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Impaired perception of mnemonic oldness, but not mnemonic newness, after parietal lobe damage

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ABSTRACT

In studies of episodic memory retrieval, recognition paradigms are known to elicit robust activations in the inferior parietal lobe. However, damage to this region does not produce severe deficits in episodic memory performance as indexed by typical accuracy measures. Rather, because problems with memory confidence are frequently reported, the observed deficits may be best described as "metamemory" or subjective memory deficits. Here, we further investigated the inferior parietal lobe's role in recognition memory as well as metamemory. We tested the hypothesis that the inferior parietal lobe gauges the perceived oldness of items, given several neuroimaging findings suggesting that a portion of the left inferior parietal lobe is sensitive to perceived oldness. We tested two patients with bilateral parietal lobe lesions and matched controls on an old/new recognition task. From these data we constructed receiver operating characteristic (ROC) curves by fitting the data with the unequal-variance signal-detection (UVSD) model. The results revealed no memory impairment in terms of patients' accuracy. However, patients exhibited lower hit rates and false alarms rates at high confidence levels. Further, patients and controls differed in how they set decision criteria for making recognition responses. Patients' decision criteria for "old" responses were shifted in a conservative fashion such that they were unwilling to endorse recognized target items with high levels of confidence. These findings provide constraints on models of inferior parietal lobe contributions to episodic memory retrieval.

1. Introduction

Memory researchers have frequently reported activations in the inferior parietal lobe during neuroimaging studies of episodic memory retrieval (for reviews see Cabeza, Ciaramelli, & Moscovitch, 2012; Vilberg & Rugg, 2008; Wagner, Shannon, Kahn, & Buckner, 2005). Recognition memory paradigms, in particular, are among the most frequent to elicit parietal lobe activations (e.g. Cabeza et al., 2012; Hayes, Buchler, Stokes, Kragel, & Cabeza, 2011; Wagner et al., 2005). Several models have been proposed to explain these findings, including the *attention to memory model* (Cabeza, Ciaramelli, Olson, & Moscovitch, 2008), the *memory buffer hypothesis* (Vilberg & Rugg, 2008), the *subjective recollection hypothesis* (Ally, Simons, McKeever, Peers, & Budson, 2008), and *mnemonic accumulator* accounts (Donaldson, Wheeler, & Petersen, 2010; Gold & Shadlen, 2007; McClelland, 2001; Ratcliff, 1978).

Some of these models have encountered the problem that there is little converging evidence for the robust fMRI findings. Although amnesic patients with damage to the hippocampus exhibit severe recognition deficits, damage to the parietal lobe does not lead to severe or consistent recognition memory deficits (for a review of this paradox, see Schoo et al., 2011). Patients do not appear to be amnesic (Berryhill, Phuong, Picasso, Cabeza, & Olson, 2007), and overall free recall and recognition accuracy in episodic memory paradigms are not impaired (Haramati, Soroker, Dudai, & Levy, 2008; Berryhill, Drowos & Olson, 2009; Dobbins, Jaeger, Studer, & Simons, 2012; Drowos, Berryhill, Andre, & Olson, 2010; Simons, Peers, Mazuz, Berryhill, & Olson, 2010).

However, there are often subtle impairments in specific memory processes (reviewed in Table 1). For instance, Berryhill et al. (2007) reported that when patients were asked to freely recall autobiographical events, their recollections lacked the richness and specificity of control participants. Even more compelling is the accruing body of findings showing that patients with parietal lobe damage have diminished memory confidence coupled with intact memory accuracy. For example, Simons et al. (2010) reported that parietal patients showed reduced confidence in their own recollections although their source memory accuracy was at normal

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Table 1
Summary of episodic memory studies conducted in patients with unilateral or bilateral parietal lobe lesions in which memory confidence or vividness was assessed. Confidence is marked as impaired if a decreased number of 'remember' responses were reported. NA=not assessed. Multiple entries refer to experimental results in multi-experiment papers.

Citation	Memory task	Memory performance		Confidence	
		Spared	Impaired	Spared	Impaired
Berryhill et al. (2009)	Audio-visual pairs	√			√
Berryhill et al. (2007)	Autobiographical memory; free recall		√- fewer episodic details	NA	NA
Berryhill et al. (2007)	Autobiographical memory; cued recall	√		NA	NA
Berryhill et al. (2010)	Constructed experience episodic future thinking		√- fewer episodic details	NA	NA
Davidson et al. (2008)	Source memory; remember/know	√			√- decreased 'remember' responses
Davidson et al. (2008)	Autobiographical memory; remember/know	√		√	
Davidson et al. (2008)	Autobiographical memory; cued recall		√- fewer episodic details	NA	NA
Drowos et al. (2010)	False memory; recognition		√- impaired performance was caused by patients reporting few "old" responses, causing a low rate of false memories.		√- decreased high confidence 'old' responses
Drowos et al. (2010)	False memory; recognition		√- impaired performance was caused by patients reporting few "old" responses, causing a low rate of false memories.		√-decreased high confidence 'old' responses
Simons et al. (2010)	Source memory; recognition	√			√
Simons et al. (2010)	Source memory; recognition	√		√	
Simons et al. (2010)	Source memory; recognition	√			√

levels. In another study, patients were tested on a remember/know false-memory paradigm in which the patients exhibited fewer "remember" responses but gave more "know" responses than controls on lure trials reflecting their lower degree of confidence (Drowos et al., 2010). Consistent with these findings, several studies in healthy individuals have reported that BOLD activations in the lateral parietal cortex increased with increasing subjective confidence ratings of recognition responses (Cabeza et al., 2012; Johnson, Suzuki, & Rugg, 2013; Vilberg & Rugg, 2008; Yonelinas, Otten, Shaw, & Rugg, 2005).

These findings have led some researchers to argue that the parietal cortex plays a key role in metamemory processes (construed as more subjective processes), rather than core memory processes (more objective processes) (Chua, Schacter, & Sperling, 2009; Elman, Klostermann, Marian, Verstaen, & Shimamura, 2012; Rugg & Vilberg, 2013). Metamemory processes encompass self-monitoring strategies that are engaged during the various stages of memory encoding, storage, and retrieval. In fact, researchers often examine monitoring during memory retrieval by collecting information about participants' retrospective confidence judgments and thresholds for setting response criteria (Modirrousta & Fellows, 2008; Pannu & Kaszniak, 2005). It is hypothesized that such monitoring techniques may interact with core memory processes in order to facilitate overall memory performance; however, it should be noted that there is an on-going debate regarding the nature of the relationship between confidence ratings and memory accuracy (see Roediger, Wixted, & DeSoto 2012 for a review).

Ally et al., 2008 proposed that parietal lobe activity indexes the subjective experience of remembering (termed the *subjective recollection account*). This signal presumably allows participants to distinguish between vividly recollected and vaguely recollected information (Ally et al., 2008). Related to this, Wheeler and Buckner (2003) argued that fMRI findings showing parietal lobe activity during memory retrieval reflect the "perception of oldness" of items. This idea was based on their finding, as well as that of other investigators (Donaldson, Petersen, Ollinger, & Buckner, 2001; Habib & Lepage, 1999; Henson, Rugg, Shallice, Josephs, & Dolan, 1999; Konishi, Wheeler, Donaldson, & Buckner, 2000; McDermott, Jones, Petersen, Lageman, & Roediger, 2000), showing that a small region of the left inferior parietal lobe is sensitive to perceived oldness, but not perceived newness, of items. Thus, it is possible that the metamemory impairments exhibited by patients with parietal lobe lesions reflect problems perceiving that a test item is actually old. In other words, prior neuroimaging data suggest that the confidence in an item being old does not become sufficiently high until the memory signal is exceptionally strong.

The aim of the current study was to further investigate whether the inferior parietal lobe is critical in metamemory. We tested two well-characterized patients with bilateral parietal lobe lesions on a standard old/new receiver operating characteristic (ROC) paradigm (Egan, 1958; Yonelinas, Kroll, Dobbins, Lazzara, & Knight, 1998). ROC analysis is one of the most common ways to study recognition memory and is often coupled with signal detection analyses. We used this approach because it allowed us to assess how patients and controls set their decision criteria for making recognition decisions associated with differing levels of confidence. Generally speaking, confidence varies with the degree of perceived oldness associated with a test item and also with the degree of perceived newness associated with a test item. Our question was whether parietal lesions symmetrically affect the subjective experience of oldness and newness, or whether they instead selectively affect the subjective experience of oldness (while leaving the subjective experience of newness intact).

We predicted that patients with bilateral parietal lobe lesions would show normal memory accuracy coupled with abnormal memory confidence, in line with prior neuropsychological findings (Berryhill et al., 2009; Davidson et al., 2008; Simons et al., 2010). We further predicted that patients would show marked differences in their ROC curves compared to controls due to problems in perceiving, accumulating, or deciding about the oldness of information (in line with the subjective recollection account). If the problem is limited to the perceived oldness of information based on the recollection of episodic detail, then the patients should be reluctant to make high-confidence "old" decisions but should not show a similar reluctance to make high-confidence "new"

decisions (because such decisions are based on a familiarity or novelty signal that is presumably devoid of recollection). More specifically, we predicted that ROC analysis would show that patients and control participants differ in terms of how they set various decision criteria for deciding whether items are old or new. We expected that patients would exhibit a marked shift towards more conservative responses for deciding that items are old, resulting in far fewer high confidence "old" ratings compared to controls. At the same time, we expected that patients would set their criteria for confidently deciding that items are new in approximately the same location that controls do, resulting in a similar number of high confidence "new" ratings. This pattern would suggest that parietal lesions do not result in a general reduction in confidence associated with making old/new recognition decisions; instead, the impairment would be limited to making metamemory decisions based on degrees of recollection associated with episodic detail.

2. Materials and methods

2.1. Participants: lesion patients

Two patients with bilateral parietal lobe damage, EE555 and TQ591, were tested in this study. Their lesions can be viewed in Fig. 1 (reprinted from Berryhill, Chein, & Olson, 2011). They have been discussed extensively in prior studies (Berryhill et al. 2007, 2009, 2011; Berryhill & Olson, 2008; Berryhill, Picasso, Arnold, Drowos, & Olson, 2010; Drowos et al., 2010); we summarize their neurological profiles here.

2.1.1. Patient EE555

EE555 is a 40-year-old former teacher with 16 years of education. In 2004, she suffered three infarcts in the watershed between the posterior and middle cerebral arteries. Her physical and perceptual symptoms are currently stable. EE555's MRI revealed symmetrical lesions in lateral aspects of the inferior parietal lobe, extending from superior aspects of the occipital lobe through the angular gyrus (Brodmann area (BA) 39) in and around inferior and middle portions of the intraparietal sulcus (IPS). Damage does not encroach into the midline (e.g. precuneus).

Patient EE555's primary deficit is simultanagnosia. When shown a line drawing of a visual scene she describes parts of the picture, 'there is a woman', and 'I see water', without attaining a global understanding of the scene. In line cancellation tasks, she crosses off items only at the center, ignoring peripheral items. She only reports the local elements when shown Navon letters. Language comprehension and speech fluency were unimpaired as assessed by her conversational skills, and by ceiling performance on the auditory tests of the Western Aphasia Battery. Her eyesight is normal.

2.1.2. Patient TQ591

TQ591 is a 49-year-old former preschool assistant teacher with 15 years of education. She suffered bilateral parieto-occipital damage due to CNS cerebral vasculitis in March 2006. TQ591's MRI revealed signs of previous subacute posterior cerebral artery infarctions. The primary lesions are in bilateral parietal regions. The left parietal lesion extends into IPS (BA 39) and precuneus (BA 7). There are two right lesion sites: the inferior lesion is in superior aspects of the occipital lobe (BA 18 and 19), and the superior lesion is in the superior parietal lobe (BA 7). In both hemispheres, the lesions extend slightly into occipital (BA 19) regions and parietal white matter.

TQ591's primary deficit is simultanagnosia. When shown pictures of scenes TQ591 is slow to describe them and complains that parts of scenes "disappear" when she looks away or blinks. In line cancellation tasks, she only identifies a few lines within a narrow visual field. She has a local bias with Navon letters. Language comprehension and speech fluency were unimpaired as assessed by her conversational skills, and ceiling performance on the auditory tests of the Western Aphasia Battery. Reading is somewhat impaired due to her simultanagnosia. Her vision is corrected-to-normal.

2.2. Neuropsychological evaluation of memory and language

In a prior study, we reported scores on standard neuropsychological evaluations of language and memory (Berryhill & Olson, 2008; Drowos et al., 2010). This testing found no evidence of aphasia or gross disturbances in the retrieval or use of semantic memory. Some dysfunction was evident in auditory subtests of the Wechsler Memory Scale (WMS-III, The Psychological Corporation), in item memory on the Logical Memory I and II subtests of the WMS-III, and in autobiographical memory as assessed by the Autobiographical Memory Inventory (AMI) (Kopelman, Wilson, & Baddeley, 1989). Performance was normal on other subscales of the WMS-III such as immediate and delayed memory.

2.3. Control participants

15 normal controls (10 males, 5 females) that were matched in age ($M=45.2$, range=35–52) and education ($M=13.8$, range=12–16) to the two patients were tested. Two controls were excluded because task performance fell below chance, resulting in an N of 13. Even after these exclusions, there were no differences between patients and controls in terms of age and education ($p>.76$ and $p>.11$, respectively). All control participants were given a short questionnaire to verify that they were not experiencing any neurological or psychiatric disorders at the time of testing. All participants were compensated for their participation in the experiment and signed consent documents. The experimental protocols were approved by the Institutional Review Boards of the University of Pennsylvania and Temple University.

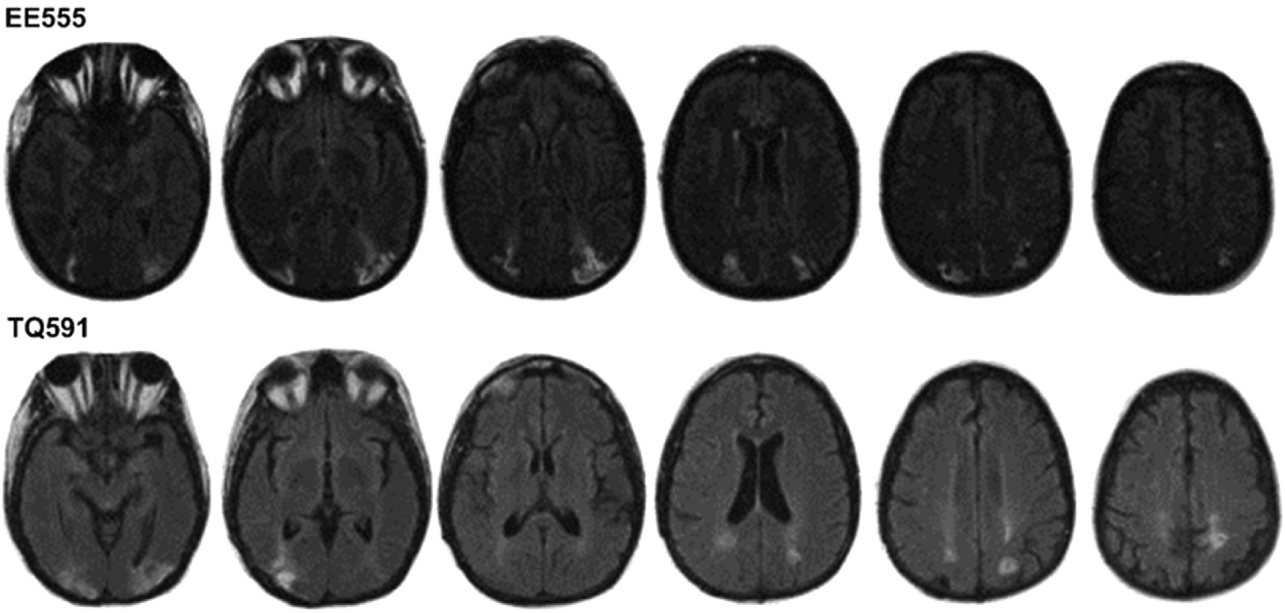

Fig. 1. Lesion tracings (from Berryhill, Chein, & Olson, 2011). Lighter hypodensities represent the lesioned regions in patients EE555 (top) and TQ591 (bottom).

2.4. Equipment

Participants were tested in their homes on a Dell laptop computer with a 15-inch monitor or in the laboratory on a Dell desktop computer using ePrime 2.0 software (Psychology Software Tools, PA, USA).

2.5. Stimuli

The stimuli consisted of 240 nouns (Kucera-Francis word frequency between 10–12) extracted from the MRC Psycholinguistic Database (Coltheart, 1981). Stimuli were presented audiovisually. The visual presentation of the words was in Courier Bold font, size 18. The audio presentation of the words was in a female voice. Volume was adjusted prior to the experiment to make sure that all participants could hear the audio files.

2.6. Task

There were two phases of the experiment: encoding and retrieval (see Fig. 2). During the encoding phase, a word was presented both visually and aurally. Based on Paivio's (1991) dual encoding theory, presenting test items in multiple modalities has an additive effect and enhances the degree to which items are encoded. Therefore, since the present study included patients with sensory deficits, dual presentation was utilized to ensure that the patients were able to encode test items adequately. The word remained visible until a response was entered. Each participant completed two separate 80-trial blocks of deep and shallow encoding. In the shallow encoding condition, participants reported the number of syllables in the word (keys 1–5). In the deep encoding condition, participants reported whether the stimulus word referred to something concrete or abstract by depressing keys 'a' or 'c'. The order of encoding conditions was randomized. There were two versions of the encoding task to counterbalance which judgment was made for each word.

During the retrieval phase, participants viewed and heard all of the stimuli presented during encoding (80 shallow, 80 deep) as well as an additional 80 new lure words for a total of 240 retrieval trials. At retrieval, participants judged whether the probe word was old or new using scores from 1 to 6 (1=Sure New, 2=Probably New, 3=Maybe New, 4=Maybe Old, 5=Probably Old, 6=Sure Old) to reflect their confidence. Again, the word remained visible until a key press response was registered.

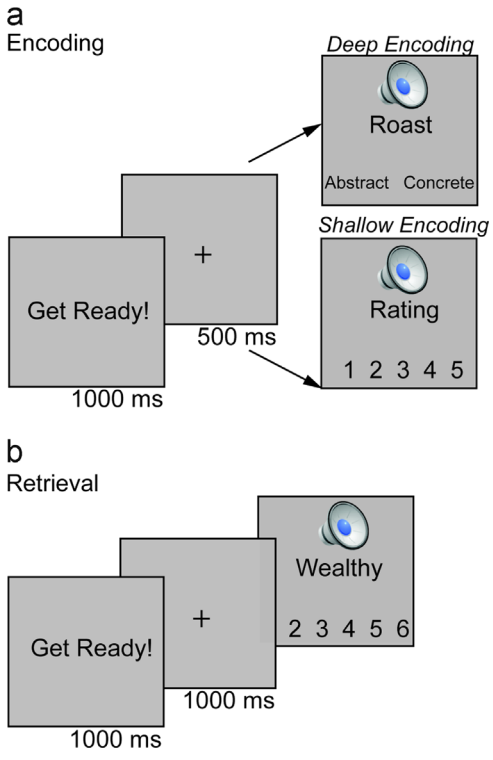

Fig. 2. Trial design. The trial sequences for the (a) encoding, and (b) retrieval phases are shown. The speaker symbol indicates that the stimuli were presented aurally as well as visually. In the deep encoding condition, participants reported whether the word was abstract or concrete. In the shallow encoding condition, participants reported the number of syllables in the word. During retrieval, participants reported whether the probe item was old or new using a 1-6 scale.

These confidence ratings were used to construct ROC curves (Egan, 1958; Macmillan & Creelman, 2005), which were then analyzed using the standard unequal-variance signal-detection (UVSD) model. ROC curves plot participants' hit rates (the proportion of target items correctly identified as old) against false alarm rates (the proportion of foil items incorrectly identified as old) for the varying levels of confidence. To construct an ROC curve, hit rate versus false alarm rate pairs are calculated with respect to each confidence rating. The leftmost point on the ROC curve represents the hit rate versus false alarm rate pair for the highest "sure old" confidence rating of 6. For this point, the hit rate reflects the proportion of targets that received a confidence rating of 6, and the false alarm rate reflects the proportion of foils that received a confidence rating of 6. The next point on the curve represents the proportion of hit rates versus false alarm rates for items that were endorsed with confidence ratings of either 5 or 6. Successively increasing points on the ROC curve are incorporated cumulatively (see Fig. 4).

2.7. Analysis

The UVSD model assumes that the targets and lures on a recognition memory test are each associated with a distribution of memory strength values (Fig. 3). The mean and standard deviation of the target distribution (d and σ, respectively) are both assumed to exceed the mean and standard deviation of the lure distribution (set to 0 and 1, respectively). The assumption that σ exceeds 1 (i.e., the assumption that the standard deviation of the target distribution exceeds the standard deviation of the lure distribution) is based on prior work involving ROC analysis (Egan, 1958; Ratcliff, Sheu, & Gronlund, 1992). Confidence ratings for each test item on a 1-to-6 scale are assumed to be made with respect to 5 confidence criteria arrayed along the memory strength axis. A test item (i.e., a target or a lure) with a memory strength that exceeds the c6 criterion receives a confidence rating of 6; a test item with a memory strength that exceeds the c5 criterion (but not the c6 criterion) receives a confidence rating of 5, and so on. A test item with a memory strength that is so low that it does not even exceed the c2 criterion receives a confidence rating of 1.

The UVSD model is fit to confidence-based ROC data using maximum likelihood estimation (Macmillan & Creelman, 2005). The fit involves estimating 7 parameters: d and σ (the mean and standard deviation of the target distribution relative to the foil distribution), plus the locations of the 5 confidence criteria (c2, c3, c4, c5 and c6). As shown in Fig. 3, c2 is placed slightly to the left of the mean of the foil distribution. Because the mean of the foil distribution is set to 0, c2 in this case would be slightly negative (its exact value in Fig. 3 is −0.5). c4 is what is often thought of as the *decision criterion* because it is the criterion that separates "new" decisions (confidence ratings of 1, 2 or 3) from "old" decisions (confidence ratings of 4, 5 or 6).

The UVSD model can be fit to group data (with the confidence ratings pooled across the 2 patients and, separately, across the 13 controls) as well as to each individual participant's data. We fit the data both ways, and the conclusions were the same in either case. Once the parameters are estimated, either for a group or for an individual participant, two derivative measures can be computed. One derivative parameter is the "slope" parameter, which is simply equal to $1/\sigma$. For example, if σ is estimated to be 1.25, the slope estimate would be 1/1.25=0.80. This parameter is referred to as the "slope" parameter because it corresponds to the slope of a straight line that characterizes another common way of plotting ROC data, which involves plotting z-transformed hit and false alarm rates. Much prior research shows that σ is usually greater than 1 when standard ROC data are analyzed, which means that the slope would be less than one if z-ROC data were analyzed instead. A second derivative parameter is d_a. This is a discriminability parameter that is much like the familiar d' score except that d_a takes into account the fact that the standard deviation of the target distribution exceeds that of the foil distribution. By contrast,

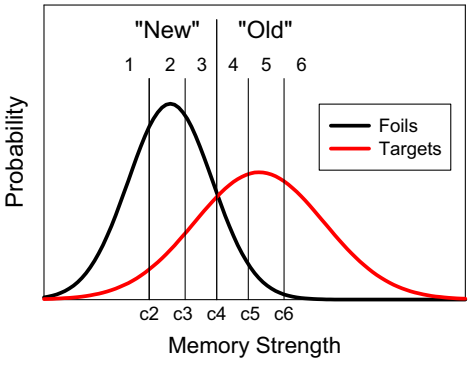

Fig. 3. An illustration of the unequal-variance signal-detection (UVSD) model. The black line reflects foil distribution; the red line reflects target distribution, and c2-c6 reflects decision criteria based on confidence ratings. (For interpretation of references to color in this figure legend, the reader is referred to the web version of this article.)

d' implicitly assumes an equal variance model. For that reason d_a is a more accurate measure of discriminability (though it usually yields conclusions that are similar to those based on the less accurate d' measure). Discriminability refers to the ability of participant to tell the difference between targets and foils. Theoretically, d_a quantifies the degree of overlap between the target and foil distributions, and it is given by $d_a = d/\text{sqrt}[.5^*(1+\sigma^2)]$. A d_a value of 0 would reflect a complete inability to differentiate targets from foils (i.e., the target and foil distributions would overlap completely); a large value of d_a (e.g., 4 or more) would reflect nearly perfect performance (i.e., the target and foil distributions would scarcely overlap at all). Note that when $\sigma = 1$ (i.e., when the target and foil distributions are estimated to have equal variance), d_a reduces to d (which, in that case is simply an estimate of d').

Our goal in analyzing the ROC data using the UVSD model was to ask whether the placements of the various confidence criteria differed between patients and controls. For example, if patients were generally more reluctant to express high confidence for old or new decisions compared to controls, then their estimated confidence criteria would be more widely separated (i.e., c2 would fall farther to the left and c6 would fall farther to the right) compared to controls. Alternatively, if patients were more reluctant to express high confidence judgments specifically related to old items, the placement of criteria related to new items would remain similar to that of controls; however, their estimated criteria for old items would be spread more widely (i.e. c4–c6 would fall farther to the right) compared to controls. We predicted that patients' confidence criteria would reflect the latter pattern, where reluctance to express high confidence would be specific to old decisions, rather than a general unwillingness to judge any decision with high confidence. Analyzing the ROC data using the UVSD model also allowed us to assess whether there were any differences in discriminability between patients and controls (i.e., do their d_a values differ?). As previous literature does not suggest a robust change in memory accuracy following damage to the parietal lobe, we expected patients' discriminability to be comparable to that of controls.

Although the group sizes were unequal, the variances between groups were homogenous. Therefore, we used standard t-tests.

3. Results

The group ROC data for the patients and controls are presented in Fig. 4. The curved lines show the best-fitting UVSD model to each condition. Table 2a shows the obtained σ and d_a parameter estimates from the deep and shallow conditions. For both groups, σ was greater than 1 (i.e., the slope was less than 1), as is typically true of confidence-based ROC data. Also, the d_a values were similar for patients and controls in both conditions (suggesting no apparent memory impairment for the patients). Table 2b shows the estimated confidence criteria for each group. Note that because the items from the deep and shallow conditions were intermingled on the recognition test, there is only one set of confidence criteria for each group. The results show that the patients and controls placed their leftmost (c2) confidence criterion at about the same place on the memory strength axis (0.22 and 0.15, respectively), just to the right of the mean of the foil distribution (0). This suggests that both groups were similarly inclined to judge an item as new with high confidence. However, for increasing levels of confidence, the criteria are shifted ever more to the right for the patients compared to the controls. This effect is particularly evident for the placement of the rightmost (c6) confidence criterion, which is placed farther to the right for patients (2.97) than for controls (1.36). This result suggests that the patients were more reluctant to make a high-confidence old decision than the controls.

Similar results were obtained when the UVSD model was fit to the ROC data from each individual participant (and the obtained parameter estimates were then averaged) instead of fitting the model to the group data. The data from two controls could not be fit because their responses were not sufficiently spread across the confidence rating scale and were instead concentrated at the extremes (ratings of 1 and 6). For the remaining individual fits, the obtained chi-square goodness-of-fit statistic was not significant for either patient or for 10 of the 11 controls. A non-significant chi-square indicates that the data do not deviate significantly from the model's predictions (i.e., a non-significant chi-square reflects a good fit). However, for one control, the chi-square value was significant, $\chi^2(6) = 17.8$, $p < .01$, indicating that the UVSD model does not accurately characterize the ROC data of that control participant. Because our conclusions are unaffected by the inclusion or exclusion of this control participant, we included these data in our analyses. Table 3a shows the obtained σ and d_a parameter estimates from the deep and shallow conditions based on the individual fits. The values are similar to those based on the group fits shown earlier in Table 2a. As in the group analysis, the

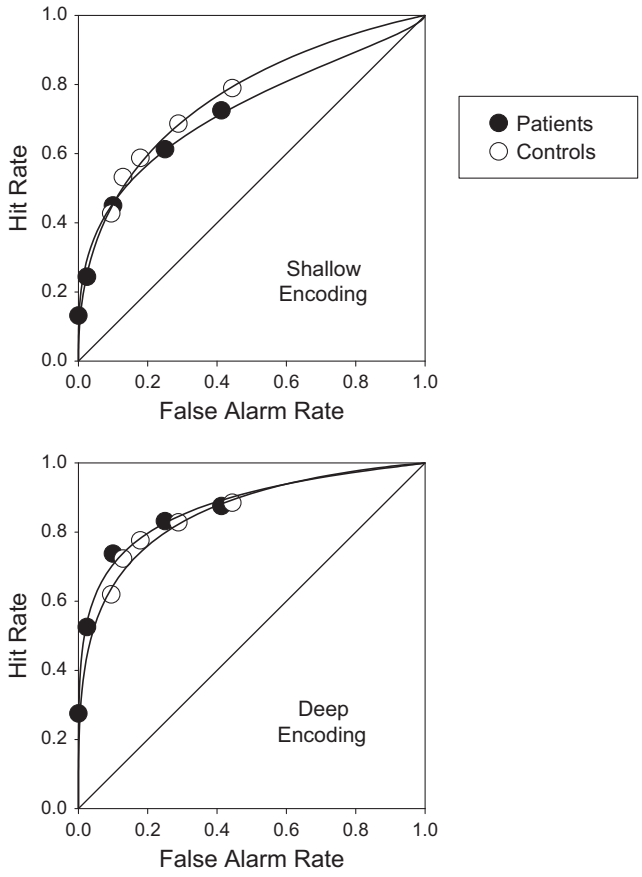

Fig. 4. Group ROC curve fit using the UVSD model for both shallow and deep encoding conditions. Filled circles reflect patient data while open circles reflect controls.

Table 2
UVSD estimates based on group model fit.

(a) Model parameter estimates

Parameter	Patients ($n=2$)		Controls ($n=13$)	
	Deep	Shallow	Deep	Shallow
σ	1.53	1.56	1.31	1.22
d_a	1.63	0.85	1.52	1.02

σ = the mean; da = discriminability parameter that takes into account the fact that the standard deviation of the target distribution exceeds that of the foil distribution

(b) Decision criteria estimates.

Parameter	Patients ($n=2$)	Controls ($n=13$)	Difference
c6	2.97	1.36	1.62
c5	2.07	1.08	1.00
c4	1.26	0.87	0.38
c3	0.66	0.55	0.12
c2	0.22	0.15	0.08

Table 3
UVSD estimates based on individual participant model fits.

(a) Model parameter estimates

Parameter	Patients ($n=2$)		Controls ($n=11$)		$t(11)$	
	Deep	Shallow	Deep	Shallow	Deep	Shallow
σ	1.31	1.39	1.28	1.28	0.09	0.40
d_a	1.84	1.03	1.60	1.01	0.48	0.03

σ=the mean; da=discriminability parameter that takes into account the fact that the standard deviation of the target distribution exceeds that of the foil distribution

(b) Decision criteria estimates.

Parameter	Patients ($n=2$)	Controls ($n=11$)	Difference	$t(11)$
c6	2.95	1.36	1.59	3.08**
c5	2.12	0.91	1.21	2.81*
c4	1.35	0.40	0.95	1.23
c3	0.77	0.24	0.53	0.58
c2	0.28	0.26	0.02	0.55

* Denotes significance at the .05 level,
** Denotes significance at the .01 level.

patients do not show impaired memory and even show a numerical, albeit non-significant, discriminability advantage in the deep condition. Keep in mind that two controls (both of whom were quite accurate) were excluded from this analysis because their data could not be fit by the UVSD model, so the significant advantage for the patients may have emerged for that reason. The key point is that there is no sign of a memory impairment in the patient data.

Table 3b shows the estimated confidence criteria for each group based on the individual fits, and it shows the same pattern that was evident from the fit of the group ROC data. The leftmost criterion (c2) is placed at approximately the same point on the memory strength axis for patients and controls (indicating a similar willingness to express high confidence in new decisions), but the rightmost criterion (c6) is placed in a more conservative location for the patients compared to the controls. As shown in the table, the difference between the placements of the confidence criteria increases from c2 through c6, and the difference between the placements for patients and controls is statistically significant for c5 and c6 ($t(11)=2.81$, $p=.02$ and $t(11)=3.08$, $p=.01$, respectively). The same conservative pattern was also found by calculating the proportion of total responses made by patients and controls for each of the confidence ratings. These results are presented in Table 4 and reveal similar distributions of new responses (confidence ratings 1–3) for both patients and controls. However, in terms of old responses (confidence ratings 4–6), controls' responses were heavily weighted towards a confidence rating of 6, while patients showed a more distributed pattern of responses.

Finally, this conservative pattern can also be observed by examining the probability that a test item is old as function of confidence rating (Table 5). For the "old" responses (confidence ratings 4–6), the corresponding probabilities are high for patients, while they are lower for controls. This suggests that patients tend to set their decision criteria for "old" responses more conservatively than controls. The patients were so conservative in this regard that they only endorsed an item as "sure old" when its memory strength was so high that they were positive a false alarm would not be made. Indeed, neither patient made a single high-confidence false alarm, whereas the controls made many. Furthermore, this reluctance to express high confidence on the part of the patients is specific to endorsing recognized target items (responding "old"), since the corresponding probabilities for "new" responses (confidence ratings 1–3) are similar for both patients and controls.

Table 4
Proportion of total responses as a function of confidence rating.

Confidence rating	Patients ($n=2$)	Controls ($n=13$)	$t(13)$
6	0.14	0.38	2.24*
5	0.13	0.08	0.82
4	0.16	0.05	3.85**
3	0.14	0.09	0.96
2	0.11	0.11	0.02
1	0.33	0.29	0.32

* Denotes significance at the .05 level,
** Denotes significance at the .01 level.

Table 5
Probability that a test item is 'old' as a function of confidence rating.

Confidence rating	Deep encoding		Shallow encoding	
	Patients	Controls	Patients	Controls
6	1.00	0.87	1.00	0.82
5	0.91	0.76	0.82	0.76
4	0.74	0.51	0.73	0.53
3	0.39	0.32	0.52	0.48
2	0.21	0.27	0.41	0.40
1	0.18	0.17	0.32	0.28

4. Discussion

In this study, we investigated subjective memory and recognition memory in two patients with bilateral parietal lobe lesions. We predicted that these patients would show normal memory accuracy coupled with abnormal memory confidence, in line with prior neuropsychological findings (see Table 1). Furthermore, we predicted that patients would exhibit reduced hit rates and false alarm rates at high confidence levels, since an inadequate perception of "oldness" would inhibit patients' willingness to endorse old decisions with high levels of confidence. Therefore, patients and control participants would differ in how they set their decision criteria, with patients exhibiting more conservative responses.

Our findings supported these predictions. Our patients did not demonstrate gross episodic memory deficits and in some instances, they performed numerically better than controls (see Table 3a). We have now tested these patients on a broad assortment of classic memory tests, and in most cases, their accuracy is no different from that of controls (see Berryhill & Olson, 2008; Drowos et al., 2010). Also consistent with prior findings (see Table 1) was the observation that patients' normal memory accuracy was accompanied by abnormal memory confidence. These findings support the claim that the parietal lobe plays a key role in metamemory (subjective) processes, rather than the core memory (objective) processes.

Furthermore, the ROC data revealed that patients were as willing as controls to express high confidence in a "new" decision but were vastly less willing to express high confidence in an "old" decision. Thus, patients do not have a general reluctance to express high confidence; just a reluctance to say that they have seen an item before. Therefore, it seems that patients are able to accumulate and perceive enough novelty information to make a high confidence "new" decision, but they show an impairment that is specific to the perception or accumulation of oldness information.

This reluctance is not limited solely to ratings of 6, but escalates in a graded fashion as the confidence scale increases. Patients give ratings of 1 just as controls do; however, as the confidence scale for items judged as "old" increases, patients exhibit a much more conservative shift in the manner in which they set their decision criteria. This conservative shift can be interpreted as the result of a reduction in subjective memory strength. Each decision criteria (e.g. c5, c6, etc.) reflects the same basic old/new response given by both patients and controls. The difference; therefore, does not concern a preference toward old or new responses. Rather, it concerns where those criteria are set on the memory strength axis. For instance, an item beyond the c6 criterion represents a "sure old" response for both patients and controls; however, the subjective memory strength required for patients to give that response surpasses the strength required for controls (see Tables 2 and 3b).

4.1. Comparison with findings from other populations

The findings from the present study can be contrasted to findings from patients with medial temporal lobe damage. In a review regarding ROC studies in medial temporal lobe amnesics, Yonelinas and Parks (2007) report gross recognition memory deficits in all but one study by Aggleton et al. (2005). The remaining studies (Cipolotti et al., 2006; Fortin, Wright, & Eichenbaum, 2004; Wais, Wixted, Hopkins, & Squire, 2006; Yonelinas, 2002; Yonelinas et al., 1998) consistently found that medial temporal lobe amnesics (and hippocampal lesioned rats; see Fortin et al., 2004) exhibited impaired recognition accuracy and produced more *symmetrical* ROC curves as compared to controls. Across studies, patients with medial temporal lobe damage exhibited reduced hit and false alarm rates at high confidence levels, but their hit rates were lower across all ROC data points. Thus, this phenomenon can be attributed to the gross memory deficits rather than to a selective conservative shift in decision criteria, as seen in our patients.

4.2. Limitations

One limitation that should be noted is the degree of parietal lobe damage that was present in the patients. While much of our predictions and findings focused on the ventral PPC and its role in memory function, the damage to patients' parietal lobes extended beyond that region. Both patients have damage in ventral PPC regions which extends into more dorsal regions of the PPC (see Fig. 1). Several researchers (e.g., Cabeza et al., 2011; Ciaramelli, Grady, & Moscovitch, 2008; Ciaramelli, et al., 2010; Hutchinson, Uncapher, & Wagner, 2009; Jaeger, Konkel, & Dobbins, 2013; Vilberg & Rugg, 2008) have demonstrated that the dorsal and ventral regions of the PPC exhibit differential function roles with respect to memory processes. The dorsal region is thought to support processes guided by top-down attention, whereas the ventral region is thought to support bottom-up attentional processes. Since the patients in our study had damage to both regions, we are unable to disentangle these functions as they relate to memory processes within this sample.

In addition, it is possible that in patient EE555, who suffered from a posterior cerebral artery (PCA) infarct, an embolus lodged in the main trunk of the PCA and affected the hippocampus. We believe that this is unlikely for several reasons. First, inspection of MRIs by a radiologist and neurologist found no evidence of damage to the hippocampus or anywhere else within the medial temporal lobe. Second, this patient does not exhibit classic symptoms of hippocampal damage such as anterograde amnesia. However, the possibility remains that patchy cell loss within the hippocampus proper could cause subtle problems such as those observed in this study.

Finally, it should be noted that the most salient presenting symptom in both patients was simultanagnosia, and this symptom could provide an alternate interpretation of our findings. Simultanagnosia is a disorder in which only a single item or detail of a complex scene can be perceived at any one time. This attentional deficit may extend to both external and internal representations. Therefore, since evidence accumulation relies on surveying our internal representations for contextual and subjective memory information, a restricted attentional window caused by simultanagnosia could presumably impact patients' abilities to accumulate enough evidence to confidently judge an item as old. This could subsequently lead to lower levels of subjective confidence but intact accuracy, consistent with our findings.

4.3. How our findings support – or fail to support – alternative models

In the introduction, we noted that several models have been constructed to explain fMRI findings relating the inferior parietal lobe to episodic memory functions. Our findings bear on some of these models. First, the current data fail to provide support for the episodic buffer account (Vilberg & Rugg, 2008). This hypothesis posits that the inferior parietal lobe works to actively maintain retrieved information that can be later manipulated and accessed by memory retrieval processes. Thus, damage to the parietal lobe should compromise the ability to maintain information in the buffer that is crucial for making recognition decisions. The current data fail to support this claim since memory accuracy was normal in our patients. Even when accompanied by low confidence ratings, patients exhibited no deficits in recognition accuracy.

Second, our findings are consistent with another model, the attention to memory model (Cabeza et al., 2008). This model holds that the lateral parietal lobe plays a role in directing attention towards memory representations such that dorsal regions support memory searches for encoded information guided by top-down attentional processes while ventral regions mediate task-relevant bottom-up attentional processes. As such, it is possible that recognition memory tasks that are more open-ended, those that query one's confidence or whether or not an image was ever seen before, are intrinsically linked to bottom-up internal attention processes that are damaged in our patients. In contrast, recognition memory tasks that are more constrained by the experimental context, requiring one to answer the question for instance, of

whether a learned item was initially spoken by a male or female voice, probably rely more on top-down internal attentional processes that may be largely intact in our patients. Additionally, this account predicts that parietal lesions should lead to a deficit in attention to retrieved information; thus, patients would exhibit lower confidence ratings for associated "old" judgments but would still be able to give accurate responses, which is consistent with the present findings.

Our findings are most consistent with the subjective recollection hypothesis (Simons et al., 2010; Ally et al., 2008). This account suggests that the parietal lobe tracks the degree of subjective experience that occurs during recognition and indexes the perceived vividness of retrieved information. The subjective recollection account would interpret our patients' hesitation to endorse old items with high confidence as the result of a deficiency in their subjective experience of encoding test items. This would lead to an inability to distinguish between vividly retrieved information and vaguely recalled information, which would ultimately lead to a diminished feeling that the event was personally experienced. Such factors would be accompanied by a shift toward more conservative responding due to the lack of subjective recollection, consistent with our data.

A less well-known model, termed the *accumulator model,* can be conceived as a mechanistic explanation for the subjective memory account. Donaldson et al. (2010) proposed that the inferior parietal lobe is responsible for accumulating task-relevant stimulus information necessary for memory decisions. For instance, evidence accumulation could include information regarding temporal context, level of familiarity, or specific details associated with a particular memory. Once the accumulation of evidence reaches a certain threshold (which may be set based on task instructions and/or other motivational factors), a response will be made. According to this particular accumulator model, evidence for old and new information can accumulate simultaneously; therefore, an old/new response will be based on whichever accumulator reaches its respective threshold criterion first. This model is bolstered by findings in nonhuman primates showing that neurons in the lateral intraparietal cortex are sensitive to the accumulation of sensory information, and the firing rates of these neurons increase as evidence necessary to make a decision accrues (Gold & Shadlen, 2007). Similar findings have been reported in human neuroimaging studies. For instance, Guerin and Miller (2011) demonstrated that the parietal cortex tracked the accumulation of retrieved information during a frequency discrimination task. Further, Yu, Johnson, and Rugg (2012) reported that activity in the angular gyrus, located within the inferior parietal lobe, covaried with confidence ratings of source memory judgments, and that the angular gyrus tracked the amount of recollected information. The nature of the information reported in these studies seems to suggest that the evidence being tracked may be related to subjective memory information. Thus, increasing decision confidence may parallel an increase in the accumulation of subjective memory evidence such that the more evidence that is accumulated and maintained by parietal regions, the higher the accompanying levels of confidence will be.

A more nuanced version of the subjective memory account, offered by Wheeler and Buckner (2003) is perhaps the best fit for our data. In this study, Wheeler and Buckner (2003) found that activation in the left inferior parietal lobe increased during trials where participants judged items as old. This pattern of increased activation was not only present for old items correctly judged as old, but also for new items that were mistakenly judged as old. Therefore, this study, along with several other fMRI studies have shown that when an item is perceived as old, regardless of whether the response is correct or incorrect, it is accompanied by heightened activity in the left inferior parietal lobe (Wheeler & Buckner, 2003; Donaldson et al., 2001; Habib and Lepage, 1999; Henson et al., 1999; Konishi et al., 2000; McDermott et al., 2000). These findings suggest that inferior parietal lobe activity reflects our internal belief that an item has been previously experienced, rather than the actual retrieval of externally validated information about the item.

These findings predict that damage to this region should result in a deficit in subjectively perceiving oldness with little to no effect on objective measures of memory retrieval, which is exactly the pattern that we observed. Thus, while a test item that generates a reasonably strong memory signal seems old to a normal control, it does not seem old to our patients. The signal has to be much stronger before the patient perceives the item to be old. However, a test item that generates a weak memory signal (or, perhaps, a novelty signal) seems new to a normal control and also seems new to our patients. It is possible that the mechanism underlying this impairment is a malfunctioning accumulator. Thus, damage to the inferior parietal lobe may lead to a problem in metamemory that is selective to the "perception of oldness".

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