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## Research report

# Confabulation: Damage to a specific inferior medial prefrontal system

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### ABSTRACT

Confabulation, the pathological production of false memories, occurs following a variety of aetiologies involving the frontal lobes, and is frequently held to be underpinned by combined memory and executive deficits. However, the critical frontal regions and specific cognitive deficits involved are unclear. Studies in amnesic patients have associated confabulation with damage to the orbital and ventromedial prefrontal cortices. However, neuroimaging studies have associated memory-control processes which are assumed to underlie confabulation with the right lateral prefrontal cortex. We used a confabulation battery to investigate the occurrence and localisation of confabulation in an unselected series of 38 patients with focal frontal lesions. Twelve patients with posterior lesions and 50 healthy controls were included for comparison. Significantly higher levels of confabulation were found in the frontal group, confirming previous reports. More detailed grouping according to lesion location within the frontal lobe revealed that patients with orbital, medial and left lateral damage confabulated in response to questions probing personal episodic memory (PEM). Patients with orbital, medial and right lateral damage confabulated in response to questions probing orientation to time (OT). Performance-led analysis revealed that all patients who produced a total number of confabulations outside the normal range had a lesion affecting either the orbital region or inferior portion of the anterior cingulate. These data provide striking evidence that the critical deficit for confabulation has its anatomical location in the inferior medial frontal lobe. Performance on tests of memory and executive functioning showed considerable variability. Although a degree of memory impairment does seem necessary, performance on traditional executive tests is less helpful in explaining confabulation.

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## 1. Introduction

Confabulation following neurological disease has been defined as “a falsification of memory occurring in clear

consciousness in association with an organically derived amnesia” (Berlyne, 1972). It involves the production of false accounts which the patient in most cases believes to be true, and involves no intent to deceive the listener. These false

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accounts may either be “provoked”, in response to a memory test or questioning, or “spontaneous”, in which there is an unprovoked outpouring of erroneous memories (Kopelman, 1987), and the content may range from subtle alterations of true events to bizarre and implausible stories. The study of amnesic conditions has been very informative in the study of memory. However, despite being a dramatic and surprising clinical phenomenon, confabulation has so far been much less informative about the organisation of memory processes, with neither the anatomical localisation nor the critical cognitive deficit being fully understood.

Confabulation is frequently reported in association with aetiologies involving frontal lobe damage, including rupture and repair of anterior communicating artery aneurysms (Alexander and Freedman, 1984; Baddeley and Wilson, 1988; Burgess and McNeil, 1999; Dab et al., 1999; Damasio et al., 1985; DeLuca and Diamond, 1995; Delbecq-Derouesne et al., 1990; Fischer et al., 1995; Kapur and Coughlan, 1980; Kopelman et al., 1995; Moscovitch, 1989; Stuss et al., 1978; Vilkki, 1985), posterior communicating artery aneurysms (Dalla Barba et al., 1997; Mercer et al., 1977), frontal tumours (Fotopoulou et al., 2004), head injury (Baddeley and Wilson, 1988; Berlyne, 1972; Box et al., 1999; Damasio et al., 1985; Demery et al., 2001; Moscovitch and Melo, 1997), frontotemporal dementia (Nedjam et al., 2000; Moscovitch and Melo, 1997), Korsakoff's syndrome (Berlyne, 1972; Benson et al., 1996; Dalla Barba et al., 1990; Kopelman, 1987; Kopelman et al., 1997; Korsakoff, 1955; Mercer et al., 1977; Talland, 1965), Alzheimer's disease (Dalla Barba et al., 1999; Kern et al., 1992; Kopelman, 1987; Nedjam et al., 2000; Tallberg and Almkvist, 2001), and herpes simplex encephalitis (Del Grosso Destreri et al., 2002; Moscovitch and Melo, 1997). Nearly all the evidence regarding the critical lesion site has come from single cases, or small series of confabulators. Gilboa and Moscovitch (2002) reviewed 39 of these studies, and reported that 81% of confabulators had damage to the prefrontal cortex. They found no evidence of lateralisation, with confabulation occurring following both left and right unilateral and bilateral frontal lesions, but reported that the most common sites of damage were the orbitofrontal and ventromedial aspects of the frontal lobe. Similarly, Schnider et al. (1996a), Schnider and Ptak (1999) and Schnider (2003) have highlighted the anterior limbic system (and particularly the orbitofrontal cortex) as the critical lesion site in spontaneous confabulation.

However, the findings from neuroimaging studies paint a different picture. Memory-control processes involved in strategic or effortful retrieval are often considered to be involved in confabulation. In particular cue specification and strategic monitoring processes are often thought to be disrupted (Burgess and Shallice, 1996; Moscovitch and Melo, 1997; Schacter et al., 1998). Evidence from neuroimaging has localised these strategic retrieval processes to regions of the right lateral prefrontal cortex (Rugg and Wilding, 2000), and in the case of monitoring, particularly to the right dorsolateral prefrontal cortex (Cabeza et al., 2003; Fletcher et al., 1998; Henson et al., 1999; see Shallice, 2001, 2006 for a review). This is in sharp contrast to patient studies reporting aetiologies involving the basal forebrain, orbitofrontal and ventromedial prefrontal cortices.

The critical cognitive deficit underlying confabulation has also remained unclear. In addition to memory deficits,

confabulation is often associated with poor performance on executive tests (Baddeley and Wilson, 1988; Cunningham et al., 1997; Kapur and Coughlan, 1980; Kopelman, 1987; Mattioli et al., 1999; Stuss and Benson, 1986; Stuss et al., 1978). Several authors have therefore proposed it to result from an amnesia overlaid with a frontal dysexecutive impairment (Baddeley and Wilson, 1988; DeLuca and Diamond, 1995; Kern et al., 1992; Kopelman, 1987; Kapur and Coughlan, 1980; Mercer et al., 1977; Shapiro et al., 1981; Stuss et al., 1978), with the degree of confabulation being determined by the degree of executive dysfunction. However, recent reports indicate that the critical cognitive deficit associated with confabulation may be more specific than general executive dysfunction. Fischer et al. (1995) reported that only those executive tests reflecting self-monitoring (set shifting and perseveration) were associated with confabulation. Cunningham et al. (1997) reported that confabulation was related to tests tapping sustained attention, set shifting and mental tracking, but not concept formation, problem-solving or verbal fluency. And Nys et al. (2004) reported that disappearance of spontaneous confabulation in a confabulating patient was associated with improvement in mental flexibility, but not in other executive functions.

Confabulation has mainly been investigated in single case studies and small series of pre-selected confabulating patients. To our knowledge there have been only two group studies examining lesion location in confabulating compared to non-confabulating patients, and these were in amnesic patients (Moscovitch and Melo, 1997; Schnider and Ptak, 1999). However, despite its strong association with frontal dysfunction, the incidence of confabulation has never been investigated in a large unselected sample of frontal patients.

The present study employed a confabulation battery to investigate the anatomical localisation of confabulation, and associated memory and executive functioning in an unselected frontal series. We set out to answer two questions: (1) is confabulation in frontal patients associated with orbitofrontal and ventromedial damage (as predicted by lesion studies in amnesic patients), or with right lateral prefrontal damage (as predicted by neuroimaging studies of memory-control processes)? and (2) can confabulation in this population be explained in terms of deficits in memory and executive functioning, and if so, which particular executive impairments are associated with confabulation?

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## 2. Methods

### 2.1. Participants

Fifty-seven patients with focal frontal lesions were recruited from the National Hospital for Neurology and Neurosurgery and tested in the Neuropsychology Department between June 2001 and April 2003. Inclusion and exclusion criteria were (1) the presence of a focal lesion confined to the frontal lobes, (2) English as a first language, (3) absence of childhood onset epilepsy (late onset seizures arising from the lesion were allowed), (4) absence of severe aphasia, and (5) absence of other significant neurological and psychiatric disorders. All patients admitted to the hospital during the recruitment

period who met these criteria and consented to take part in the study were included. Nineteen patients were subsequently excluded at the scan analysis stage due to insufficient detail in the scan (for localisation purposes), lack of a post-operative magnetic resonance imaging (MRI) or computerised tomography (CT) scan, or significant posterior involvement. Aetiologies of the remaining 38 patients in the frontal group were as follows: anterior communicating artery aneurysm ( $n = 12$ ), meningioma ( $n = 6$ ), glioma ( $n = 11$ ), metastasis ( $n = 2$ ), haematoma ( $n = 4$ ), abscess ( $n = 1$ ), arteriovenous malformation (AVM) ( $n = 1$ ), and lymphoma ( $n = 1$ ).

Sixteen patients with posterior lesions were recruited in the same way as the frontal group, but with the inclusion criteria of a posterior lesion which did not encroach into the frontal lobe. Four patients were subsequently excluded at the scan analysis stage due to significant frontal involvement or insufficient detail in the scan. Of the remaining 12 posterior patients, seven had lesions affecting the temporal lobe, two parietal, two temporo-parietal and one parieto-occipital. Aetiologies were glioma ( $n = 6$ ), meningioma ( $n = 4$ ), unspecified lesion ( $n = 1$ ), and temporal lobectomy ( $n = 1$ ). The performance of patients was compared to that of 50 healthy controls. All participants gave informed consent before being tested, and the study was approved by the National Hospital for Neurology and Neurosurgery and the Institute of Neurology Joint Research Ethics Committee.

## 2.2. Lesion analysis

Analysis of lesion site amongst the frontal group was conducted following an approach based on that of [Stuss et al. \(2002\)](#). A radiologist (TY) blind to the nature of the patient's behavioural deficit examined MRI (or CT where MRI was unavailable) scans and coded each for the presence or absence of lesion in 12 prefrontal areas in each hemisphere (24 in total). These areas were orbital, sub-genu, anterior cingulate (anterior and posterior portions), medial surface of the superior frontal gyrus (anterior and posterior portions), lateral superior frontal gyrus (anterior and posterior portions), lateral middle frontal gyrus (anterior and posterior portions), and lateral inferior frontal gyrus (anterior and posterior portions). On the medial surface the anterior/posterior border was taken as the point midway between the frontal pole and the ramus marginalis. On the lateral surface the anterior/posterior border was taken as the point midway between the frontal pole and the precentral sulcus. An area was only coded as damaged if at least 25% of that area was affected (areas of oedema were coded in the initial analysis but did not affect final groupings and were not common enough across patients to be included in the final analysis).

These 24 regions were then collapsed into four groups for group comparisons. Patients were assigned to the following groups according to the region of greatest damage: orbital ( $n = 11$ ), in which greatest damage was to the orbital surface of one or both lobes; medial ( $n = 8$ ), in which greatest damage was to the sub-genu, anterior cingulate or medial surface of the superior frontal gyrus of one or both lobes; left lateral ( $n = 8$ ), in which greatest damage was to the left lateral superior, middle or inferior frontal gyri; and right lateral ( $n = 7$ ), in which greatest damage was to the right lateral superior,

middle or inferior frontal gyri. Four patients were excluded from these groupings as they had lesions which were too extensive to be accurately assigned only to one grouping, and were included only in analysis of the frontal group as a whole. [Fig. 1](#) shows the proportion of patients in each group with damage to each of the 24 coded areas.

[Table 1](#) shows demographic data for the control, posterior and frontal groups and the four frontal subgroups. No groups differed significantly in terms of age, years of education or time since surgery. The large variance in time since surgery was introduced by five re-admitted patients (one in the posterior group, and four in the frontal group) who were tested between 1 and 24 years after initial surgery. However, the majority of patients were tested during the acute phase (median = 8 days post-surgery).

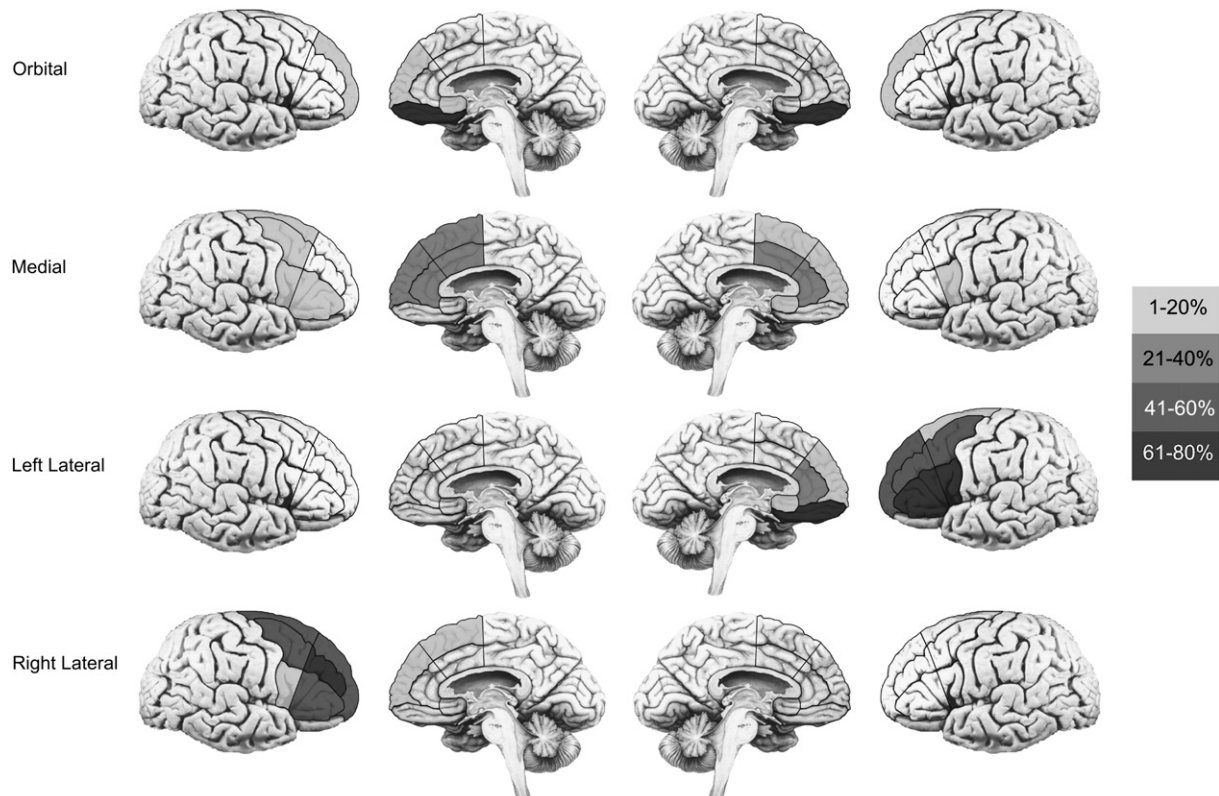
## 2.3. Confabulation battery

A modified version of the confabulation battery developed by [Dalla Barba et al. \(1990\)](#) was developed by selecting five questions from each of the following categories: general semantic memory (GSM) (for example, "What happened to President Kennedy?"), personal semantic memory (PSM) (for example, "What is your address?"), personal episodic memory (PEM) (for example, "What did you do yesterday?"), orientation in time (OT) (for example, "What month is it?"), orientation in place (OP) (for example, "What city are we in?") and questions to which participants were expected to respond "don't know" (DK) (for example, "Who is the current world fencing champion?"). Participants were also asked to tell the story of Little Red Riding Hood. Questions were put to participants in a random order, and responses were scored as "correct", "don't know" or "confabulation". For PSM and PEM questions, all answers were checked with a relative of the patient. For all other categories there were clear correct answers and these were scored by the examiner (MT). Answers were only classified as "confabulation" if the information given was clearly incorrect. If participants gave a vague answer they were asked for clarification, so in no case was there any uncertainty of how to classify responses. A "don't know" response for a follow-up answer was allowed. Orientation questions were included given the high frequency of temporal and spatial distortions in confabulation, and given the links between confabulation and orientation established in the literature ([Schnider et al., 1996b](#)).

## 2.4. Memory and executive batteries

Measures of recognition and recall of both verbal and visual materials were obtained using the Recognition Memory Test ([Warrington, 1984](#)), the Story Recall component of the Adult Memory and Information Processing Battery ([Coughlan and Hollows, 1985](#)), the Rey-Osterrieth Complex Figure Test ([Osterrieth, 1944](#)), and the Doors and People Test ([Baddeley et al., 1994](#)).

The following measures of executive functioning were also employed: resistance to interference was assessed using the Stroop Neuropsychological Screening Test colour-word score ([Trenary et al., 1989](#)); set shifting was assessed using the Trail Making Test ([Reitan and Wolfson, 1985](#)); sustained attention was assessed using the Elevator subtest of the Test of Everyday Attention ([Robertson et al., 1994](#)); verbal fluency was



**Fig. 1 – Lesion location by frontal subgroup. Shaded areas represent the proportion of patients within each group who have lesions affecting at least 25% of the depicted region.**

assessed using Controlled Oral Word Association (COWA) with the letters F, A and S (1 min each). A measure of concrete/abstract thinking was also obtained using a Proverb Interpretation test, in which participants were asked to explain the meaning of eight common proverbs. Responses were scored using a three point system, with two points awarded for a full, appropriate and abstract interpretation, one point for a partially accurate or concrete interpretation, and zero for an inaccurate interpretation.

## 2.5. Statistical analysis

### 2.5.1. Level 1 analysis

At the first level of analysis the performance of frontal, posterior and control participants was compared in order to establish whether there was an effect of a frontal lesion. Significant

results were followed by pairwise comparisons to look for differences between the groups. Adjustment for multiple comparisons was made using a Bonferroni correction for three comparisons. Uncorrected significance levels are reported, but results are only treated as significant if they achieve  $p < .017$ .

### 2.5.2. Level 2 analysis

In the event of a significant frontal impairment being found at level 1, further analysis was undertaken to explore the specificity of the frontal effect. Performance of the orbital, medial, left lateral and right lateral subgroups was compared to the control group (posterior patients were not included in this analysis). As the aim of this analysis was to provide more anatomical specificity for a lesion effect already established at the first level, Bonferroni corrections were not applied to pairwise comparisons.

**Table 1 – Demographic data**

	Sex	Age	Years of education	Time since surgery (days)
Control (n = 50)	25M, 25F	48.62 ± 15.96	13.06 ± 3.05	N/A
Posterior (n = 12)	6M, 6F	46.67 ± 14.22	12.33 ± 3.11	49.08 ± 130.87
Frontal (n = 38)	22M, 16F	47.47 ± 13.81	12.79 ± 3.36	361.67 ± 1541.68
Orbital (n = 11)	10M, 1F	45.73 ± 16.75	13.45 ± 3.64	239.50 ± 570.03
Medial (n = 8)	5M, 3F	41.38 ± 11.04	12.88 ± 3.36	6.67 ± 5.28
L lateral (n = 8)	3M, 5F	49.88 ± 13.97	12.38 ± 2.77	19.17 ± 30.62
R lateral (n = 7)	2M, 5F	51.43 ± 12.63	12.43 ± 3.74	78.43 ± 169.14

Data are presented in the form: mean ± standard deviation.



In analysis of the confabulation battery data non-parametric statistics (Kruskal–Wallis) were used as the control group was frequently at floor for errors. In the absence of an accepted non-parametric post-hoc testing procedure, significant Kruskal–Wallis tests were followed by pairwise Mann–Whitney *U* tests to look for differences between the groups. This method has exactly the same logic as “least significant difference” tests if only applied when Kruskal–Wallis gives a significant result.

### 3. Results

#### 3.1. Baseline neuropsychological test performance

Table 2 shows performance on baseline neuropsychological tests. Level 1 analysis comparing frontal, posterior and control groups revealed a significant effect of group on Raven’s Advanced Progressive Matrices (APM) performance (one-way Analysis of Variance (ANOVA) effect of group:  $F_{(2, 97)} = 3.56$ ,  $p = .032$ ) and on National Adult Reading Test (NART) estimated full scale IQ (Kruskal–Wallis effect of group:  $\chi^2 = 7.90$ ,  $p = .019$ ). Pairwise comparisons revealed a trend towards lower Raven’s performance in the frontal group (compared to controls) and towards lower NART FSIQ scores in the frontal and posterior groups (compared to controls). However, none of these comparisons reached the corrected significance level of  $p < .017$ . The frontal and posterior groups did not differ significantly (nor show a trend) on any measure. Level 2 analysis comparing the orbital, medial, left lateral and right lateral frontal subgroups to the control group showed that the medial group had slightly depressed general intelligence as measured by Raven’s APM performance compared to controls (one-way ANOVA effect of subgroup:  $F_{(4,77)} = 2.58$ ,  $p = .044$ ; pairwise comparisons: medial < control  $p = .007$ ). However, there were no differences in NART estimated full scale IQ, and no naming or visual perception impairments in any group.

#### 3.2. Confabulation battery: grouping by lesion site

Analysis of the confabulation battery data was first conducted by comparing the performance of patients grouped by lesion site. These analyses were first conducted on the total number of confabulations produced on the battery overall, and then on critical subcomponents.

#### 3.2.1. Analysis of overall confabulation battery

On the first level of analysis a frontal localisation for confabulation was obtained (Fig. 2a) the frontal group produced a significantly higher number of confabulations across the battery than the control group (Kruskal–Wallis effect of group:  $\chi^2 = 10.92$ ,  $p = .004$ ; pairwise Mann–Whitney *U* comparisons: frontal > control  $p = .003$ ). There was no evidence of excess confabulations in the posterior group, who did not differ significantly from the controls.

The second level of analysis (Fig. 2b) revealed that the orbital and medial frontal subgroups were driving this effect, being the only groups to differ significantly from controls (Kruskal–Wallis effect of subgroup:  $\chi^2 = 12.94$ ,  $p = .012$ ; pairwise Mann–Whitney *U* comparisons: orbital > control  $p = .001$ ; medial > control  $p = .049$ ). No other significant subgroup differences were found.

Having established the presence of confabulations in the frontal group, analyses were conducted to investigate which types of questions were eliciting them. Fig. 3 shows the distribution of confabulations across question type by the frontal, posterior and control groups. No significant group effects were found in confabulations produced in response to questions probing GSM, PSM, OP, or DK questions. However, the frontal group produced significantly higher levels of confabulation than controls in response to questions probing PEM (Kruskal–Wallis effect of group:  $\chi^2 = 17.20$ ,  $p < .001$ ; pairwise Mann–Whitney *U* comparisons: frontal > control  $p < .001$ ) and OT (Kruskal–Wallis effect of group:  $\chi^2 = 7.89$ ,  $p = .019$ ; pairwise Mann–Whitney *U* comparisons: frontal > control  $p = .005$ ). Therefore, the anatomical localisation of confabulation in PEM and OT was examined in more detail.

#### 3.2.2. Analysis of two critical subcomponents

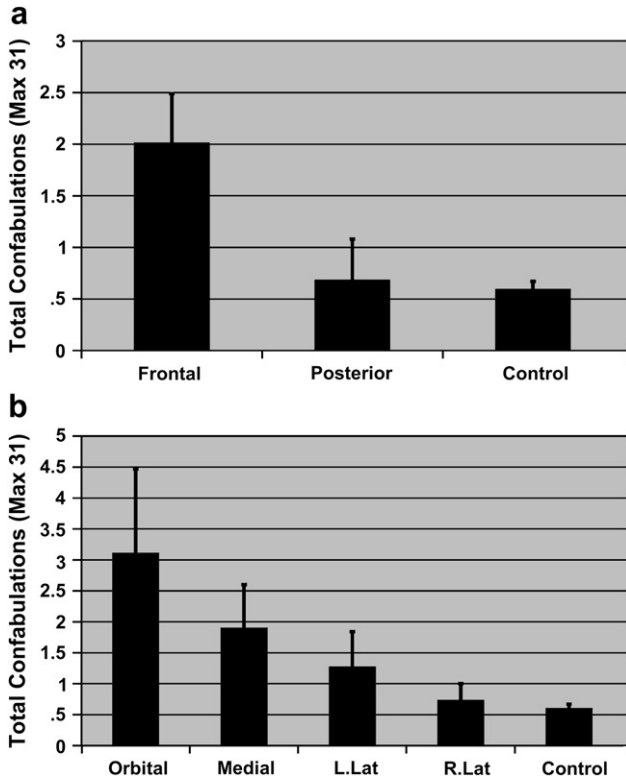
3.2.2.1. PERSONAL EPISODIC MEMORY. More detailed examination using level 2 analysis revealed that the orbital, medial and left lateral groups produced significantly more confabulations in this category than controls (Kruskal–Wallis effect of subgroup:  $\chi^2 = 25.75$ ,  $p < .001$ ; pairwise Mann–Whitney *U* comparisons: orbital > control  $p < .001$ ; medial > control  $p < .001$ ; left lateral > control  $p < .001$ ). No significant differences were found for “don’t know” responses (Fig. 4).

3.2.2.2. ORIENTATION IN TIME. Level 2 analysis revealed that in response to questions probing orientation in time, the orbital,

**Table 2 – Baseline neuropsychological testing**

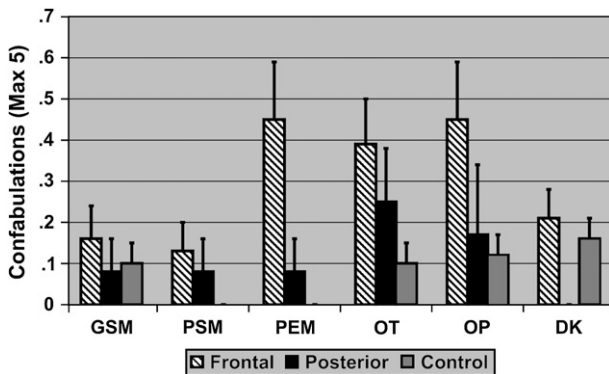
	Raven’s APM	NART FSIQ	Graded Naming Test	Incomplete Letters
Control ( $n = 50$ )	11.18 ± 2.85	110.64 ± 9.78	11.04 ± 3.49	19.26 ± .88
Posterior ( $n = 12$ )	9.33 ± 3.73	100.92 ± 12.00	10.50 ± 2.75	19.50 ± .67
Frontal ( $n = 38$ )	9.58 ± 3.27	103.97 ± 15.05	9.38 ± 3.74	19.11 ± 1.52
Orbital ( $n = 11$ )	11.09 ± 2.30	105.73 ± 10.15	9.18 ± 3.89	19.64 ± .50
Medial ( $n = 8$ )	8.13 ± 3.68	96.50 ± 16.29	8.71 ± 3.20	18.63 ± 2.00
L lateral ( $n = 8$ )	9.25 ± 1.58	108.38 ± 18.72	11.25 ± 2.96	19.25 ± 1.04
R lateral ( $n = 7$ )	11.14 ± 2.19	110.43 ± 13.09	10.29 ± 3.50	19.29 ± .76

Performance of control, posterior and frontal groups, and the four frontal subgroups on the following general baseline tests: Raven’s APM age-scaled score (Raven et al., 1988), NART full scale IQ (Nelson, 1982), Graded Naming Test age-scaled score (McKenna and Warrington, 1983) and Incomplete Letters subtest of the Visual Object and Space Perception Battery (Warrington and James, 1991). Data are presented in the form: mean ± standard deviation.



**Fig. 2 – Total number of confabulations produced across the battery by (a) frontal, posterior and control groups (level 1 analysis) and (b) orbital, medial, left lateral, right lateral and control groups (level 2 analysis). Bars represent mean scores and error bars represent standard error of the mean.**

medial and right lateral groups produced significantly more confabulations in this category than controls (Kruskal-Wallis effect of subgroup:  $\chi^2 = 10.88, p = .028$ ; pairwise Mann-Whitney U comparisons: orbital > control  $p = .012$ ; medial > control



**Fig. 3 – Distribution of number of confabulations by question type. GSM = general semantic memory; PSM = personal semantic memory; PEM = personal episodic memory; OT = orientation to time; OP = orientation to place; and DK = “Don’t Know” questions. Bars represent mean scores and error bars represent standard error of the mean. Responses to the retelling of Little Red Riding Hood are omitted as this was a single item.**

$p = .018$ ; right lateral > control  $p = .011$ ). No significant differences were found for “don’t know” responses (Fig. 5).

**3.3. Confabulation battery: grouping by performance**

A performance-led analysis was also performed, in which patients were grouped not by lesion site, but by their total number of confabulations across all items of the battery. Eight patients produced a total number of confabulations in excess of two standard deviations outside the normal control range (i.e., three or more confabulatory responses across the battery), and were classified as “high-confabulators”. All patients were from the frontal group, and no posterior patients fell into this range. The remaining frontal patients were classified as “low-confabulators”.

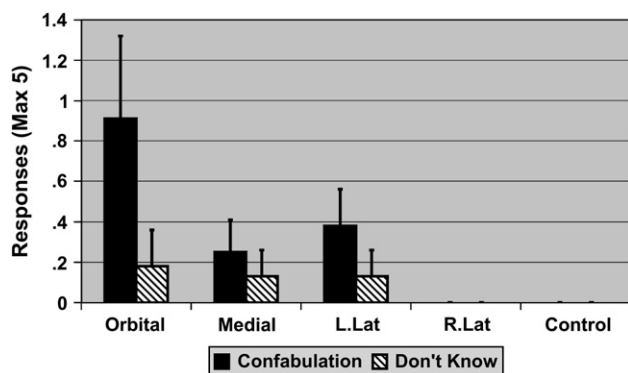
Confabulations produced in response to questioning of this type are strictly speaking provoked confabulations rather than spontaneous confabulations (Kopelman, 1987). However, at least three of these patients were observed to produce frequent, florid and unprovoked confabulations both in and out of testing (patients 143, 131 and 150). Four further patients (108, 106, 136 and 145) produced responses during the confabulation battery that were fluent and unusual enough to indicate that they may also have been spontaneously confabulating. However, an accurate assessment of the confabulation of these patients outside testing situations was not possible due to rapid transfer from the hospital.

Confabulatory responses on the confabulation battery were classified into three types:

- (1) Mundane wrong or guessed responses (W), such as getting the floor of the hospital wrong, getting the date wrong by less than three days, giving the name of an incorrect but local tube station when asked which was the nearest, or mistaking Little Red Riding Hood for a different fairy tale (e.g., Goldilocks).
- (2) Responses that indicated confusion in time or place (C), such as reporting that they were in a different city, getting the date wrong by more than 3 days, reporting that John Major was the current Prime Minister,<sup>1</sup> or reporting a correct event but in the wrong time or place.
- (3) Invented or bizarre responses (I), for example, believing that he had spent the previous Christmas in an underground bunker (patient 143), believing that he worked at the hospital (patient 108), providing an invented Little Red Riding Hood story in which she is raped (patient 150), or reporting that as part of his treatment he had had probes attached to his head that produced a graph when he responded to questioning (this bore no relation to any treatment or testing situation that he had been involved in; patient 131).

Details of confabulation type, aetiology, time since surgery and lesion location for the eight high-confabulators are shown in Table 3. It is notable that severity affects the rate of C

<sup>1</sup> John Major was British Prime Minister from 1990 to 1997. Testing was conducted between 2001 and 2003.



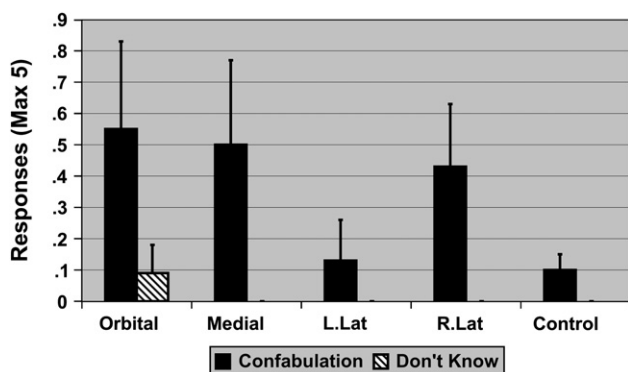
**Fig. 4 – Number of confabulations and “don’t know” responses produced to questions probing PEM by orbital, medial, left lateral, right lateral and control groups (level 2 analysis). Bars represent mean scores and error bars represent standard error of the mean.**

(confusion in time and place) and I (invented or bizarre) confabulations, but not W (mundane wrong or guessed) responses.

The most striking result to emerge from this performance-based analysis is the consistency of the lesion localisations for each of the high-confabulators. Every patient who produced an abnormal number of confabulations, without exception, had a lesion affecting the inferior medial frontal lobe. Six of the eight had a lesion affecting the orbital region, and the remaining two had a lesion affecting the inferior parts of the anterior cingulate cortex (ACC) (one bilateral and one right sided). Comparison with the remainder of the group revealed that 8/8 (100%) of the high-confabulating patients had inferior medial damage (orbital, sub-genual or ACC regions), in comparison to 16/29 (55%) of the low-confabulating frontal patients (Fisher’s exact test  $p = .03$ ).

### 3.4. Confabulation: a combined memory and executive function deficit?

Confabulation has frequently been characterised as an amnesia overlaid with a dysexecutive syndrome (Baddeley and



**Fig. 5 – Number of confabulations and “don’t know” responses produced in response to questions probing OT by orbital, medial, left lateral, right lateral and control groups (level 2 analysis). Bars represent mean scores and error bars represent standard error of the mean.**

Wilson, 1988; DeLuca and Diamond, 1995; Kern et al., 1992; Kopelman, 1987; Kapur and Coughlan, 1980; Mercer et al., 1977; Shapiro et al., 1981; Stuss et al., 1978). If so, confabulation in these eight patients should be attributable to a combination of impairments in memory and executive functioning. In order to explore this hypothesis, a “memory score” was calculated by taking the mean of the z-scores obtained from six memory measures: the “words” and “faces” scores from the Recognition Memory Test, Immediate and Delayed Recall scores from the Story Recall component of the Adult Memory and Information Processing Battery, 40 min delayed recall of the Rey–Osterrieth Complex Figure Test, and overall age-scaled score from the Doors and People Test. Similarly, an “executive score” was calculated by taking the mean of the z-scores achieved on five executive measures: Stroop colour-word (interference) score, Trail Making Test: time to complete Part B, COWA score (total number of words produced), Proverb Interpretation score, and Elevator subtest score from the Test of Everyday Attention. All z-scores were calculated using the mean and standard deviation of the control group performance. These scores can be seen in Table 4.

All groups had impaired memory scores compared to controls (Kruskal–Wallis effect of group:  $\chi = 27.01$ ,  $p < .001$ ; pairwise Mann–Whitney  $U$  comparisons: high-confabulator frontal  $<$  control  $p < .001$ ; low-confabulator frontal  $<$  control  $p = .005$ ; posterior  $<$  control  $p = .001$ ).<sup>2</sup> All groups also had impaired executive scores compared to controls (Kruskal–Wallis; effect of group  $\chi = 30.54$ ,  $p < .001$ ; pairwise Mann–Whitney  $U$  comparisons; high-confabulator frontal  $<$  control  $p < .001$ ; low-confabulator frontal  $<$  control  $p < .001$ ; posterior  $<$  control  $p = .009$ ). Comparison of the high-confabulator frontal group to the low-confabulator frontal group showed that although the high-confabulator frontal group had significantly lower memory scores than the low-confabulator frontal group ( $p = .008$ ), the difference in their executive scores failed to meet significance ( $p = .06$ ).

Examination of the individual memory and executive scores within the high-confabulator frontal group reveals great variability in performance. There are two patients whose performance on both the memory and executive score falls more than two standard deviations outside the range of controls (patients 136 and 150). However, there are also two patients who are within the range of controls on both measures (patients 108 and 110). It appears that individual confabulating patients may show very different patterns in terms of memory and executive functioning.

Given that these scores are determined by performance on several different tests, which may be measuring different functions (especially in the case of “executive” tests) we also explored whether individual tests differed in their ability to discriminate between the high-confabulator and low-confabulator frontal groups. Table 5 shown mean z-scores for the two groups on each measure. Independent  $t$ -tests revealed that whilst all memory measures yielded a significant difference between the two groups, only two executive

<sup>2</sup> Post-hoc analyses in these comparisons are Bonferroni corrected for four comparisons. Non-corrected significance levels are reported, but results are only treated as significant if they achieve  $p < .012$ .

**Table 3 – Lesion localisation of eight high-confabulator frontal patients**

Patient ID	Total confabulations	Aetiology	Time since surgery (days)	Level 2 subgroup	Orb	Medial						Lateral				
						SG	ACC		SFG		SFG		MFG		IFG	
							Ant.	Pos.	Ant.	Pos.	Ant.	Pos.	Ant.	Pos.	Ant.	Pos.
143	16 (3W, 6C, 7I)	ACoAA	16	Orbital	BL	0	0	0	0	0	0	0	0	0	0	0
131	7 (2W, 2C, 3I)	ACoAA	8755	N/A	BL	BL	BL	0	L	0	L	0	0	0	0	0
108	5 (2W, 1C, 2I)	ACoAA	8	Orbital	L	0	0	0	0	0	0	0	0	0	0	0
106	5 (2W, 2C, 1I)	Meningioma	6	L lateral	L	0	0	0	0	0	0	0	0	0	L	L
136	5 (2W, 2C, 1I)	Lymphoma	5	N/A	L	0	0	0	0	0	0	0	0	0	L	L
145	5 (3W, 2C)	ACoAA	17	Medial	0	0	R	0	0	0	0	0	0	0	0	0
150	5 (1W, 2C, 2I)	ACoAA	7	Medial	0	0	BL	BL	R	0	0	0	0	0	0	0
110	3 (3W)	AVM	8	Orbital	R	0	0	0	0	0	0	0	0	0	0	0

Orb = orbital; SG = sub-genu; ACC = anterior cingulate cortex; SFG = superior frontal gyrus; MFG = middle frontal gyrus; IFG = inferior frontal gyrus; ant. = anterior; pos. = posterior; 0 = no damage; L = left lateralised damage; R = right lateralised damage; and BL = bilateral damage.

measures were able to discriminate the high-confabulator from the low-confabulator group. These were the Stroop colour-word score, and COWA score. Examination of the individual z-scores for the high-confabulator group (Table 6) again reveals considerable variability on all measures. On the critical executive measures, only 50% of the group performed outside the normal range on the COWA test. However, impairments on the Stroop test were more consistent, with 5/7 patients performing more than two standard deviations below the control mean.

#### 4. Discussion

In previous work, confabulation has frequently been associated with frontal damage and deficits in executive functioning. However, confabulation has never previously been investigated systematically in the context of frontal patients. Using a quantitative battery, this study confirmed the presence of confabulation in an unselected group of patients with focal frontal lesions. By contrast no patient with a posterior lesion scored outside the normal range in the confabulation battery. More detailed anatomical localisation procedures revealed strikingly consistent orbital and medial frontal effects, with performance-led analysis confirming an inferior medial prefrontal localisation for confabulation: all

eight patients who produced a total number of confabulations outside the normal range had a lesion affecting the inferior medial frontal region (either orbital or anterior cingulate). Performance on batteries of memory and executive functioning revealed that all six measures of memory functioning were able to distinguish between high and low-confabulator frontal groups, and two measures of executive functioning also differed significantly between the groups. However, there was considerable individual variability, with some confabulating patients performing in the normal range on both. We will discuss first the findings related to the anatomical localisation of confabulation, and second the contribution of executive and memory processes to confabulation.

In terms of lesion location, the most striking feature of these results is the consistent involvement of the inferior medial prefrontal cortex in the production of confabulations. This is consistent with group studies carried out in patients selected on the basis of their amnesia (Moscovitch and Melo, 1997; Schnider and Ptak, 1999), and fits with previous case reports (Baddeley and Wilson, 1988; Box et al., 1999; Dab et al., 1999; Damasio et al., 1985; Delbecq-Derouesne et al., 1990; Demery et al., 2001; Fotopoulou et al., 2004; Kapur and Coughlan, 1980; Kopelman et al., 1997; Mattioli et al., 1999; Mercer et al., 1977; Shapiro et al., 1981; Stuss et al., 1978; Vilkki, 1985; see Gilboa and Moscovitch, 2002 for a review). It is also

**Table 4 – Mean memory z-scores and executive z-scores for control, posterior and high-confabulator and low-confabulator frontal groups (mean ± standard deviation) and individual scores for the eight high-confabulator frontal patients**

	Total confabulations	Memory score	Executive score
Control	.58 ± .64	.00 ± .69	.00 ± .58
Posterior	.67 ± 1.44	-1.73 ± 1.87	-.77 ± .99
Low-confabulator frontal	.80 ± .71	-.75 ± 1.26	-1.24 ± 1.56
High-confabulator frontal	6.50 ± 4.00	-2.76 ± 2.05	-2.38 ± 1.91
<i>Individual scores</i>			
143	16	-4.74	-1.74
131	7	-3.26	-1.01
108	5	-.90	-.09
106	5	-1.79	-3.46
136	5	-5.64	-6.38
145	5	-.65	-2.30
150	5	-4.56	-2.62
110	3	-.54	-1.43



**Table 5 – Mean z-scores on individual memory and executive measures for high-confabulator and low-confabulator frontal groups (mean ± standard deviation)**

	High-confabulator frontal	Low-confabulator frontal	p value
<i>Memory measures</i>			
RMT words	−5.65 ± 5.69	−2.23 ± 3.55	p = .041
RMT faces	−2.08 ± 2.80	−.21 ± 1.04	p = .004
Story recall: immediate	−2.05 ± 1.43	−.44 ± 1.41	p = .007
Story recall: delayed	−2.09 ± 1.36	−.44 ± 1.50	p = .008
Rey figure delayed recall	−1.72 ± .89	−.19 ± 1.02	p = .001
Doors and People	−2.04 ± 1.12	−.81 ± 1.06	p = .024
<i>Executive measures</i>			
Stroop colour-word score	−3.29 ± 2.29	−.88 ± 1.83	p = .006
Trails B errors	−2.48 ± 2.07	−1.76 ± 3.63	p = .647
COWA	−2.15 ± .94	−1.12 ± 1.15	p = .026
Proverb Interpretation	−2.01 ± 1.49	−.81 ± 1.53	p = .056
Elevator test	−1.70 ± 4.11	−1.60 ± 3.06	p = .940

consistent with clinical observations of confabulation following rupture and repair of anterior communicating artery aneurysms, which tend to result in ventromedial and basal forebrain lesions (Alexander and Freedman, 1984; DeLuca and Diamond, 1995; Fischer et al., 1995). One difference between the current anatomical findings and previous reports is the suggestion that in some cases (patients 145 and 150) confabulation may result from damage to anterior cingulate regions alone, in the absence of damage to sub-genual and orbital areas. Reanalysis of the scans for these patients confirmed that there was no positive evidence of lesion in these more ventral regions. However, we cannot definitely exclude the possibility of damage that was not visible on the scans, particularly given the typical pattern of lesions following anterior communicating artery aneurysms (the aetiology in both cases), and given the fact that patient 150 had a severe

memory deficit, often associated with damage to the basal forebrain. Alternatively, it may be that damage to the anterior cingulate in the absence of more ventral lesions is associated with transitory confabulation in the acute phase (these patients were tested 17 and 7 days after surgery), but that additional ventral damage is necessary for more chronic confabulation.

In contrast to the patient literature, our anatomical findings do not relate so simply to neuroimaging results. Cue specification and strategic monitoring processes thought to be involved in confabulation have been localised to the right lateral prefrontal cortex (Cabeza et al., 2003; Fletcher et al., 1998; Henson et al., 1999; see Shallice, 2001, 2006 for reviews). Our findings indicate that inferior medial frontal regions are more important than right lateral frontal regions in confabulation. However, this result may be less inconsistent with neuroimaging evidence than it at first appears. Recent neuroimaging findings have reported orbital and medial activations specifically in studies examining autobiographical retrieval (Graham et al., 2003; Maguire, 2001). The orbital and medial frontal lobes have also been associated in neuroimaging studies with functions including reflecting on one's own mental states (Frith and Frith, 2003), and recall of time contextual memory (Fujii et al., 2002). Both of these processes have been theoretically implicated in the production of confabulation. Moreover, Gilboa (2004) has recently proposed that the right dorsolateral PFC and the ventromedial PFC may subservise two distinct monitoring processes, with the right dorsolateral PFC involved in the types of conscious elaborate monitoring often required in experimental episodic memory tasks, and the ventromedial PFC involved in quick intuitive "feeling of rightness" responses relating to the self and involved in autobiographical memory retrieval (see also King et al., 2005). It is this second sort of monitoring process which would be impaired in confabulation. Given the fact that orbital and medial regions are a major component of the limbo-thalamic system underlying memory (Petrides, 2000), and that they are also involved in cholinergic mechanisms known to modulate learning and memory (Gold, 2003; Thiel, 2003), there seems strong support for the idea that inferior medial regions might be critical in confabulation.

**Table 6 – z-scores on individual memory and executive measures for the eight high-confabulator patients**

	143	131	108	106	136	145	150	110
<i>Memory measures</i>								
RMT words	−11.94	−6.35	−.14	−3.86	−15.04	−.76	−7.59	.48
RMT faces	−2.78	−3.13	.20	.20	−3.48	−.50	−7.69	.55
Story recall: immediate	−4.10	−2.49	−1.35	−1.12	−3.87	.15	−2.15	−1.46
Story recall: delayed	−4.02	−3.12	−1.32	−1.10	−3.24	−.20	−2.79	−.99
Rey figure delayed recall	−2.58	−1.21	−.40	NA	−2.58	−1.77	−2.58	−.96
Doors and People	−3.05	NA	−2.42	−3.05	NA	−.85	NA	−.85
<i>Executive measures</i>								
Stroop colour-word score	−2.82	−.98	.00	−5.27	−5.57	−2.69	−5.70	NA
Trails B errors	−2.21	−3.17	1.10	−4.99	NA	−3.72	NA	−1.90
COWA	−1.33	−1.25	−1.00	−3.21	−3.45	−2.15	−2.88	−1.90
Proverb Interpretation	−2.73	−.05	−.95	−1.84	−4.96	−.95	−2.29	−2.29
Elevator test	.38	.38	.38	−2.00	−11.52	−2.00	.38	.38

"NA" indicates data not available for that patient.

As spontaneous confabulations cannot be quantified and statistically investigated in a controlled group study, the present study used a confabulation battery to examine provoked confabulations. It has previously been claimed that provoked confabulations have no anatomic specificity (Schnider, 2003). Therefore, our findings might be seen as surprising. However, that work defined provoked confabulations as false recalls in a word list learning paradigm (Schnider et al., 1996a). The confabulation battery employed here elicited a rather different type of confabulation, in personal episodic memory (PEM) and orientation to time (OT). As described in Section 3.3, at least three of the high-confabulating frontal patients also produced spontaneous confabulations. Moreover, six of the eight patients produced invented or bizarre responses, indicating a qualitatively different process to that involved in producing the normal memory errors which Kopelman (1987) intended to capture by the term “provoked”. The confabulations elicited by this paradigm might therefore be more similar to spontaneous confabulations. Certainly our results suggest that they share a similar anatomical basis.

The results obtained relating to the left and right lateral regions were unexpected. Overall, lateral regions appear to be less involved in confabulation. However, our data suggest that different patterns of impairment ensue following damage to these areas. The left lateral group produced more confabulations compared to controls in response to PEM questions, whilst those with right lateral damage were unimpaired. This left lateral confabulation effect in PEM is consistent with reports that left lateralised regions are more active in retrieval of autobiographical event memories (Maguire, 2001). Conversely, the right lateral group produced more confabulations compared to controls in response to OT questions, whilst those with left lateral damage were unimpaired. Therefore, although lateral regions do not appear to be the most critical in the production of confabulation (in comparison to inferior medial regions), they may contribute via control of processes relating specifically to autobiographical memory or orientation in time.

One issue which is key to any neuroanatomical account of confabulation is the transitory nature of the phenomenon. Confabulation is often an acute rather than a chronic feature, tending to reduce or disappear a few months after injury. The use of a largely acute sample enabled us to identify confabulatory tendencies in this phase and the site of the associated lesions. However, it is very probable that some of these patients will not have been confabulating several months later, despite naturally having the same site of lesion. This may reflect reorganisation of function in the chronic phase. On the other hand, one of our high-confabulating patients (patient 131) continued to confabulate 24 years after his anterior communicating artery aneurysm. The factors determining the variable course of confabulation, even when associated with the same aetiology and lesion site, clearly require future research attention.

The findings regarding the contribution of memory and executive functioning to confabulation were complex. At the group level, general memory performance was able to distinguish between confabulating and non-confabulating frontal groups, whilst the difference between the groups in general executive functioning narrowly missed significance.

Examination of individual tests revealed that all memory measures discriminated between confabulating and non-confabulating patients, but only two executive measures: COWA and Stroop interference score, distinguished the groups. Our findings therefore indicate that executive processes involved in verbal fluency (COWA) and resistance to interference (Stroop colour-word score) are associated with confabulation, whereas set shifting (Trails B errors), sustained attention (Elevator test) and abstract/concrete thinking (Proverbs test) are not.

These results are puzzling in the light of previous reports that found no association between confabulation and verbal fluency (Cunningham et al., 1997; Fischer et al., 1995; Nys et al., 2004), or interference as measured by Stroop colour-word score (Nys et al., 2004). Cunningham et al. (1997) reported a trend towards lower Stroop colour-word scores amongst their high-confabulator group, but this did not reach significance. Instead previous studies have associated confabulation with sustained attention (as measured by time to complete parts A and B of the Trail Making Test, Cunningham et al., 1997), set shifting (as measured by time and errors on part B of the Trail making Test; Cunningham et al., 1997; Fischer et al., 1995), mental flexibility (as measured by the Brixton Spatial Anticipation and Visual Elevator tests, Nys et al., 2004), and perseveration (as measured by the Wisconsin Card Sorting Test; Fischer et al., 1995).

Examination of the individual scores of the high-confabulating frontal group may go some way to explaining these discrepancies. There was in fact considerable variability in performance, with some confabulating patients performing in the normal range on many memory and executive measures. Moreover, there was no single measure on which all confabulating patients showed an impairment, nor on which they were all preserved. Whilst some form of memory impairment does seem to be a necessary condition for confabulation to occur, the critical executive deficit, if any, is less clear.

We suggest that the executive impairments found in confabulating patients may be peripheral rather than causative, with the variation in presentation resulting from variation in lesion location or aetiology. Instead it seems likely that a memory impairment in confabulating patients is overlain with a confabulation-specific impairment, localised to the inferior medial frontal lobe, and not reliably tapped by traditional executive tests. Three theories proposing selective memory-control processes associated with the inferior medial frontal lobe have been proposed. Damasio et al. (1985) have proposed that whilst confabulating patients are able to learn individual modal stimuli, the critical deficit is an inability to integrate these stimuli in the correct context at retrieval due to a modal mismatching effect, particularly regarding the temporal relations of memory fragments. They propose that this deficit is a direct result of damage to the basal forebrain and orbitofrontal cortex, which reduces cholinergic innervation of the hippocampus and disrupts hippocampal functioning. Schnider (2003) and Schnider and Ptak (1999) have proposed that the critical deficit in confabulation is an inability to suppress memories that do not pertain to “now”. This function has also been specifically linked to the anterior limbic system, of which the orbitofrontal cortex is a central component (Schnider et al., 2000; Treyer et al., 2003). Most

recently, Gilboa (2004), Gilboa and Moscovitch (2002) and Gilboa et al. (2006) have suggested that confabulation results from failure of a monitoring system which facilitates early, intuitive rejection of false memories on the basis of “feeling of rightness”. This system is proposed to be localised to the ventromedial PFC. Impairments in these functions seem more likely to distinguish between confabulating and non-confabulating patients than performance on traditional executive tests. Further work is required to establish which of these accounts is best able to account for all of the characteristics of confabulation. However, the position that confabulation is a result either of general frontal damage, or of a general executive deficit in combination with a memory impairment is not specific enough to account for the available evidence. Instead confabulation must result from disruption of a selective executive or memory-control function localised to the inferior medial frontal lobe.

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