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Susceptibility to false memories in patients with ACoA aneurysm

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ABSTRACT

We examined ACoA patients regarding their susceptibility to a range of false memory phenomena. We targeted provoked confabulation, false recall and false recognition in the Deese–Roediger–McDermott-paradigm (DRM-paradigm) as well as false recognition in a mirror reading task. ACoA patients produced more provoked confabulations and more false recognition in mirror reading than comparison subjects. Conversely, false recall/false recognition in the DRM-paradigm were similar in patients and controls. Whereas the former two indices of false memories were correlated, no relationship was revealed with the DRM-paradigm. Our results suggest that rupture of ACoA aneurysm leads to an increased susceptibility to a subset of false memories types.

1. Introduction

Rupture of aneurysms of the anterior communicating artery (ACoA) may lead to the so-called ACoA syndrome. The 'full blown' ACoA syndrome is characterized by amnesia, personality changes, executive dysfunctions, and confabulations (for a review see DeLuca & Diamond, 1995). However, the concomitant occurrence of all symptoms is rather rare due to significant improvements in neurosurgery and medical care (DeLuca & Locker, 1996; Eslinger & Damasio, 1984; Hütter & Gilsbach, 1992; Teissier du Cros & Lhermitte, 1984).

Brain damage after ruptured ACoA aneurysm primarily affects the basal forebrain and the frontal lobes (DeLuca, 1993; DeLuca & Diamond, 1995; Parkin, Yeomans, & Bindschaedler, 1994; Van der Linden, Bruyer, Roland, & Schils, 1993). Impairment of anterograde memory, especially a reduced delayed recall, is the most robust and consistent finding in patients with ruptured ACoA aneurysm (Damasio, Graff-Radford, Eslinger, Damasio, & Kassell, 1985; DeLuca, 1993; DeLuca & Diamond, 1995). Its anatomical substrate is generally ascribed to lesions within the basal forebrain (Alexander & Freedman, 1984; Babinsky, Spiske, Markowitsch, & Engel, 1997; Böttger, Prosiegel, Steiger, & Yassouridis, 1998; Damasio et al., 1985). Meanwhile, executive dysfunctions and personality changes following rupture of aneurysm of the ACoA are generally associated with lesions in frontal lobe regions (Böttger et al., 1998; Damasio et al., 1985; Fujii et al., 2005; Stenhouse, Knight, Longmore, & Bishara, 1991). The anatomical basis of confabulations that can be found in a subset of ACoA patients is less clear. Some authors assume that damage either to the (pre)frontal cortex or the basal forebrain is alone sufficient to produce confabulations (Gilboa et al., 2006; Hashimoto, Tanaka, & Nakano, 2000; Schnider, von Däniken, & Gutbrod, 1996; Stuss, Alexander, Lieberman, & Levine, 1978). Others found that only patients with combined damage of the basal forebrain and the frontal lobes will show confabulations (DeLuca, 1993; DeLuca & Cicerone, 1991).

Confabulations together with intrusions and false recognition are generally subsumed under the topic of false memory phenomena. Intrusions refer to the production of non-studied information in memory experiments (Dodson & Schacter, 2002; Schacter, Norman, & Koutstaal, 1998). False recognition describes the effect that subjects falsely recognize a novel item, object or event as familiar even though it was not presented during studying (Dodson & Schacter, 2002; Schacter, Norman, et al., 1998). In the laboratory, intrusions and false recognition can be reliable elicited with the so-called Deese–Roediger–McDermott-paradigm (DRM-paradigm). In this paradigm, individuals study word lists of

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semantic associates that are all converging on a non-studied critical word ('critical lure') representing the gist of the list. In a subsequent test phase, subjects disproportionately often falsely recall or recognize the critical lure (e.g. Blair, Lenton, & Hastie, 2002; Ciaramelli, Ghetti, Frattarelli, & Ladavas, 2006; Coane & McBride, 2006; Melo, Winocur, & Moscovitch, 1999; Roediger & McDermott, 1995).

While characteristics of intrusions and false recognition are more clear-cut, a unique definition of confabulation does not exist. In a very broad sense, confabulations can be defined 'as statements or actions that involve distortions of memory' (Metcalf, Langdon, & Coltheart, 2007). Often they concern retrograde aspects of autobiographical/episodic memory (Benson et al., 1996; Dalla Barba, Cappelletti, & Denes, 1990; Gilboa & Moscovitch, 2002), but they may also affect current reality or personal future (Dalla Barba, Cappelletti, Signorini, & Denes, 1997; Dalla Barba, Nedjam, & Dubois, 1999). Occasionally, confabulations even affect semantic memory (Dalla Barba, 1993b; Kopelman, Ng, & Van Den Brouke, 1997; Moscovitch & Melo, 1997). Confabulations may occur 'spontaneously' in a patient's everyday life without any external trigger or they may arise 'provoked' by questions probing memory. Whether these two subtypes of confabulation are the same or distinct entities with similar or different anatomical correlates is controversial. While some authors consider only patients to be confabulators who display spontaneous confabulation in everyday life (Kopelman, 1987, 1999; Schnider, 2003), others consider also experimentally induced false narratives as confabulations (Dalla Barba, Boisse, Bartolomeo, & Bachoud-Levi, 1997; Fotopoulou, Conway, & Solms, 2007; Nedjam, Devouche, & Dalla Barba, 2004; Turner, Cipolotti, Yousry, & Shallice, 2008).

Partly due to this conceptual ambiguity in the definition of confabulation, it is difficult to compare studies of ACoA patients' confabulation and those of ACoA patients' false memory tendencies. Comparability between studies is further complicated by the fact that the vast majority of studies describe single cases of ACoA confabulators (e.g. Dalla Barba, Cappelletti, et al., 1997; Fotopoulou, Conway, Griffiths, Birchall, & Tyrer, 2007; Schnider, Bonvallat, Emond, & Leemann, 2005), whereas others examine groups of confabulating patients per se, but with different aetiologies and different onset times after surgery or in the course of diseases (e.g. Moscovitch & Melo, 1997; Schnider et al., 1996; Stuss et al., 1978). Moreover, most of the studies investigating false memories in ACoA patients concentrate only on one form of manifestation, i.e. provoked confabulation (e.g. Ciaramelli, Ghetti, & Borsotti, 2009; Dalla Barba, Cappelletti, et al., 1997; Fotopoulou, Conway, Griffiths, et al., 2007) or susceptibility to intrusions or false recognitions (e.g. Hanley et al., 2001; Parkin, Ward, Bindschaedler, Squires, & Powell, 1999; Schnider, 1999).

Likewise, although some have postulated that (provoked) confabulation and false recall/recognition are dissociated phenomena of false memories (for a review see Schnider, 2001, 2008), experimental studies examining the relationship between these different types of false memories are rare (for exceptions see Fischer, Alexander, D'Esposito, & Otto, 1995; Gilboa et al., 2006; Van Damme & d'Ydewalle, in press). For that reason, in our study we examined a larger group of ACoA patients regarding their susceptibility to different types of false memory phenomena, i.e. provoked confabulations, intrusions and false recognitions. Further, we investigated whether these different forms do have a relationship or whether they are independent from each other and hence can be regarded as distinct false memory phenomena. To elicit provoked confabulations we selected the Confabulation Interview (Borsutzky, Fujiwara, & Markowitsch, 2006), the German adaptation of Dalla Barba's Confabulation Battery (Dalla Barba, 1993a, 1993b; Dalla Barba, Cappelletti, et al., 1997). With this test, confabulation tendencies in different memory domains, such as personal semantic memory, episodic/autobiographical memory, general semantic memory, personal future and orientation, can reliably be assessed. To provoke intrusions and false recognition we used the DRM-paradigm, well-known for its reliability of inducing false memories: Whereas healthy subjects produce false recognition to critical lures in the DRM-paradigm (Blair et al., 2002; Roediger & McDermott, 1995), amnesic patients show reduced rates of false recognition to critical lures, possibly due to a lack of gist memory (Melo et al., 1999; Schacter & Slotnick, 2004). In contrast, confabulating patients again tend to be more susceptible to false recognition of unrelated lures in the DRM, as several studies suggest (Ciaramelli et al., 2009; Ciaramelli et al., 2006; Van Damme & d'Ydewalle, in press). Therefore, if our ACoA patients should represent a group of noticeable confabulators, they may also produce higher rates of false recognitions of unrelated lures in the DRM. Moreover, we were interested in whether false memories may also occur in non-declarative memory, i.e. procedural memory. To our knowledge studies investigating false memories in the procedural memory domain do not yet exist. Procedural memory skills are usually preserved after ACoA rupture (Bondi, Kaszniak, Rapcsak, & Butters, 1993; Stefanova, Kostic, Ziropadja, Markovic, & Ocic, 2000; Thomas-Anterion et al., 1996). However, procedural memory performance may also depend on explicit memory strategies, so that interference or other problems affecting explicit memory processes in pathological conditions may also impact implicit memory tasks (Bayley, Frascino, & Squire, 2005) Thus, it may be possible to observe ACoA patients' faulty memory production also within procedural memory. A new version of the mirror reading task was created for this purpose, suitable to elicit reading errors reflecting false recognitions that in turn affected procedural memory.

2. Methods

2.1. Participants

2.1.1. ACoA patients

Patients were recruited from two neurosurgical units (Clinic for Neurosurgery, Bielefeld-Bethel, House Gilead I and the Clinic for Neurosurgery, Medical University Hannover). All patients had rupture and repair of an aneurysm of the anterior communicating artery (ACoA). Of the total of 30 patients initially recruited 12 were excluded from the study for the following reasons: two patients died, three patients refused to take part in the study, four patients were unable to perform the tests due to extensive cognitive deficits and/or aphasic disorder, two patients had insufficient knowledge of German and one patient's address was unknown. The remaining 18 ACoA patients volunteered for the study. They did not receive financial incentives for their participation. Before examination, patients as well as healthy controls gave their informed consent to participate in the study according to the Declaration of Helsinki. All patients underwent surgical repair of their aneurysm, 17 by clipping the aneurysm and one by coiling. On admission, six patients were classified with Hunt and Hess (Hunt & Hess, 1968) neurological Grade I, five with Grade II, two had been considered to be in Grade III and one to be Grade IV. For four patients classification was not available. Amount and distribution of subarachnoid blood was classified in four grades according to Fisher, Kistler, and Davis (1980). In two patients no blood was detected, four patients had a diffuse deposition or thin layer with all vertical layers of blood less than 1 mm thick, four patients showed localized clots and/or vertical layers of blood 1 mm or greater in thickness and two patients had diffuse or no subarachnoid blood, but showed intracerebral or intraventricular clots. For the remaining six patients no classification was made. None of the patients had any history of neurologic disorder prior to their actual subarachnoid haemorrhage. Time between surgery and testing ranged from 30 to 57 days ($M = 39.11$, SD = 9.21). Only one patient exhibited spontaneous confabulation during testing.

Of the 18 patients 11 were women and seven were men. The patients' age ranged from 33 to 68 years ($M = 52.67$, SD = 10.00). Average years of school education was 9.11 years (SD = 1.60). Two patients were unskilled workers, 15 had an apprenticeship and one patient had an academic occupation. Demographic characteristics of the ACoA group and the comparison group are presented in Table 1.

2.1.2. Comparison group

The comparison group consisted of 17 adults without any neurological or psychiatric history. They were between 35 and 71 years old ($M = 56.18$, SD = 10.50) and were matched to the patients for sex, age, years of education and occupation (see Table 1).

Table 1
Demographic variables of ACoA patients (ACoA) and comparison group (CG).

		ACoA $N = 18$	CG $N = 17$	Significance
Age	Mean (SD[a])	52.67 (10.00)	56.18 (10.50)	$t = -1.01$ ($p = .318$)
Gender	Male	7	7	$\chi^2 = 0.02$ ($p = .890$)
	Female	11	10	
Years of school education	Mean (SD[a])	9.11 (1.60)	8.94 (1.35)	$t = .34$ ($p = .737$)
Occupation	Unskilled	2	3	$\chi^2 = .32$ ($p = .854$)
	Apprenticeship	15	13	
	Academic	1	1	

[a] Standard deviation.

2.2. Material

All participants underwent extensive neuropsychological evaluation. They were tested individually in two sessions each taking between 1.5 and 2 h. Breaks were given on demand of the participants. The identical test battery was administered in the same order to all participants. Neuropsychological tests were administered according to standard published procedures.

2.3. Neuropsychological test battery

2.3.1. Intelligence

To estimate intellectual functioning we used the subtest 'Reasoning' from the Leistungsprüfsystem (Horn, 1983). This subtest measures logical reasoning and is highly correlated with the overall result of the test. Therefore, it provides a valid estimation of the global intelligence quotient.

2.3.2. Memory

Short term memory was assessed with the 'Digit span forward' and working memory was assessed with the 'Digit span reversed', both from the German version (Härting et al., 2000) of the Wechsler Memory Scale-Revised (Wechsler, 1987). Verbal learning was evaluated by the mean recall of words across five learning trials from a selective reminding task (Memo-Test, Schaaf, Kessler, Grond, & Fink, 1992) and verbal long-term memory was measured with the 20-min delayed recall of words from the Memo-Test (Schaaf et al., 1992). Visual long-term memory was evaluated by the 30-min delayed recall of the Rey-Osterrieth Complex Figure (Osterrieth, 1944).

2.3.3. Attention

To measure speed of information processing we used the Trail making Test Part A (TMT-A) (Reitan, 1958) as well as the 'Word Trial' and the 'Colour Trial' of the German version (Farbe-Wort-Interferenztest, Bäumler, 1985) of the Stroop Test (Stroop, 1935). Sustained attention was examined with the Concentration Endurance Test 'd2′ (Aufmerksamkeits-Belastungs-Test, Brickenkamp, 1994).

2.3.4. Executive functions

To assess the ability to form concepts, to shift and maintain set, and to utilize feedback, we administered a computerised version of the Modified Wisconsin Card Sorting Test (Nelson, 1976). Susceptibility to interference was measured with the 'Interference Trial' of the Colour Word Interference Test (Bäumler, 1985). Mental flexibility was assessed by the Trail making Test Part B (TMT-B, Reitan, 1958). To examine verbal fluency we used the FAS-Test (Spreen & Strauss, 1998).

2.3.5. Visual-spatial abilities

The copy of the Rey-Osterrieth Figure (Osterrieth, 1944) was used to evaluate visual-spatial abilities.

2.4. False memory tests

2.4.1. Confabulation Interview

For quantitative measurement of confabulation, we used the Confabulation Interview (from the Testbatterie zur Erfassung von Konfabulationstendenzen, TEKT, Borsutzky et al., 2006), the German adaptation of the Confabulation Battery of Dalla Barba and colleagues (Dalla Barba, 1993a; Dalla Barba et al., 1999).

The Confabulation Interview is a semi-structured interview consisting of 55 questions probing different memory domains. It contains 10 questions on personal semantic memory, 10 on episodic/autobiographical memory, 10 on general semantic memory, 10 questions on personal future and 10 on orientation. Questions on personal semantic memory address general personal facts, such as name, date and place of birth. Questions on episodic (autobiographical) memory address autobiographical events. Questions on general semantic memory prompt knowledge of German news events and famous people from several decades. Questions on personal future concern prospective planned activities. Questions probing orientation in time and place were taken from the Mini-Mental-State-Examination (Kessler, Markowitsch, & Denzler, 1990). Furthermore, five distractor questions on general semantic memory and 5 on episodic memory were included. These questions are logically coherent but have no correct answer, so that the only appropriate response to these questions would be 'I don't know'. One example for each domain is given in Table 2. Participants' answers were scored as 'correct', 'omission' or 'confabulation'. The correctness of answers concerning personal semantic memory, episodic memory and personal future was verified by relatives. If relatives were not able to judge correctness, participants' answers were scored as 'correct'. For distractor questions only answers implying 'I don't know' or the like were accepted as 'correct'; any other response was scored as 'confabulation'. For more details on test application and confabulation scoring see Borsutzky, Fujiwara, Brand, and Markowitsch (2008).

2.4.2. DRM-paradigm

To measure false memories such as intrusions and false recognition in anterograde memory we used the Deese–Roediger–McDermott (DRM) paradigm (see Deese, 1957; Roediger & McDermott, 1995). We selected and translated five word lists (each list containing of 15 semantically associated words) from those used by Roediger and McDermott (Roediger & McDermott, 1995). Critical lures of these lists were: *Chair*, *doctor*, *sleep*, *anger* and *sweet*. As customary, the critical lures are semantically closely associated to the items of their respective list, but were never presented. The five lists were read by the experimenter and participants were instructed to remember as many words as possible for a subsequent free recall test that followed each list, but they were not informed about the relatedness of studied words. Immediately after learning and free recall of the five word lists an unexpected final recognition test followed. The recognition test consisted of 25 words in total: 15 old (studied) and 10 new (unstudied) words. Old words included the 1st, 8th and 11th word of each list. The 10 new words were the five list-related 'critical lures' and five unrelated lures. All words were presented in one fixed randomized order. Participants were required to indicate for each word whether it was a studied ('old') item or whether the word was presented for the first time now ('new').

Table 2
Items of the Confabulation Interview: one example for each memory domain and the number of the corresponding items.

Domain	Example-Item	Number of questions
Personal semantic memory	What are your parents' first names?	10
Episodic memory	What have you done last New Year's Eve?	10
General semantic memory	Why did the sheep 'Dolly' become prominent?	10
Personal future	When will you be off for your next big vacation? Where to?	5
Orientation	What city are we in here?	10
Distractor episodic	Do you remember what exactly you did on March 13th, 1985?	5
Distractor semantic	Why did the pharmaceutical company 'Espelkamp' make the headlines?	5

2.4.3. Mirror reading

To measure false memory in procedural memory we constructed a new version of the mirror reading task: A series of 15 cards each consisting of two German words in mirror writing (30 words in total, all words with eight until ten letters) were visually presented to the participants. The subjects were asked to read the words as quickly and accurately as possible. Upon correct reading of the two words of one card, the next card was presented. The time by which a subject had read both words correctly constituted the reading time measure. If a subject read a word incorrectly, they were being told so. However, the correct word was not provided. In addition, all incorrect responses were counted. After a delay of 30 min an unannounced second trial was administered. Herein, ten words (on five cards) were identical with the first trial, ten words were new and ten words were also new but similar in orthography to ten words of trial 1 (e.g. *Explosion and Exkursion*, see Table 3). The sequence of cards was randomized for both trials.

Improvement in reading speed from trial 1 to trial 2 of the recurring words served to measure priming performance ('Priming'). Improvement in reading time of new words was used for an index of skill acquisition, i.e. procedural learning ('Procedural Memory'). Those words of trial 2 that were orthographically similar to words of trial 1 ('Interference') were created to induce interference in reading. We assumed that due to these similarities in orthography with the words of trial 1, subjects were disturbed in automatic reading processes. In detail, in trial 2 subjects may initially tend to recognize the similarly looking words of trial 1 due to priming effects, but during reading, they will notice most discrepancies and will then have to execute a more effortful analysis. On the one hand, this may lead to longer reading times. On the other hand, if subjects have deficits in suppressing the activated, but irrelevant memory trace (i.e., the 'Interference' words of trial 1), they may produce more mistakes in reading of 'Interference' words in trial 2 compared to 'Priming' and 'Procedural Memory' items.

3. Results

3.1. Neuropsychological test battery

Table 4 shows the results of the psychometric tests in both groups. There was no evidence for intellectual deterioration in ACoA patients. In contrast, ACoA patients exhibited anterograde memory impairment typically found in patients with rupture of the ACoA: They showed difficulties with verbal learning (Memo-Test Trial 5: ACoA: $M = 6.33$, SD = 1.88; CG: $M = 7.82$, SD = 1.42; $t = .291$, $p = .013$) and exhibited a reduced delayed recall of verbal material (Memo-Test delayed recall: ACoA: $M = 5.67$, SD = 1.17; CG: $M = 6.63$, SD = 1.17, $t = -2.40$, $p = .022$). Furthermore, ACoA patients were obviously impaired in verbal fluency (FAS-Test: ACoA: $M = 12.72$, SD = 14.65; CG: $M = 66.53$, SD = 27.19; $t = -7.24$, $p < .001$). In all other cognitive domains ACoA patients scored within normative data in tests for which norms were available, although they were often outperformed by controls (see Table 4 for details).

In summary, our patients showed the typical memory impairment following rupture and repair of the ACoA. In contrast, with the exception of verbal fluency, patients performed all other tests within the normal range according to test norms even though at lower levels than their healthy comparison subjects.

3.2. False memory tests

3.2.1. Confabulation Interview

Since the different domains of the Confabulations Interview comprise different numbers of questions (5 versus 10), for purposes of comparison all scores were converted into percentages. Table 5 shows the results in the Confabulations Interview of both groups.

In the Confabulation Interview, ACoA patients exhibited a strikingly poorer memory performance than healthy controls: Their total score of correct answers ('Memory Score') was significantly lowered (ACoA: $M = 83.33\%$, SD = 10.40; CG: $M = 92.51\%$, SD = 3.45; $t = -3.54$, $p = .002$) and their total number of confabulations ('Confabulation Score') was significantly elevated (ACoA: $M = 5.35\%$, SD = 6.64; CG: $M = 1.39\%$, SD = 1.51; $t = 2.46$, $p = .024$).

Taking a look at each single domain, we found group differences in episodic and general semantic memory as well as in orientation: To episodic memory questions, patients gave fewer correct answers (ACoA: median = 90%, range = 20–100; CG: median = 100%, range = 90–100; $U = 83.50$, $p = .020$). Patients also showed more episodic memory confabulations than controls (ACoA: median = 0%, range = 0–70; CG: median = 0%, range = 0; $U = 93.50$, $p = .049$). Regarding general semantic memory and orientation we only detected differences in the number of correct answers, with patients giving fewer correct answers than comparison subjects (general semantic memory: ACoA: median = 50%, range = 20–90; CG: median = 60%, range = 50–100; $U = 91.00$, $p = .041$; orientation: ACoA: median = 100%, range = 60–100; CG: median = 100%, range = 90–100; $U = 91.00$, $p = .041$). Performance in the other domains did not differ between groups (p's > .05).

Within-group comparisons revealed that patients performed worst in general semantic memory, compared to all other domains of the Confabulation Interview (p's < .001, Bonferroni-corrected significance threshold: $p \leq .002$). All other comparisons failed to reach significance. The same pattern was found in the comparison group (general semantic memory compared to all other domains: $p < .002$) indicating that questions probing general semantic memory were most difficult for all subjects. The domains of the Confabulation Interview did not differ significantly from each other in the number of confabulations they evoked, both within the patient group and the comparison group (all p's > .002). From a more qualitative perspective, it should be noted that some patients displayed a considerable number of confabulations in episodic memory (range = 0–70). This may suggest that our patients are most susceptible to provoked confabulations within episodic memory. In contrast, the range of 'confabulations' in healthy subjects was most inflated in the semantic distractor domain (range = 0–40).

3.2.2. DRM-paradigm

In accordance with previous research (Ciaramelli & Ghetti, 2007; Schacter, Verfaellie, Anes, & Racine, 1998), we calculated the mean proportions of recalled and recognized words. In Table 6 the mean proportions for accurate and false recall as well as the mean proportions for correct and false recognition are presented.

3.2.3. Free recall

As can be seen in Table 6 ACoA patients and comparison subjects differed in mean recall proportions for word lists with patients recalling significantly fewer list words than healthy controls ($t = -4.12$, $p < .001$). On the other hand groups did not differ in mean recall proportions for critical lures ($t = -.95$, $p = .352$).

Within-group comparisons revealed that patients produced about the same proportion of accurate and false recall (.32 versus .30; $t = .34$, $p = .740$). Healthy subjects showed a marginally but non-significantly larger mean recall proportion for list words compared to critical lures (.49 versus .38; $t = 1.77$, $p = .097$).

3.2.4. Recognition

We found a significant group difference regarding correct recognition of studied words (hit rate), with patients recognizing significantly fewer studied words ($t = -3.11$, $p = .004$). In contrast, for recognition proportions of critical lures (false alarms) no significant group-difference was revealed ($t = -1.76$, $p = .088$). Furthermore, ACoA patients and comparison subjects had similar false recognition proportions of unrelated lures (.06 versus .03, $t = .77$, $p = .445$) (see Table 6).

Within both groups, proportions of correctly recognized studied words and the proportion of falsely recognized critical lures did not differ from each other (ACoA: $t = 1.17$, $p = .262$, CG: $t = .94$, $p = .359$). Compared to recognition of studied words and critical lures, both

Table 3
Items of the mirror reading task: one example for each item type[a].

	Priming	Procedural memory	Interference
Trial 1:	Umarmung Zwietracht	Eifersucht Sympathie	Schrecken Wohlstand
Trial 2:	Umarmung Zwietracht	Detonation Erkenntnis	Schrecken Wohlklang

[a] The English translation of the words:
Priming: embracement and discord.
Procedural/Trial 1: jealousy and sympathy, Trial 2: detonation and insight.
Interference/Trial 1: horror and prosperity, Trial 2: snails and harmony.

Table 4
Results in the neuropsychological test battery.

	ACoA		CG		Significance	
	Mean	SD[1]	Mean	SD[1]		
Intelligence screening						
LPS[a]-Reasoning (IQ)[2]	101.24	9.09	106.00	6.44	$t = -1.76$	$p = .087$
Memory						
Digit span forward[3]	33.22	30.80	68.71	25.63	$t = -3.69$	$p = \mathbf{.001}$
Digit span reversed[3]	27.17	29.57	43.35	27.96	$t = -1.66$	$p = .106$
Memo-Test (trial 1)[4]	4.72	1.36	4.88	1.80	$t = -.30$	$p = .768$
Memo-Test (trial 5)[4]	6.33	1.88	7.82	1.42	$t = .291$	$p = \mathbf{.013}$
Memo-Test (delayed)[4]	5.67	1.40	6.63	1.17	$t = -2.40$	$p = \mathbf{.022}$
Rey-Osterrieth Figure (delayed)[3]	37.56	22.19	47.71	27.36	$t = -1.21$	$p = .235$
Attention/information processing						
Word Trial (FWIT[b])[3]	43.28	24.75	71.77	19.01	$t = 3.80$	$p = \mathbf{.001}$
Colour Trial (FWIT[b])[3]	49.56	32.22	76.29	20.74	$t = 2.94$	$p = \mathbf{.006}$
TMT[c]-A[3]	33.50	27.43	58.41	29.20	$t = -2.60$	$p = \mathbf{.014}$
d2[d] (total – error)[3]	28.97	27.76	68.73	18.89	$t = -4.84$	$p < \mathbf{.001}$
Executive functions						
Interference Trial (FWIT[b])[3]	47.67	35.56	68.06	17.29	$t = -2.18$	$p = \mathbf{.039}$
FAS[e]-Test[3]	12.72	14.65	66.53	27.19	$t = -7.24$	$p < \mathbf{.001}$
TMT[c]-B[3]	27.89	33.00	53.65	20.27	$t = -2.80$	$p = \mathbf{.009}$
MWCST[f]						
Correct[4]	36.82	9.15	36.44	6.36	$t = .14$	$p = .890$
Errors[4]	7.53	5.20	8.69	4.11	$t = .15$	$p = .485$
Visuo-constructive abilities						
Rey-Osterrieth Figure (copy)[3]	37.94	29.82	45.06	20.91	$t = -.82$	$p = .418$

[a] Leistungsprüfsystem.
[b] Farbe-Wort-Interferenztest (German version of the Stroop Test).
[c] Trail Making Test.
[d] Concentration Endurance Test.
[e] Verbal fluency.
[f] Modified Wisconsin Card Sorting Test.
[1] Standard deviation.
[2] IQ-scaled score.
[3] Percentile.
[4] Row scores.

groups exhibited very low rates of unrelated lure recognition (all p's < .001).

3.2.5. Mirror reading

We calculated two separate ANOVAS with repeated measures, one on reading time (dependent) and one on reading mistakes (dependent) both including the between-subject factor 'Group' (ACoA patients versus comparison group) and the within-subjects factors 'item type' (priming versus procedural memory versus interference) and 'trial' (trial 1 versus trial 2).

For reading time a main significant effect was revealed for group ($F = 9.75$, $p = .004$) due to extended reading times of ACoA patients. Furthermore, there was a main significant effect for 'trial' ($F = 27.68$, $p < .001$) showing that reading times differed between trials. Additionally a significant interaction for 'item type' × 'trial' was found ($F = 17.52$, $p < .001$), which may be due to a missing improvement in the speed of reading the 'Interference' words in trial 2. Within-group comparisons revealed that both groups improved significantly in reading speed of priming and procedural memory words from trial 1 to trial 2 (ACoA patients/priming: $t = 8.24$, $p < .001$; ACoA patients/procedural memory: $t = 2.74$, $p = .015$; CG/priming: $t = 4.69$, $p < .001$; CG/procedural memory: $t = 3.30$, $p = .004$). In contrast, for 'interference' words there was no improvement from trial 1 to trial 2, neither in the patient group ($t = -1.14$, $p = .269$) nor in the comparison group ($t = .50$, $p = .627$).

An analogous repeated measures ANOVA on reading mistakes showed a significant main effect for 'item type' ($F = 26.96$, $p < .001$), a significant 'item type' × 'trial' interaction ($F = 30.04$, $p < .001$) qualified by a three-way interaction of 'item type' × 'trial' × 'group' ($F = 4.24$, $p = .019$). Following up on the three-way interaction showed that patients made more reading mistakes to interference items in trial 2 than comparison subjects ($t = 2.05$, $p = .049$). Furthermore, between trial 1 and trial 2, patients, but not controls had an increase in reading mistakes to interference items (ACoA patients:

Table 5
ACoA patient's and control subject's percentage of correct responses and confabulations in the Confabulation Interview.

	ACoA N = 18		CG N = 17		Significance	
	Median/Mean[a]	Range/SD[a]	Median/Mean[a]	Range/SD[a]		
Total scores						
Memory Score	83.33%[a]	10.40[a]	92.51%[a]	3.45[a]	$t = -3.54$	$p = .002$
Confabulation Score	5.35%[a]	6.64[a]	1.39%[a]	1.51[a]	$t = 2.46$	$p = .024$
Personal semantic memory						
Correct answers	100.00%	70–100	100.00%	80–100	$U = 116.50$	$p = .232$
Confabulations	0.00%	0–10	0.00%	0	$U = 144.50$	$p = .782$
Episodic memory						
Correct answers	90.00%	20–100	100.00%	90–100	$U = 83.50$	$p = .020$
Confabulations	0.00%	0–70	0.00%	0	$U = 93.50$	$p = .049$
General semantic memory						
Correct answers	50.00%	20–90	60%	50–100	$U = 91.00$	$p = .041$
Confabulations	10.00%	0–20	0.00%	0–10	$U = 112.00$	$p = .184$
Personal future						
Correct answers	100.00%	60–100	100.00%	100	$U = 136.00$	$p = .590$
Confabulations	0.00%	0–40	0.00%	0	$U = 136.00$	$p = .590$
Orientation						
Correct answers	100.00%	60–100	100.00%	90–100	$U = 91.00$	$p = .041$
Confabulations	0.00%	0–30	0.00%	0–10	$U = 118.00$	$p = .258$
Distractor episodic						
Correct answers	100.00%	40–100	100.00%	80–100	$U = 136.00$	$p = .590$
Confabulations	0.00%	0–60	0.00%	0–20	$U = 136.00$	$p = .590$
Distractor semantic						
Correct answers	100.00%	80–100	100.00%	60–100	$U = 139.00$	$p = .660$
Confabulations	0.00%	0–20	0.00%	0–40	$U = 139.00$	$p = .660$

[a] In case of normal distributed data means and standard deviation are given.

$t = -3.78$, $p = .002$, CG: $t = -1.24$, $p = .234$). All other comparisons failed to reach significance (p's > .016).

Reading times for the three different item types (priming, procedural memory and interference) are depicted in Fig. 1a; the mean numbers of mistakes are presented in Fig. 1b.

3.3. Correlations

3.3.1. Correlations between types of false memories

In the ACoA group, we found a significant correlation between confabulations in the Confabulations Interview (Confabulation Score) and reading mistakes of interference words in trial 2 of the mirror reading task ($r = .534$, $p = .027$). Proportions of false recall and false recognition of critical lures in the DRM-paradigm did not have any relationship with the two other tests measuring false memories (p's > .05).

In the comparison group no relationships were found between tests inducing false memories (p's > .05). Results of the three tests measuring false memories as well as demographic clinical data are presented for each ACoA patient in Table 7.

Table 6
Results in the DRM-paradigm: mean recall proportions and mean recognition proportions for list words and lures.

	ACoA	CG		
Free recall				
Item type	Mean	Mean	Significance	
Studied words	.32	.49	$t = -4.122$	$p < .001$
Critical lures	.30	.38	$t = -.946$	$p = .352$
Recognition				
Item type	Mean	Mean		
Studied words	.67	.82	$t = -3.107$	$p = .004$
Critical lures	.63	.76	$t = -1.761$	$p = .088$
Unrelated lures	.06	.03	$t = .773$	$p = .445$

3.3.2. Correlations between false memories and other neuropsychological functions

Correlations between false memories and the results in the neuropsychological test battery are summarized in Table 8.

In the ACoA group the Confabulations Score of the Confabulation Interview was negatively correlated with reasoning ($r = -.617$, $p = .008$), verbal learning ($r = -.537$, $p = .022$), delayed memory recall (Memo-Test: $r = -.529$, $p = .024$; Rey-Osterrieth Figure: $r = -.658$, $p = .003$) and some components of attention (Word Trial/FWIT: $r = .475$, $p = .047$, d2: $r = -.515$, $p = .041$). Regarding executive functions all correlations failed to reach significance (p's > .05). Furthermore, the Confabulation Score was negatively correlated with ability to copy the Rey-Osterrieth Figure ($r = -.580$, $p = .012$).

For the DRM-paradigm, we found a negative relationship between the false recall of critical lures and the results in the Colour Trial of the FWIT ($r = -.537$, $p = .032$). For proportions of false recognition (critical lures) no relationship with any of the neuropsychological test results was revealed (p's > .05).

Finally, the number of reading mistakes of interference words in mirror reading was negatively correlated with delayed recall of verbal and figural material (delayed recall Memo-Test: $r = -.590$, $p = .013$, delayed recall Rey-Osterrieth: $r = -.556$, $p = .020$).

For correlations within the comparison group, see Table 8.

To summarize, we found false memories in the Confabulation Interview and mirror reading were associated with disturbed anterograde memory in our ACoA patients. Additionally, their Confabulation Score was correlated with further cognitive aspects such as reasoning, attention and visual-spatial abilities. This may be due to the fact that the Confabulation Interview comprises different memory domains so that an overall score such as the Confabulation Score may also be related to several cognitive functions. In contrast to the Confabulation Interview and the mirror reading task, false memories in the DRM-paradigm were not correlated with memory functions at all, but only with information processing speed. Note

Table 7
Demographic and clinical data of the ACoA patients together with their results in the false memories tests.

Pat.-No.	Gender	Age	Years of school education	Hunt and Hess[a]	Fisher Score[b]	Lesion site	Confabulation Score (Confabulation Interview)	Proportions of false recall of critical lures (DRM-paradigm)	Proportions of false recognition of critical lures (DRM-paradigm)	Reading mistakes of interference words (mirror reading, trial 2)
1	Male	47	8	I	4	Dorsolateral, white matter	1,81	0,2	0,6	1
2	Female	50	13	I	1	Basal forebrain	1,81	0,6	0,6	1
3	Female	68	10	IV	2	Unilateral orbitofrontal (right), dorsolateral	1,81	0	0,4	4
4	Female	51	9	n.s.[c]	n.s.[c]	No scan available	1,81	0,2	0,8	3
5	Male	46	8	III	4	Dorsolateral, temporal	1,81	0	0,2	0
6	Female	40	9	II	3	No scan available	0	0,6	1	2
7	Male	59	13	I	3	Unilateral orbitofrontal (right), dorsolateral, right temporal	0	0,2	0,8	2
8	Male	61	8	n.s.[c]	n.s.[c]	No scan available	1,81	0	0,2	3
9	Male	63	8	I	4	No scan available	1,81	0,6	0,6	3
10	Female	50	8	II	3	No scan available	12,72	0	0,4	2
11	Female	33	9	n.s.[c]	n.s.[c]	No scan available	27,27	0,6	0,8	6
12	Female	60	10	III	n.s.[c]	No scan available	7,27	0,4	0,4	2
13	Female	42	9	II	3	No scan available	3,63	0,2	0,6	2
14	Female	64	10	II	2	Bilateral orbitofrontal	3,63	Not evaluable[d]	Not evaluable[d]	3
15	Female	64	8	I	n.s.[c]	Bilateral orbitofrontal	12,72	0,4	1	5
16	Male	41	8	I	2	Extensive left side brain damage with medial-temporal involvement and damage to the white matter within dorsal/prefrontal and parietal regions	7,27	Not evaluable[d]	Not evaluable[d]	Not evaluable[d]
17	Male	59	8	I	1	Bilateral orbitofrontal	5,45	0,2	0,8	7
18	Female	50	8	II	2	Unilateral orbitofrontal	3,63	0,6	0,8	1

[a] Hunt and Hess Classification of Subarachnoid Hemorrhage (SAH), Grade I–V.

[b] Severity of SAH according to the CCT grading system of Fisher: 1 = no blood detected; 2 = diffuse disposition or a thin layer with all vertical layers of blood < 1 mm; 3 = localized clots and/or vertical layers of blood > 1 mm; 4 = diffuse or no subarachnoid blood, but with intracerebral or intraventricular clots.

[c] Not specified.

[d] Test has to be terminated due to exhaustion.

Table 8
Correlations between false memories in the Confabulation Interview, DRM-paradigm, mirror reading and neuropsychological tests.

Test	Confabulation Score (Confabulation Interview)		Proportions of false recall of critical lures (DRM-paradigm)		Proportions of false recognition of critical lures (DRM-paradigm)		Reading mistakes of interference words (mirror reading, trial 2)	
	ACoA *r*	CG *r*	ACoA *r*	CG *r*	ACoA *r*	CG *r*	ACoA *r*	CG *r*
Intelligence								
LPS[a]-Reasoning	−**.617****	−.072	−.078	.091	−.178	−.078	−.398	.366
Memory								
Digit Span forward	−.136	−.305	.228	.063	.061	.129	.109	−.228
Digit Span reversed	−.458	−.208	.455	.257	.158	.059	−.463	−.333
Memo-Test (trial 1)	−.393	−.438	−.081	−.110	.010	−.154	−.245	−.318
Memo-Test (trial 5)	−**.537***	−.196	.277	−.100	.296	−.202	−.405	−.163
Memo-Test (delayed)	−**.529***	−.201	.237	.067	.155	−.356	−**.590***	−.193
Rey-Osterrieth Figure (delayed)	−**.658****	−.404	−.113	.016	−.091	−**.684****	−**.556***	.266
Attention/information processing								
Word Trail (FWIT[b])	**.475***	−.306	−.234	−.126	−.041	.102	.201	−.082
Colour Trail (FWIT[b])	.468	−.137	−***.537****	−.187	−.461	.292	−.067	.064
TMT[c]-A	.054	−.049	−.371	−.179	−.268	.011	.033	.030
d2[d] (total – error)	−**.515***	−.104	.095	−.214	.038	−.121	−.170	−.102
Executive functions								
Interference (FWIT[b])	.423	.055	−.034	−.297	.218	−.308	.265	−.173
FAS-Test[e]	−.342	.084	.306	.062	.361	−.187	−.102	−.033
TMT-B[c]	.213	.102	−.379	.167	−.445	.349	−.063	−.045
MWCST[f]								
Correct	−.395	.431	.175	−.350	.303	−.240	−.154	.392
Errors	.266	−**.524***	−.115	.340	−.426	.119	.072	−.400
Visual-spatial abilities								
Rey-Osterrieth Figure (copy)	−**.580***	−.146	−.177	−.143	−.327	−.091	−.290	.196

[a] Leistungsprüfsystem.
[b] Farbe-Wort-Interferenztest (German version of the Stroop Test).
[c] Trail Making Test.
[d] Concentration Endurance Test.
[e] Verbal fluency.
[f] Modified Wisconsin Card Sorting Test.
* $p < .05$ (Pearson correlated).
** $p < .01$ (Pearson correlated).

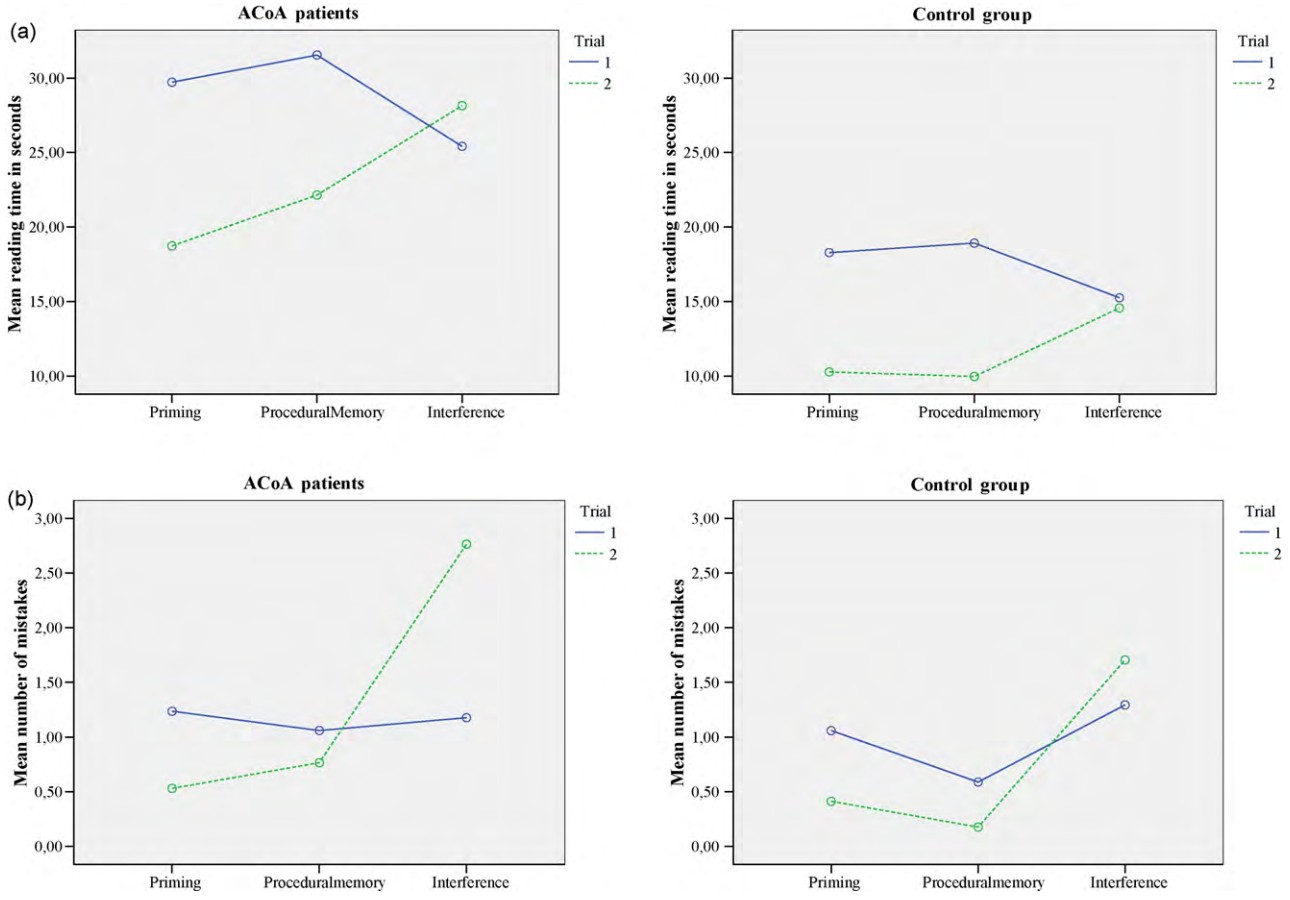

Fig. 1. (a) Results in mirror reading: reading speed. (b) Results in mirror reading: reading mistakes.

that, as assessed here, we found no relationship between any of our false memory measures and executive functions.

3.4. Lesion analysis

From our total of 18 patients we could obtain 10 clinical scans (2 MRT and 8 CT scans). Lesion location (see Table 7) was determined by a senior neuroradiologist blind to the neuropsychological findings. The poor quality of most of the available scans due to technical artifacts (e.g. clips) did not allow precise lesion location in each individual or determining lesion overlap of the group. However, on a descriptive level we proceeded in the following way. First, we divided the ten patients in high- and low-confabulators according to their Confabulations-Score in the Confabulation Interview. Patients with a Confabulation Score of 1.5 standard deviations below the average of healthy controls' Confabulation Score were classified as high-confabulators, whereas patients with lower Confabulation Scores were termed low-confabulators. According to this classification each group contained five patients. Within the group of high-confabulators we found that four patients had lesions to the orbitofrontal cortex with three of them having bilateral damage. The fifth patient exhibited an extensive left-sided brain lesion with medial-temporal involvement and damage to the white matter within dorsal/prefrontal and parietal regions. Of the five low-confabulators, two patients also showed lesion to the orbitofrontal cortex, but in contrast to high-confabulators these lesions were unilateral right. In addition to their primary right-sided orbitofrontal damage, the two patients also showed signs of dorsolateral prefrontal involvement, in one patient probably extending to the right temporal lobe. Of the three patients that presumably were not suffering from orbitofrontal damage, two showed lesions in dorsolateral prefrontal cortex with additional white matter atrophy in one case, and minor pathology of the temporal cortex in the other case. The last low-confabulator showed restricted damage to the basal forebrain.

In sum, nearly all patients showed signs of damage to the prefrontal cortex regardless of whether they were high- or low-confabulators. However, four of five patients that were more susceptible to provoked confabulation showed damage to the orbitofrontal cortex – a result that is typically found in confabulating patients. Only one high-confabulating patient did not show signs of orbitofrontal involvement, but suffered from widespread brain damage involving the medial-temporal, parietal and dorsolateral/prefrontal cortex. In the group of low-confabulators three patients shared damage to the dorsolateral prefrontal cortex, one patient had restricted basal forebrain damage and two patients showed orbitofrontal lesions. However, compared to high-confabulators these orbitofrontal lesions were unilateral. Thus, bilateral damage to the orbitofrontal cortex may be the most distinct feature of high- and low-confabulators among the ten patients examined here.

4. Discussion

The results of our study suggest that rupture of aneurysms of the ACoA may enhance susceptibility to different manifestations of false memories, although our results indicate that ACoA patients – four weeks after neurosurgery – on the whole are only mildly producing false memories, especially compared to other patients groups, for example patients with Korsakoff's syndrome. Likely, there might have been more pronounced false memories

in the acute state of their illness (Schnider, Ptak, von Daniken, & Remonda, 2000). However, even at a later post-surgery time, our ACoA patients did, as a group, produce more confabulations in the Confabulation Interview and were also more susceptible to false recognition of interference items in mirror reading than their healthy matched comparison group.

Moreover, in the ACoA group only, we found a significant correlation between confabulations in the Confabulation Interview and errors (i.e., false recognition of interference words) in our mirror reading task. This suggests that these two tests probably measure at least some common aspects of false memories. In contrast to these results, in the DRM-paradigm – a verbal learning task prominent for inducing false memories in healthy subjects – ACoA patients did not differ in false recognition from healthy subjects neither to related nor to unrelated lures. Furthermore, no relationship with the two other false memories indices was found indicating that false memories in the DMR-paradigm are dissociable from the other two and therefore probably represent a distinctive entity of false memories.

Taking a look at the single tests we used to evaluate false memories, ACoA patients showed inferior memory performance compared to their healthy controls in the Confabulation Interview, beside their slightly increased susceptibility to provoked confabulation. Comparisons between the groups revealed differences for the three domains: episodic memory, general semantic memory and orientation. In all of these, the ACoA patients gave fewer correct answers than controls, with the only significant difference in confabulations emerging in episodic memory. Although only a trend effect probably due to the limited total number of confabulations, this result indicates that confabulation in episodic memory may be most suitable to distinguish between healthy and brain damaged individuals. Hence, in accordance with various other studies our results suggest that after brain injury, episodic memory seems to be most vulnerable to confabulations compared to all other memory domains (Borsutzky et al., 2008; Dalla Barba, 1993a; Dalla Barba, Cappelletti, et al., 1997; Fotopoulou, Conway, Griffiths, et al., 2007; Kato & Anamizu, 2001; Kopelman et al., 1997; Van Damme & d'Ydewalle, in press).

As was the case in the Confabulation Interview, in the DRM-paradigm ACoA patients exhibited an impaired memory performance, too: They recalled and recognized significantly fewer studied words than healthy participants. However, patients and controls did not differ in false recall and false recognition of non-presented critical lures or unrelated lures. These results are in line with other patient studies that failed to find increased or even found lower false recognition rates to related lures in amnesic patients (Ciaramelli et al., 2006; Melo et al., 1999; Schacter & Slotnick, 2004; Schacter, Verfaellie, & Pradere, 1996). This finding is usually attributed to deficits of amnesics in extracting or remembering gist information from the word lists making them less susceptible to remember or recognize semantically associated lures (Dodson & Schacter, 2002; Schacter & Dodson, 2001; Schacter & Slotnick, 2004). Moreover, some studies revealed reduced false recognition proportions to related lures together with increased false recognition proportions to unrelated lures in confabulating patients, which was interpreted as defective strategic monitoring at memory retrieval (Ciaramelli et al., 2009; Ciaramelli et al., 2006; Gilboa et al., 2006; Melo et al., 1999; Van Damme & d'Ydewalle, in press). We did not replicate this pattern of result, since our ACoA patients were not more susceptible to false recognition of unrelated lures than their comparison subjects. This may have several reasons: Primarily, in contrast to other studies (Ciaramelli et al., 2009; Ciaramelli et al., 2006; Van Damme & d'Ydewalle, in press) our unselected group of ACoA patients included only mild confabulators, as the results in the Confabulation Interview suggest. Probably, increased false recognition to unrelated lures would only appear in more severe forms of confabulatory syndromes such as Korsakoff's syndrome (Van Damme & d'Ydewalle, in press, 2010) or even only in spontaneous confabulators (Ciaramelli et al., 2006; Melo et al., 1999). Furthermore, executive dysfunctions rather than confabulatory tendencies may be responsible for false recognition of unrelated lures in the DRM-paradigm. Thus, while in other studies patients suffered from executive dysfunctions (e.g. Ciaramelli et al., 2009; Melo et al., 1999), our patients scored within the normal range (with the exception of verbal fluency). Consequently, possible underlying dysexecutive processes causing false recognition in the DRM-paradigm may have been intact in our ACoA patients', and this may be the reason that they were as unsusceptible as controls to false recognition of unrelated lures. Methodological aspects may also account for the divergent pattern of result: In contrast to other studies we only employed five word lists at study and presented only 25 words in the recognition test to avoid fatigue and exhaustion in our patients. Probably due to a lower total number of items it may have been easier to differentiate between studied words and critical/non-critical lures. Thus, a possible impairment may not have become apparent with this version of the DRM-paradigm.

While ACoA patients exhibited memory impairments in all of the verbal memory tests that we applied, their abilities in non-declarative memory seemed to be intact. For example, between two task administrations of our mirror reading task patients improved in reading speed of most words – as did their comparison group. However, both groups did not improve from trial 1 to trial 2 in reading of interference words (i.e., trial 2 words that look similar but are not identical to corresponding items from trial 1). This result is in accordance with our assumption that reading of the interference items in trial 2 would require more cognitive resources because the priming effects due to trial 1 would have to be suppressed. Even if reading times for these words were not significantly lengthened, in contrast to priming and procedural memory items no improvement was found, so that the results are generally in line with our original assumption.

Regarding reading mistakes with respect to priming and procedural memory, again groups did not differ from each other. However, ACoA patients tended to make more reading mistakes in interference items in trial 2 compared to trial 1, whereas in the comparison group no difference could be revealed. This finding matches well with our expectations and suggests that false memories, i.e. false recognition goes beyond declarative memory and may also occur in the procedural memory domain. Furthermore, susceptibility to false recognition in procedural memory seems to be restricted to brain damaged individuals, since healthy subjects did not show this tendency.

Correlations between provoked confabulations (Confabulation Interview) and false recognition of interference words (mirror reading) suggest that these two tests share some underlying faulty mechanism, whereas false recall and false recognition in the DRM-paradigm probably depend on other parameters. This assumption is further strengthened by the pattern of results with respect to the correlations with our neuropsychological test battery: Whereas in the ACoA group the Confabulation Score as well as the reading mistakes of interference words had negative correlations with memory performance (delayed recall in the Memo-Test and the Rey-Osterrieth Figure), there was no relationship between false recall or false recognition in the DRM-paradigm and memory performance. This connection between false memories and memory disturbances is in line with various other studies (DeLuca, 2000; Fischer et al., 1995; Tallberg & Almkvist, 2001; Turner et al., 2008) postulating that memory disturbances are at least a necessary condition for false memories, especially in the case of confabulation. However, since our ACoA patients showed no relevant deficits in procedural skill learning, but nonetheless simultaneously slightly increased false recognition, memory impairment alone cannot be a sufficient condition. Accordingly, various neuropsychologi-

cal approaches postulate that the incidence of false memories or in particular the occurrence of confabulations depends on faulty mechanism within memory retrieval (for a review see Gilboa & Moscovitch, 2002; Metcalf et al., 2007; Schnider, 2008). Although determining the precise involvement of these various mechanisms is beyond the scope of our study, our results correspond mostly with the assumption that false memories may be due to an inability to suppress other emerging, but irrelevant memories (Ciaramelli & Ghetti, 2007; Ciaramelli et al., 2009; Schnider, 2003; Schnider & Ptak, 1999). This assumption is obvious for the following reasons: Firstly, the confabulations in the Confabulation Interview affect mainly remote memory that is acquired before the brain damage (rupture of aneurysm). Hence, patients possess – even if autobiographical or semantic memory may be somewhat reduced – a sufficient pool of available remote memories. Secondly, in mirror reading, ACoA patients exhibited intact procedural memory learning. They were able to learn and store new procedural skills. Thus, in both cases patients may rely on a (partly) functioning memory system which in general allows the retrieval of information from long-term memory. In contrast, in the DRM-paradigm patients failed to store new information that may have led to reduced correct memories but also reduced false memories. Hence, our patients may have only exhibited false memories if they had been able to retrieve gist information from long-term memory. This suggests that within memory retrieval (concomitant) activated, but irrelevant memories cannot be adequately inhibited and consequently false memories may arise. Although Schnider (Schnider, 2003, 2008; Schnider et al., 1996) postulates that this failure in suppressing task-irrelevant information only applies to spontaneous confabulators and would distinguish spontaneous confabulations from other forms of false memories, Gilboa et al. (2006) found evidence that a deficit to suppress irrelevant memories also occurs in non-spontaneous confabulating ACoA patients. To conclude, our results support the assumption that provoked confabulations have to be distinguished from intrusions and false recognition in classical verbal learning tasks (see also Kessels, Kortrijk, Wester, & Nys, 2008) and therefore cannot be regarded as the same phenomenon. However, the common ground of provoked confabulation and false recognition in procedural memory needs closer consideration in future studies.

As was the case in a previous study (Borsutzky et al., 2008), we could not reveal any relevant relationship between false memory parameters and components of executive functions that were examined here. Overall, our ACoA patients did not suffer from an obvious dysexecutive syndrome sometimes observed after ACoA aneurysm rupture (DeLuca & Diamond, 1995), with the only significant frontal-lobe associated impairment in a verbal fluency task. The involvement of executive dysfunctions in confabulations or other forms of memory errors is unresolved and previous findings are quite heterogeneous. It may depend on the specific pattern of frontal brain damage to what extent false memories are accompanied by executive dysfunctions. Gilboa and Moscovitch (2002) emphasize that brain areas crucial for confabulation differ from those responsible for executive function. Due to their analysis of 79 spontaneous confabulating patients, they concluded that the medial prefrontal cortex is mainly involved in spontaneous confabulation, whereas brain regions relevant for performance in standard executive tests of executive functions affect rather lateral frontal areas.

Quite unexpectedly, we found the Confabulation Score to be correlated with 'Reasoning' (LPS-4), some tests of attention (Word Trial of the FWIT and overall performance in the d2) and also with the copying performance of the Rey-Osterrieth Figure. One may argue that the performance in the subtest 'Reasoning' as well as that in the copy of the Rey-Osterrieth Figure depends on intact attention functions: In both tests relevant information has to be distinguished from irrelevant. Furthermore, 'Reasoning' is a test that is a highly speed-dependent intelligence measure requiring relatively intact attention abilities. As Ciaramelli and colleagues (Ciaramelli et al., 2009) have recently pointed out, processes of (divided) attention may modulate the amount of false recognition in confabulating patients. Further studies should address the impact of attention in the occurrence of false memories in more detail.

Unfortunately, brain images were not available for all our patients so that we could not include consistent neuroimaging data in our study. Furthermore, the clinical MRT or CT scans we could obtain for ten of our patients were of poor quality overall, so that precise determining of lesion locations was limited. Therefore, assumptions concerning the relationship between lesion site and false memories in our patient group are preliminary as well. However, on a descriptive level we found that the majority of those ten patients with available imaging data showed damage to regions of the prefrontal lobe and/or basal forebrain typically found after rupture and repair of ACoA aneurysm (Alexander & Freedman, 1984; Babinsky et al., 1997; Böttger et al., 1998; Damasio et al., 1985; Eslinger & Damasio, 1984). Furthermore, we found that six of the ten patients showed orbitofrontal lesions consistent with findings generally reported in spontaneously confabulating patients (Benson et al., 1996; Moscovitch & Melo, 1997; Schnider, 2003). Furthermore, only patients with bilateral orbitofrontal pathology as well as a patient with widespread brain damage showed a pronounced susceptibility to provoked confabulations (i.e., above 1.5 standard deviations over the norm) suggesting that the extent of brain damage may account for susceptibility to false memories. Notwithstanding, our findings are in line with the assumption that damage to ventromedial regions or to the orbitofrontal cortex respectively may be a necessary but not a sufficient condition for the production of confabulations (Gilboa & Moscovitch, 2002; Metcalf et al., 2007), since the majority of patients with lesions in this critical brain region does not confabulate at all (Dalla Barba, 1993b; Gilboa et al., 2006; Metcalf et al., 2007). In this spirit, several recent studies re-emphasize motivational, emotional and/or personality components as essential for the production of confabulations, additional to clear-cut neuroanatomical correlates (for review see Fotopoulou, 2008, 2009).

Beyond that, in accordance with various findings (for a review see Gilboa & Moscovitch, 2002) basal forebrain damage alone does not seem to be a sufficient cause to produce spontaneous confabulations, since our patient group included only one spontaneous confabulator. However, procedural and priming performance were not impaired in our ACoA patient group, indicating that procedural memory skills as well as priming are not dependent upon the basal forebrain (see also Bondi et al., 1993; Thomas-Anterion et al., 1996).

To summarize, our results suggest that ACoA aneurysms or damage to the basal forebrain may lead to a modestly enhanced vulnerability to certain false memories, i.e., provoked confabulation and false recognition in procedural memory. Conversely, we were not able to find increased false recall or false recognition in the DRM-paradigm neither to related nor to unrelated distracters in our patient group. Memory deficits that were present in our patients seem to be a necessary, but not a sufficient condition for false memories. However, executive functions examined here, and mostly intact in our patients, were not related with the false memory indices of our study. Compatible with several theoretical accounts postulating that defective processes within memory search, such as an inability to suppress irrelevant memories, are involved in the occurrence of false memories, our patients only produced false memories whenever they actually had a sufficient level of memories (or learning) available. Finally, our results confirm the assumption that provoked confabulations are dissociable from false recall and false recognition in verbal learning tasks such

as the DRM-paradigm and therefore represent a distinctive entity of false memory phenomena. Even if underlying defective mechanisms responsible for false recognitions in procedural memory have to be examined in more detail in future studies, for the first time, we could provide evidence that false memories may also occur in procedural memory.

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