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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).