drowos, Berryhill, Andre, & Olson, in press).

These findings prompt the question: does the PPC, like the hippocampus, play a functional role in constructed experience? To test this hypothesis, participants with bilateral PPC lesions were compared to a group of patients with unilateral ventrolateral or dorsal prefrontal (PFC) lesions, as well as age- and education-matched controls. In order to permit a full comparison between autobiographical memory and constructed experience, we first tested PFC patients on the autobiographical memory task described above; see Experiment 1. The purpose of this step was to confirm that brain damage alone did not account for poor performance on the free recall portion of the autobiographical memory retrieval task. Subsequently, in Experiment 2, PFC, PPC and control participants were tested in a constructed experience task previously used to identify deficits in patients with MTL damage (Hassabis et al., 2007). Participants were supplied with cues describing commonplace events ("Imagine you are on a white sandy beach") and were then asked to verbally describe a mental image associated with the cue in as much multisensory detail as possible.

1. General methods

1.1. Neuropsychological participants

All patients were obtained through the Center for Cognitive Neuroscience Patient Database at the University of Pennsylvania (Fellows, Stark, Berg, & Chatterjee, 2008). All patients and controls signed informed consent documents for each experimental protocol. The Institutional Review Board of the University of Pennsylvania approved all experimental protocols. The bilateral PPC patients EE555 and TQ591 have been described in detail previously (Berryhill & Olson, 2008; Berryhill et al., 2007). Both PPC patients have bilateral inferior parietal lesions; see Fig. 1 and Supplementary Materials for full descriptions of all patients. Neither PPC patient has any damage to the frontal lobe. Their primary clinical symptom is simultanagnosia, the inability to simultaneously see multiple objects. However, visual perception and acuity remains normal.

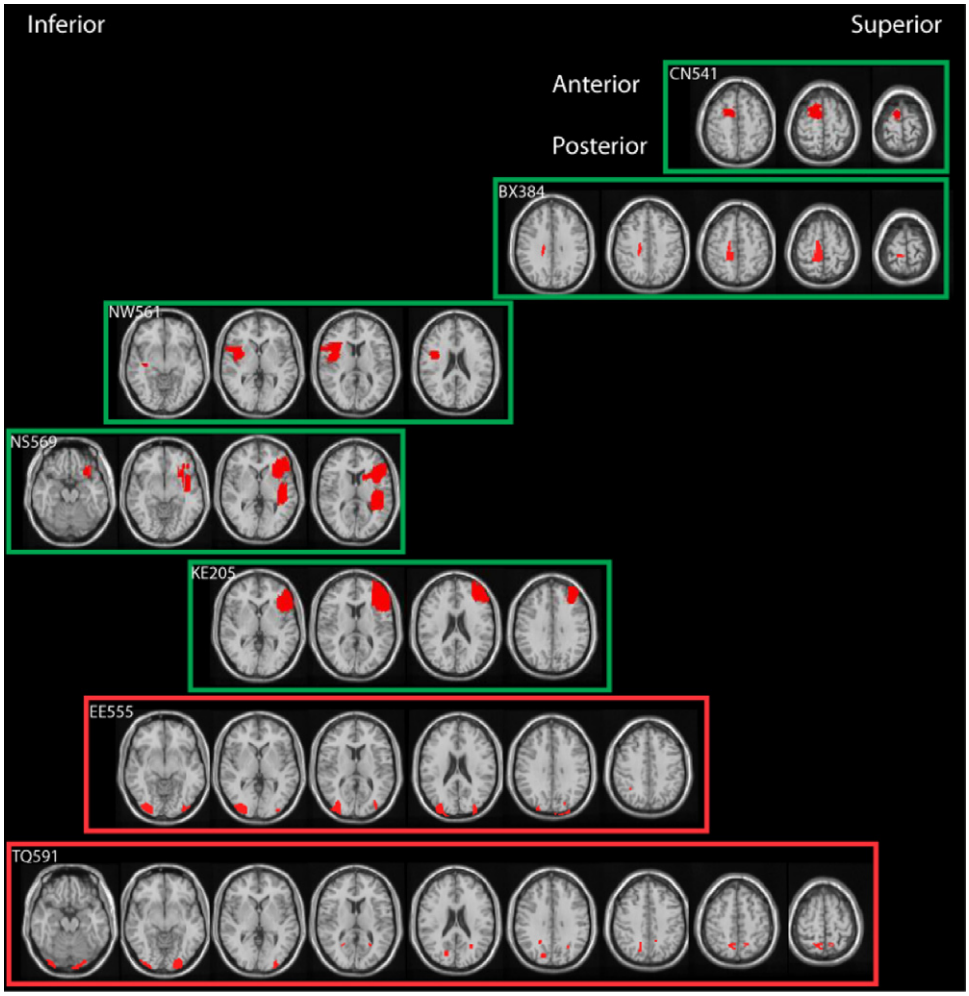

Fig. 1. Patient lesions projected on a standard brain. The green outlines indicate PFC patients and the red outline indicates PPC patients. Brain slices are arranged along the *z*-plane, with inferior slices to the left and more superior slices to the right. The anterior portion is oriented towards the top of the image and the posterior portion of the bottom of the image. Left is depicted on the left. (For interpretation of the references to color in this figure legend, the reader is referred to the web version of the article.)

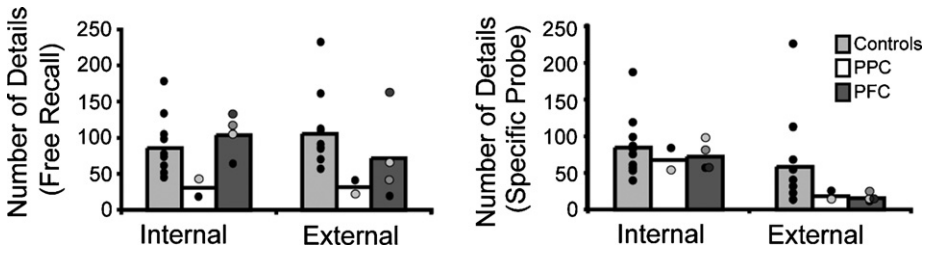

Fig. 2. Autobiographical memory comparison for the control, PPC and PFC groups for the free recall (left) and specific probe (right) recall phases. Bars reflect the group mean; symbols represent individual participants' performance. Patients' symbols are shaded to represent the age of the patient; the darker the symbol, the older the patient. The shading consistently applies to the same patient across figures. PPC patients: black = TQ591, light grey = EE555; PFC patients: black = KE205, dark grey = NS569, grey = CN541, and light grey = NW561.

PFC patients were selected who did not have lesions in medial or ventromedial PFC, since these areas are generally activated in neuroimaging studies of constructed experience (Abraham, Schubotz, & von Cramon, 2008; Addis et al., 2007; Botzung et al., 2008; Hassabis et al., 2007; Okuda et al., 2003; Szpunar & McDermott, 2008); see Figure 1 for lesion tracings and the Supplementary Materials for detailed descriptions. The PFC patients do not have parietal damage. The five patients with PFC lesions have mixed aetiologies. All of the PFC patients are high functioning and score within the normal range in screening tests of attention, cognition, memory and depression (Beck Depression Inventory < 10). Although the age range of the PFC patients is considerable, we observed no correlation between age and performance and therefore elected to include all of the patients regardless of age.

The PPC and PFC patients did not significantly differ in terms of age (*M* PPC: 44.0, range 39–49, *M* PFC: 59.6, range 35–81) or education (*M* PPC: 15.5, range 15–16, *M* PFC: 16.4, range 12–20, Mann–Whitney's tests both z's < −.78, both p's > .44). Although it was not significant, there was a greater age-range in the PFC group. Because there were small numbers of patients we relied on non-parametric statistics. As a result we were unable to use age as a covariate. To clarify the relationship between age and performance each patient's performance is indicated in Figs. 2, 3 and 5.

2. Experiment 1: autobiographical memory following PFC damage

We previously reported that bilateral PPC lesions created a specific deficit in the ability to freely recall autobiographical memories (Berryhill et al., 2007). A limitation of that study was that it did not include a brain damaged control group to account for the possibility that deficits were side effects of brain damage. Here, we tested a diverse group of PFC patients in the autobiographical memory task described previously (Levine et al., 2002).

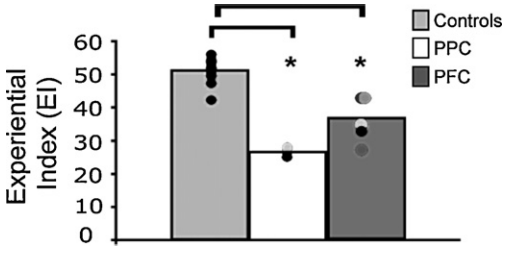

Fig. 3. Experiential index (0–60) scores for control participants, parietal and frontal patients. Symbols represent individual participants' performance, the bar indicates the group mean and the asterisks indicate significance between the patient groups and the control group. Patients' symbols are shaded to represent the age of the patient; the darker the symbol, the older the patient. The shading consistently applies to the same patient across figures. PPC patients: black = TQ591, light grey = EE555; PFC patients: black = KE205, dark grey = NS569, medium grey = BX384, grey = CN541, and light grey = NW561.

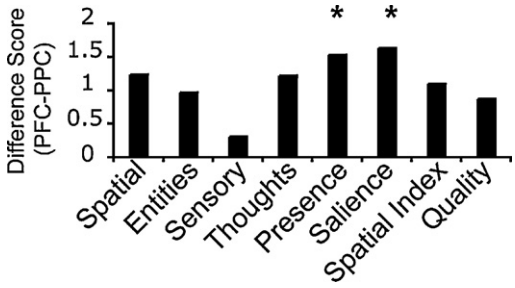

Fig. 4. The difference scores between the PFC and PPC groups. Positive values indicate PFC superiority across all of the subcomponent measures. The asterisk indicates a statistically significant difference between PFC and PPC groups. The following abbreviated titles have been used: spatial = spatial references, entities = entities present, sensory = sensory descriptions, thoughts = thoughts, emotions, actions, presence = sense of presence, salience = perceived salience, spatial index = spatial coherence index, and quality = scorer rating quality judgment.

There is considerable reason to suspect that portions of the PFC are involved in episodic memory. Broad frontal activations are reliably reported during episodic memory tasks (Fletcher & Henson, 2001; Rugg, Otten, & Henson, 2002; Tulving, Kapur, Craik, Moscovitch, & Houle, 1994). However, since PFC damage does not lead to general autobiographical memory deficits these activations may reflect cognitive control processes or strategic operations (reviewed in Badre, 2008). For instance, a recent study assessed autobiographical memory in patients with surgical frontal lobe resections. The results showed that PFC patients had unimpaired autobiographical memory, but they failed to use temporal order information to guide their narratives (Thaiss & Petrides, 2008). In

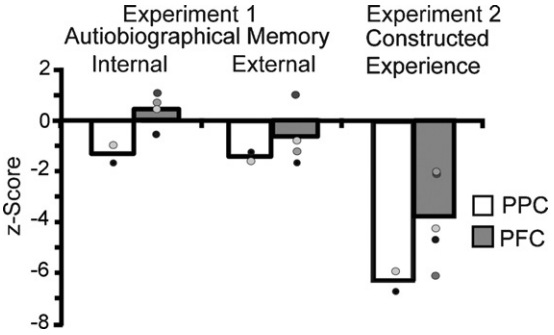

Fig. 5. Between task comparison. Standardized z-scores for each patient group (PPC, PFC) compared to the respective control groups for Experiment 1 and Experiment 2. For Experiment 1, the separate tallies indicate internal (left) and external (middle) freely recalled details compared to controls. The Experiment 2 (right) comparison uses the constructed experience Episodic Index scores. Patients' symbols are shaded to represent the age of the patient; the darker the symbol, the older the patient. The shading consistently applies to the same patient across figures. PPC patients: black = TQ591, light grey = EE555; PFC patients: black = KE205, dark grey = NS569, medium grey = BX384, grey = CN541, and light grey = NW561.

addition, McKinnon and colleagues (2008) recently tested patients with frontotemporal lobar degeneration in the autobiographical memory task we selected (Levine et al., 2002). They found that a subgroup of these patients, those with temporal lobe degeneration, failed to elicit richly detailed episodic memories. However those with frontal degeneration performed normally (McKinnon et al., 2008). These findings predict that although recollection strategy may shift following frontal lobe damage, generally these patients should perform the autobiographical memory task normally. This was tested in Experiment 1.

3. Methods

3.1. Participants

The data from the 10 control participants (age: mean 46.7 years, range 37–57, 6 males; education: mean 15 years, range 10–19) and the PPC patients (*M* age = 44.0, range 39–49, 0 males; *M* education = 15.5, range 15–16) reported in Berryhill et al. (2007) were compared with four PFC patients (*M* age = 57.3, range 35–81, 1 male; *M* education = 17.5, range 14–20). Data from PFC patient BX384 was unavailable. There were no significant differences in age or education between patient and control groups as determined by non-parametric Kruskal–Wallis tests (both $\chi^2_{(2)} < 2.35$, both *p*'s > .31).

3.2. Autobiographical memory procedure

The methods followed those of Levine et al. (2002) and are briefly reviewed here. Participants were asked to recount a memory from 5 different time periods: childhood, adolescence, through high school, early adulthood, middle adulthood and within the past year. For each memory there were two phases of memory recall: free recall and specific probe. In the free recall phase, participants verbally recounted their memory with as much multisensory detail as possible. In the specific probe phase, a series of seventeen specific probe questions were posed (e.g. What were you wearing? What was the weather like?). Patients were recorded with GarageBand software (Apple Inc, Cupertino, CA).

3.3. Scoring

The recordings were transcribed off-line and subjected to text analysis (Levine et al., 2002). In brief, details related to the central event (internal event details, time, place, and thoughts/emotions) and other utterances unrelated to the central event (external event details, semantic facts, repetitions and other) were tallied. The free recall and specific probe data were tallied separately. For each internal category the coders supplied a 0–3 rating as a measure of response quality. In addition, the entire memory was rated for quality rating on a scale of 0–6. Two raters independently scored the transcripts and agreement between scorers was high (intraclass correlation = .97). The raters' scores for each memory were averaged and then subjected to statistical analyses.

3.4. Analysis

To identify whether the PFC group had impaired autobiographical memory we compared the PFC patients to the control group described previously (Berryhill et al., 2007). They were separately compared to the bilateral PPC patients. To assess main effects, we replicated the non-parametric statistical approach used previously (Berryhill et al., 2007). This approach was followed because the small number of patients prohibited the use of parametric statistics. We first calculated the standard *F*-statistic according to a two-factor ANOVA. Subsequently, the group labels were permuted and a one-tailed count with 1000 replications was used to determine the significance. Where appropriate, pairwise comparisons using non-parametric Mann–Whitney's tests identified the significant differences and the *z*-score as well as two-tailed effects are reported.

4. Results

4.1. Quantitative analysis: PFC versus controls

The free recall data were subjected to a permutation analysis with the factors of group (PFC, control) and detail type (internal, external; see Fig. 2 and Table 1). The PFC patients performed normally ($F_{1,\ 13} < 1$, p = n.s.) with one exception. The interaction of group and detail type was significant ($F_{1,\ 13} = 5.15$, $p = .04$) due to the fact that the PFC patients reported relatively more internal details (*M* control = 86.35, *M* PFC = 104.38), but relatively fewer external details (*M* control = 105.80, *M* PFC = 71.88).

Two additional permutation tests assessed whether the PFC patients performed abnormally across any subcategory of internal details (internal event details, time, place, perceptual, and thoughts/emotions), or external details (external event details, semantic, repetitions and other). The PFC patients performed normally across all measures (internal: $F_{1,\ 13} = 8.02$, $p = .42$; external: $F_{1,\ 13} = 1.39$, $p = .73$). No interaction reached significance ($F_{1,\ 13} < 1$, p = n.s.). Permutation analyses were conducted on the specific probe data where no significant main effects or interactions were found (all *p*'s > .09).

4.2. Qualitative analysis of memory ratings

The ratings provided by the experimenters for the free recall and specific probe phases were also analyzed using permutation tests. This measure provides a qualitative assessment of the PFC patients' memories. These data echoed that of the quantitative analysis: PFC patients performed normally. None of the relevant main effects or interactions were significant (all *p*'s > .30).

4.3. Free recall and specifically probed recall: comparison to PPC patients

The two patient groups (PFC, PPC) were compared using Mann–Whitney's tests for the total number of internal and external details for both the free recall and specific probe phases. We suffered from limited power and restricted our comparisons to these contrasts. The PPC patients had numerically lower performance across all measures. For both free recall measures, internal and external details, the PPC patients reported numerically fewer internal details ($z = -1.85$, $p = .06$; *M* PPC = 31.0, *M* PFC = 104.0) and fewer external details ($z = -.93$, $p = .36$; *M* PPC = 31.2, *M* PFC = 71.3). Neither condition reached statistical significance, presumably due to the large variability in the PFC group. The groups performed more similarly on the specific probe questions for both internal ($z = 0$, $p = 1$; *M* PPC = 68.0, *M* PFC = 72.1) and external ($z = -.46$, $p = .64$; *M* PPC = 18.8, *M* PFC = 16.4) details.

5. Discussion

Experiment 1 showed that across multiple measures, a group of PFC patients did not exhibit impaired autobiographical memory retrieval. Indeed, the PFC patients performed similarly to the control population in both the free recall and specific probe phases. The interaction between control subjects and PFC patients who freely recalled a greater number of internal details and a smaller number of external details also counters age concerns. Previous work has found that older adults tend to exhibit the opposite pattern (Addis, Wong, & Schacter, 2008; Levine et al., 2002). The PFC results contrast with the PPC results. PPC lesions selectively impaired the free recall phase of autobiographical memories (Berryhill et al., 2007). These data confirm that brain damage alone cannot account for the abnormal autobiographical memory observed in PPC patients.

6. Experiment 2: constructed experience

The goal of Experiment 2 was to examine whether PPC damage leads to impaired performance on constructed experience tasks. Since the same cortical networks are observed during episodic memory and constructed experience tasks, we predicted that the PPC patients would perform poorly. Because the PPC group described past events in impoverished detail, we predicted they would also describe imaginary events in impoverished detail. In contrast, since the PFC group performed normally in the autobio-

Table 1
Experiment 1 performance. The group mean and standard error of the mean for each measure are presented. Asterisks indicate that the patient group was significantly impaired when compared to the control group.

Measure	Mean (SEM)		
	PPC patients, $n = 2$	PFC patients, $n = 4$	Controls, $n = 10$
Free recall: internal	31.00 (12.5)*	104.37 (14.5)	86.35 (13.5)
Internal details			
Internal event details	22.50 (10.5)*	54.0 (7.7)	43.45 (6.4)
Time	1 (0)*	9.38 (1.46)	6.20 (1.2)
Place	2 (1)*	9.63 (.89)	6.51 (.47)
Perceptual	2 (1.5)*	12.63 (2.9)	13.5 (3.3)
Thought/emotions/actions	3.25 (1.4)*	18.5 (4.0)	17.45 (5.1)
Free recall: external	31.75 (9.3)*	71.88 (31.0)	105.8 (16.9)
External details			
External event details	14.75 (4.2)*	29.25 (15.1)	40.85 (10.1)
Semantics	5.75 (2.7)*	20.13 (5.5)	30.7 (5.4)
Repetitions	2.5 (.5)	10.63 (3.9)	7.65 (1.9)
Other	4.25 (1.3)*	12.38 (1.3)	11.85 (.9)
Free recall: rating			
Episodic richness	10.0 (3)*	17.38 (1.8)	17.85 (.9)
Time	1.0 (0)*	7.88 (1.2)	5.35 (.9)
Perceptual	2.75 (1.3)	6.88 (.72)	7.2 (.9)
Thoughts/emotions/actions	5.0 (1.5)*	11.0 (1.3)	10.2 (.9)
Specific probe: internal	68.0 (15.0)	72.9 (10.0)	85.05 (13.4)
Internal details			
Internal event details	22.25 (8.7)	16.5 (2.2)	27.5 (7.2)
Time	12.5 (.50	19.5 (2.3)	15.5 (1.1)
Place	6.5 (1.5)	6.5 (1.1)	7.2 (1.4)
Perceptual	14.0 (1.1)	22.25 (4.2)	21.4 (4.5)
Thoughts/emotions/actions	12.75 (1.8)	8.0 (1.7)	13.45 (2.6)
Specific probe: external	18.75 (5.7)	16.38 (2.9)	58.75 (20.9)
External details			
External event details	10.75 (1.3)	9.0 (1.9)	19.25 (6.1)
Semantics	2.5 (2.5)	3.0 (1.7)	14.85 (5.5)
Repetitions	1.25 (1.25)	2.75 (1.0)	3.35 (1.6)
Other	1.5 (1.50)	1.13 (.7)	6.85 (1.4)
Specific probe: rating			
Episodic richness	13.75 (2.8)*	20.5 (.9)	21.45 (.6)
Time	10.25 (.8)	14.13 (.2)	12.9 (.7)
Place	9.0 (2.0)	10.63 (.7)	11.15 (.6)
Perceptual	7.75 (2.2)	12.63 (.7)	10.8 (.6)
Thoughts/emotions/actions	9.0 (.5)*	11.4 (1.3)	12.65 (.6)

graphical memory task we predicted that they would also perform normally in the constructed experiences task.

7. Method

7.1. Participants

A second group of ten normal, healthy controls (*M* age = 47.8, *M* years of education = 15.4, 3 males) with normal neurological histories were tested. Both PPC patients (*M* age = 44.0, *M* education = 15.5) and five PFC patients (*M* age = 59.6, *M* education = 16.4) participated. There were no significant differences in age or education between patient and control groups as determined by non-parametric Kruskal–Wallis tests (both $\chi^2_{(2)} < 1.76$, both *p*'s > .41).

7.2. Constructed experience procedure

Our protocol followed Hassabis et al. (2007). In the free recall phase the participants described ten commonplace scenarios in as much sensory detail as possible. There were seven commonplace scenarios ("Imagine you are in a museum") and three episodic future thinking scenarios that were explicitly self-relevant ("Imagine the next holiday").

In the second part of each trial, participants were asked to provide more detail if possible. Subsequently, participants were asked to rate their sense of presence in the image (1, "did not feel like I was there at all;" 5, "felt strongly like I was really there") as well as their sense of salience (1, "couldn't really see anything;" 5, "extremely salient"). Finally, a series of twelve sentences were read and the participant responded whether they felt the statement applied to their image of the scenario ("It was quite fragmented"). These probe questions were designed to assess the spatial coherence of the participants' image. Participants were recorded with GarageBand (Apple, Cupertino, CA) or Audacity (Freeware) software for off-line analysis.

7.3. Scoring

We applied the scoring methodology used by Hassabis et al. (2007) and summarized here. For each trial, the comprehensive Experiential Index (EI) score (score: 0–60) totaled the subscales for content, participant ratings, spatial coherence, and quality rating. The content score (0–28) tallied details referring to spatial reference, entity presence, sensory description, or thought/emotion/action. The number of details for each category were counted and capped at seven and summed. The participants' ratings of presence and salience were each rescaled to range from 0 to 4, both values were included in the EI score. The spatial coherence index assessed the responses to the twelve probe questions and assigned one point for each response demonstrating spatial coherence (e.g. "I could see it as one whole scene in my mind's eye") and subtracted a point for each response indicating fragmentation (e.g. "It wasn't so much a scene as a collection of separate images"). These values were summed and rescaled to a range of −6 (fragmented) to +6 (coherent). Only positive values were included in the EI score. The last component of the EI was the experimenter's assessment of the participants' memory quality as a function of completeness, vividness, and richness. Quality was measured on a scale of 0 (no image) to 10 (vivid image). This quality rating value was multiplied by 1.8 for range of 0–18. To summarize, the content score (0–28) + presence (0–4) + salience (0–4) + spatial coherence (0–6) + quality rating (0–18) were summed to create the EI (0–60). Two raters independently scored every scenario and agreement between scorers was high (intraclass correlation = .97). The raters' scores for each constructed experience were averaged and the averaged scores were subjected to statistical analyses.

7.4. Analysis

The content, participant ratings, spatial coherence index and quality rating measures were calculated and summed to determine the EI. The data from the commonplace events and episodic future thinking scenarios were collapsed because

Table 2
Experiment 2 results per measure. The group mean and standard error of the mean for each measure are presented. Asterisks indicate that the patient group was significantly impaired when compared to the control group.

Measure	Mean (SD)		
	PPC patients, $n=2$	PFC patients, $n=4$	Controls, $n=10$
Overall richness: experiential index	26.67 (1.3)*	36.35 (3.0)*	50.88 (1.2)
Subcomponents			
Content			
Spatial references	0.87 (.5)*	2.14 (.6)	4.23 (.3)
Entities present	4.29 (.6)*	5.27 (.5)*	6.66 (.1)
Sensory descriptions	3.37 (.9)*	3.70 (.7)*	6.51 (.1)
Thought/emotions/actions	3.64 (.1)*	4.89 (.5)*	6.79 (.1)
Participant ratings			
Sense of presence	2.64 (.1)	4.20 (1.9)*	3.07 (.2)
Perceived salience	2.64 (.1)	4.30 (.4)*	3.34 (.2)
Spatial coherence index	2.40 (.9)*	3.52 (.5)*	5.92 (.04)
Scorer rating: quality judgment	7.02 (.1)*	7.91 (1.2)*	14.57 (.5)

there was no difference in performance between scenario types. The mean scores for each measure and group are shown in Table 2. Because there were three participant groups we elected to use the non-parametric Kruskal–Wallis one-way analyses of variance (ANOVA). Subsequently, as in Experiment 1, we determined which group difference drove the observed main effect by conducting Mann–Whitney's *z*-tests between each patient group and the control group. The two-tailed significance values are reported for these scores. In order to compare the performance of the PFC group with the PPC group, we calculated Mann–Whitney's *z*-tests between the two patient groups.

8. Results

8.1. Experiential index (EI)

The overall ability to imagine a scenario was comprehensively measured by the EI; see Fig. 3. A non-parametric Kruskal–Wallis's one-way ANOVA was performed using the data from the three groups. The analysis indicated a significant effect of group ($\chi^2_{(2)} < 12.11$, $p = .002$). In order to determine whether each patient group was significantly impaired with regard to the control group, we conducted Mann–Whitney's tests. These comparisons indicate that both patient groups had significantly lower EI scores than controls (PPC: $z = -2.15$, $p = .03$, PFC: $z = -3.06$, $p = .002$). This finding suggests that patients with PFC or PPC damage have a diminished ability to imagine events. To evaluate patients' deficits in detail, EI subcomponents were considered separately.

8.2. Experiential index subcomponents

8.2.1. Content

The numbers of details for each of four categories – spatial references, references to entities present in the environment, number of sensory details, number of references to own thoughts/emotions/actions – were compared across groups (controls, PPC, PFC) using Kruskal–Wallis tests and then by pairwise Mann–Whitney's tests. For the number of spatial references there was no significant main effect of group ($\chi^2_{(2)} < 5.24$, $p = .07$), although Mann–Whitney's pairwise tests found a significant impairment in the PPC group when compared to the controls ($z = -2.15$, $p = .03$). There was a significant effect of group in the three remaining subcomponents: references to entities present in the environment ($\chi^2_{(2)} < 9.65$, $p = .008$), the number of sensory details ($\chi^2_{(2)} < 9.71$, $p = .008$), the number of references to their own thoughts/emotions/actions ($\chi^2_{(2)} < 12.09$, $p = .002$). Both the PPC and PFC groups were impaired relative to controls across these three subcomponents (all p's < .03).

8.2.2. Participant self-ratings

In the self-ratings portion, the participants rated their own constructed experiences along dimensions of presence and salience. There was a main effect of group membership for presence ($\chi^2_{(2)} < 7.26$, $p = .03$) and salience ($\chi^2_{(2)} < 7.44$, $p = .02$). Mann–Whitney's tests suggest that this effect was driven by the PFC group who gave their constructed experiences inflated ratings despite describing impoverished constructed experiences (presence: $z = 2.33$, $p = .02$, salience: $z = 2.08$, $p = .04$). In contrast, the PPC group gave their constructed experiences numerically, but not statistically, lower ratings, than did the control group (p's > .12). This finding indicates that PFC patients inaccurately provided inflated ratings, while the PPC patients accurately gave their impoverished constructed experiences low ratings.

8.2.3. Spatial coherence index

Spatial coherence measured the integrated nature of the participants' constructed experiences. The spatial coherence index was calculated based on the participants' responses to the twelve probe questions. On this measure, the patients were significantly lower than controls ($\chi^2_{(2)} < 12.81$, $p = .002$). Mann–Whitney's tests revealed significant effects in both patient groups (both p's < .02). These results indicate that both PFC and PPC patients constructed experiences that were significantly more fragmented than those of the control participants.

8.2.4. Quality judgment

For every memory, the experimenters rated the overall quality of the constructed experiences. The rating for each scenario was compared between groups. These ratings demonstrated significantly lower quality ratings for the patient group ($\chi^2_{(2)} < 11.71$, $p = .003$). Mann–Whitney's tests demonstrate significant effects in both patient groups (both p's < .03).

8.3. Between patient groups comparison

The small number of patients in the PPC and PFC groups provided limited power to assess differences between the patient groups. Two measures did reveal a statistically significant difference between groups, the self-rating measures of presence (Mann–Whitney's $z = -1.94$, $p = .05$) and salience ($z = -1.94$, $p = .05$). In these measures, the PFC patients had significantly higher self-ratings than the PPC patients. However, across all measures, there was a numerical difference such that the PFC group always performed better than did the PPC group. To illustrate this difference, Fig. 4 presents the difference score for each of the measures.

8.4. Experiment 1 and Experiment 2 comparison

Because we used previously published tasks with different scoring measures, we turned to standardized z-scores to permit a comparison across the autobiographical memory task (Experiment 1) and the constructed experience task (Experiment 2). For Experiment 1, we separately tallied all of the internal and external details from the free recall portion. The details were separated following the suggestion that the internal details may best reflect episodic memory performance whereas the external details may primarily reflect semantic memory performance. The specific probe measures were not included because there was no analogous measure in Experiment 2. For Experiment 2, the Experiential Index (EI) measure was selected. This measure provides a measure of overall performance. Standardized scores are a very conservative measure of performance. As shown in Fig. 5, only the PPC patients showed impairment for the autobiographical memory task (Internal: *M* PPC = −1.29, *M* PFC = .41; External: *M* PPC = −1.38, *M* PFC = −.63), although both groups were considerably impaired at the constructed experience task (*M* PPC = −6.26, *M* PFC = −3.77). However even here, the PPC patients were relatively more impaired (6 standard deviations from the mean versus more than 3 standard deviations).

9. Discussion

Experiment 2 confirmed that the PPC patients were impaired across most measures. Unexpectedly, the PFC group was also impaired across most measures. Both the PPC and PFC patients mentioned fewer items in the environment, and described fewer sensory and spatial details, suggesting that their constructed experiences were impoverished. The PPC and PFC patients made relatively fewer comments about their own thoughts, emotions, and actions, suggesting that their representation of themselves in a constructed scene was also deficient. Both PPC and PFC patients had lower spatial coherence indices, indicating that their constructed experiences lacked a coherent spatial framework.

Although the PPC and PFC groups were both impaired, there were notable patient group differences. The PFC group consistently performed better than the PPC group across all measures. These differences reached statistical significance when the patients themselves were asked to rate their own sense of presence and salience. The PFC patients overestimated the quality of their constructed experiences. In contrast, the PPC patients gave themselves lower ratings, indicating that they more accurately assessed the quality of their constructed experiences. The PPC patients also made fewer spatial references, perhaps due to their impoverished spatial imagery (Berryhill et al., 2007).

It is worth noting that when our PPC patients were compared to the MTL amnesics studies by Hassabis et al. (2007) the PPC patients exhibited lower performance on the spatial references component but higher performance on the spatial coherence index. As a point of clarification, these two spatial measures arise from different sources. The spatial references component measure reflects the number of spatial references included in the constructed experience. It is not surprising that the PPC patients mentioned fewer spatial details given the spatial deficits associated with parietal lobe damage, and the deficits in these patients' spatial imagery (Berryhill et al., 2007). In contrast, the spatial coherence index reflects the participants' subjective responses to the questions aimed at assessing the quality of spatial integration in their constructed memories. We suspect that the PPC patients responded that their memories were highly integrated because they are somewhat insensitive to their impaired spatial perception.

10. General discussion

The question posed by this study was whether portions of the PPC play a functional role in constructed experience. To assess this possibility, we compared patients with bilateral PPC damage to control participants and to a lesion control group, patients with unilateral lesions in ventrolateral or dorsal PFC.

We first tested whether the PFC patient group exhibited the same pattern of episodic memory retrieval deficits that we had previously observed in the PPC patients (Berryhill et al., 2007). In Experiment 1, the PFC group performed normally on the autobiographical memory test (Levine et al., 2002). This finding established that brain damage alone could not account for our earlier findings and also that the PFC group was an appropriate lesion control group.

In Experiment 2, PPC, PFC and control groups were tested on a constructed experience task. Participants were asked to describe commonplace scenarios, such as going to the beach, with the caveat that they be imagined (Hassabis et al., 2007). As predicted, the PPC group showed impaired performance when compared to controls across most measures. An unanticipated result was that the PFC group was also impaired across most measures. The PPC and PFC patients described events with fewer details across all categories including spatial, sensory, objects, and actions. Finally, standardized scores were derived for each task to permit between-task comparisons. These scores clearly showed that performance for both patient groups was markedly worse for Experiment 2 than Experiment 1.

10.1. Constructed experience task and cognitive demands

In the constructed experience task, we anticipated the poor performance of the PPC group, given their impaired free recall on a similar task that required them to recall autobiographical memories (Berryhill et al., 2007). However, we were surprised by the moderate impairment shown by the PFC group since they were unimpaired at the autobiographical memory task. These high-functioning PFC patients have no language deficits, and do not exhibit signs of significant impairment associated with dorsal or ventrolateral PFC damage—for example, many of them continue to work full-time jobs. There were no systematic differences in performance as a function of lesion location (dorsal or ventrolateral, left or right hemisphere). Nor do these PFC patients have damage in the ventromedial PFC regions typically activated in fMRI studies of constructed experience (reviewed in (Buckner & Carroll, 2007; Gilbert & Wilson, 2007; Schacter & Addis, 2007; Schacter et al., 2007, 2008); see also (Hassabis et al., 2007). These findings raise the possibility that *any* kind of cortical brain damage impairs constructed experience, at least as measured by the present task. Several other studies describe impaired constructed experience following MTL damage (Hassabis et al., 2007; Jensen et al., 2008; Klein, Loftus, & Kihlstrom, 2002; Tulving, 1985) or other disorders such as depression or schizophrenia (discussed in Addis, Pan, Vu, Laiser, & Schacter, 2009), which impaired episodic memory. Here we found that PPC *or* PFC damage impairs constructed experience. In the case of the PFC group, we find an asymmetry such that only constructive memory is impaired while autobiographical memory is preserved. As noted, the diversity of lesion location and volume in the reviewed sample raises the question of whether constructed experience can be pinpointed to a specific brain region or even to a clear set of cognitive processes.

The constructed experience task we chose has a number of favorable features—it is simple to administer and appropriate for use with neuropsychological patients. However, closer examination of the task suggests that it requires a heterogeneous mix of cognitive functions including strategic planning and utilization of a narrative structure, mental imagery, working memory,

self-reflection, and episodic memory. Consequently, brain damage in any number of regions may impair performance, but for different root causes. These observations suggest that a constructed experience task with greater specificity is needed before strong conclusions regarding the neural correlates of constructed experience can be made. This challenge compounds the intrinsic difficulties in examining constructed experience: it cannot be externally verified, as the events have never happened. Thus, the present data cannot rule out the possibility that poor performance is a general effect of brain damage.

10.2. Frontal lesions and constructed experience

Here, with the benefit of hindsight we reevaluate the broad cognitive demands imposed by the constructed experience task and identify functions associated with PFC and PPC function that might help explicate these findings. As mentioned, one possibility is that the constructed experience task imposed broad cognitive demands, leading to the recruitment of frontoparietal networks. Frontoparietal networks are frequently activated in cognitive tasks requiring attention, working memory, or memory retrieval (reviewed in Naghavi & Nyberg, 2005). The role of the PPC in constructed experience may relate to its poorly understood involvement in episodic memory retrieval (reviewed in Ally, Simons, McKeever, Peers, & Budson, 2008; Cabeza, 2008; Cabeza et al., 2008; Olson & Berryhill, 2009; Wagner et al., 2005), discussed below. In contrast, ventrolateral and dorsal PFC contributions may be associated with selection and organizational functions during episodic retrieval. Ventrolateral PFC is recruited in tasks requiring conceptual or perceptual selection and is associated with the cognitive control of episodic memory (reviewed in Badre & Wagner, 2007). Lesions in these lateral areas might impair the ability to access and select elements from memory to create a constructed experience. The dorsal PFC is recruited to organize the selected information for working memory manipulation (reviewed in Blumenfeld & Ranganath, 2007). Lesions in these areas might prevent patients from coherently integrating relevant memory elements in a successful collage.

A second reason we might have anticipated PFC impairment is due to the high degree of interconnectivity between PPC and PFC regions (reviewed in Goldman-Rakic, 1988; Olson & Berryhill, 2009). Damage to either node might lead to significant impairment upstream or downstream. Thus, the degree of PPC-PFC interconnectivity and roles in strategic planning might explain why the PFC group was impaired at the constructed experience task. However, in order to account for the normal performance demonstrated by the PFC group in the autobiographical memory task, it presupposes that greater strategic planning processes are required for constructing novel experiences.

10.3. Theoretical explanations of memory deficits after parietal lobe damage

These data support the view that the PPC is functionally involved in episodic memory (reviewed in Olson & Berryhill, 2009). This result is consistent with the view that constructed experience uses episodic retrieval as the source material for constructing new experiences. However, constructed experience paradigms were not designed to isolate component memory processes. Thus, interpreting the significance of the constructed experience deficits in PPC and PFC patients remains an open challenge.

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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.2010.01.004.

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