NOTE

## CONFABULATION FOLLOWING RUPTURE OF POSTERIOR COMMUNICATING ARTERY

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### ABSTRACT

In this study we report a patient, MG, who following rupture of left posterior communicating artery exhibited an amnesic-confabulatory syndrome. Neuropsychological examination showed severe impairment on episodic memory tasks, which were marred by florid but plausible and semantically appropriate confabulation. Performance on tasks involving various kinds of semantic knowledge was normal or only mildly impaired.

Performance on tasks traditionally considered sensitive to frontal dysfunction was severely impaired with the exception of Cognitive Estimates where MG's performance was completely normal. There was no evidence of structural (CT scan) or metabolic (SPECT) damage to the frontal lobe. It is argued that tasks traditionally considered sensitive to frontal dysfunction are not specifically implemented by cognitive resources based on frontal structures. MG's confabulation is discussed in terms of a possible disruption of cognitive functions involved in the control of the subjective experience of feeling of remembering.

## INTRODUCTION

Confabulation, the unintentional production of actions and verbal statements that are incongruous to the patient's history, background and present situation (Dalla Barba, 1993a), is a symptom that accompanies many neuropsychological disorders.

The quality of confabulation's content can vary considerably, ranging from minor distortions of facts and information to really implausible and bizarre reports. According to its content, a distinction between two different types of confabulation have been proposed (Bonhoeffer, 1904; Berlyne, 1972; Kopelman, 1987). The first type involves minor distortion of true autobiographical events or their misplacement in time and place and is usually produced in response to specific questions, whereas the second consists of the spontaneous fabrication of fantastic and implausible tales. Following a different perspective, Dalla Barba (1993a, 1993b) suggested that the confabulation content varies along a gradient from minor distortion to bizarre and semantically anomalous tales according to the level of semantic memory impairment.

A number of ideas about mechanisms underlying confabulation emphasise the role of a frontal executive dysfunction in confabulation (Baddeley and Wilson, 1986; Kopelman, 1987; Baddeley and Wilson, 1988; Moscovitch, 1989; Delbecq-Derouesne, Beauvois and Shallice, 1990; Johnson, 1991; Moscovitch, 1995; Burgess and Shallice, 1996). The impairment of executive functions resulting from damage to the frontal lobe would prevent both the active process of retrieval and the evaluation of its output and thus would be directly responsible for confabulatory behaviour. However, two patients have been described with a confabulatory syndrome, limited to the autobiographical component of episodic memory, where there was

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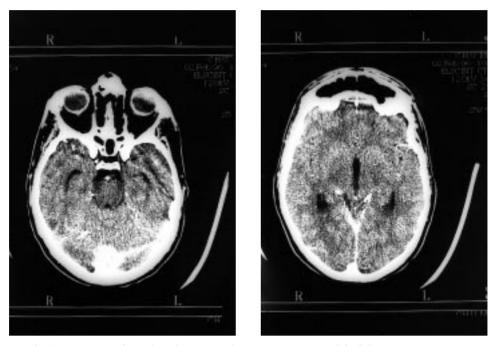


Fig. 1 – Contrast-enhanced CT brain scan showing an aneurysm of the left posterior communicating artery and signs of subarachnoidal haemorrage, in the absence of frontal and thalamic lesions.

no frontal pathology and spared executive functions (Dalla Barba, Cipolotti and Denes, 1990; Dalla Barba, 1993a). In addition the confabulating patient described by Delbecq-Derouesne, Beauvois and Shallice (Delbecq-Derouesne et al., 1990) had a documented frontal lobe lesion but performed normally on executive tasks supposed to be sensitive to frontal lesion. These findings cast doubts both on the hypothetical prominent role of a frontal-executive dysfunction in confabulation and on the anatomical basis of executive tasks.

In this study we describe a patient, MG, who developed an amnesic-confabulatory syndrome following the rupture of an aneurysm of the posterior communicating artery (PCoA). While several studies report confabulation following the rupture of the anterior communicating artery (ACoA) (Luria, 1976; Stuss, Alexander, Lieberman et al., 1978; Kapur and Coughlan, 1980; De Luca and Cicerone, 1991), to our knowledge this is the first case report of confabulation following the rupture of an aneurysm of the PCoA. In addition MG showed a massive impairment on tasks supposed to be sensitive to frontal dysfunction without showing any evidence of damage to the frontal lobe or related structures.

## CASE REPORT

MG is a 42-year-old right-handed clerk with a high-school education, with no history of psychiatric or neurologic diseases. On February 2, 1996 he was admitted to the Neurology department of the Hôpital Henri Mondor for a meningeal syndrome with neck rigidity without photofobia and nausea. A CT brain scan performed on that day showed a small intra-ventricular haemorrhage involving both occipital horns. There were no signs of any focal lesion. In particular, there was no evidence of frontal or thalamic lesion (Figure 1). Also, in this and subsequent scans there was no evidence of hydrocephalus. A brain arteriography revealed the presence of an aneurysm of the left posterior communicating artery. On a second CT scan performed three days later the haemorrhage was no longer observable and there were no signs of residual focal lesions. On February 9 he was operated in order to clip the aneurysm of the

PCoA. A SPECT HMPAO perfusion scanning 13 did not show any brain hypoperfusion. A third CT scan performed ten days after the operation was unremarkable.

Since his admission to the hospital MG was never in a confusional state nor has he ever shown any minimal sign of confusion. Indeed, apart from his verbal confabulation, he behaved appropriately and was totally independent in his daily life at the hospital. For example, he never got lost inside the hospital, he frequently went to the coffee shop located on the ground floor, and would return to his room located on the third floor. He also used to leave the hospital without permission, but he always came back for lunch and dinner. He is a charming and kind man who used to talk to other people in the ward, and nobody, apart the authors of the present paper, ever realised that he was suffering from any neuropsychological impairment.

## Neuropsychological Examination

MG was tested on various occasions between February and March 1996. His collaboration during testing sessions was excellent. MG's performance on the WAIS is given in Table I. He obtained a Verbal IQ of 82 and a Performance IQ of 73 (full scale = 75). On bedside tests of oral expression, understanding of oral language, reading, writing, neglect, praxis and calculation, the patient was judged to be normal.

A variety of memory tests, including the Wechsler Memory Scale (WMS) (Table II) was administered to MG. He achieved a MQ of 62. On the recall of the Logical Memory Passages he produced completely confabulatory takes. On the Paired Associate subtest he was able to recall only 4 easy and 2 difficult items in three trials and produced 19 intrusions. On forced-choice recognition tests MG's performance was also markedly impaired. On these tests (Warrington, 1984), 50 items were presented one after the other for 3 s each, together with the orienting task used by Warrington (1984) (the patient had to say whether he found

Verbal IQ	82
Performance IQ	73
Full scale	75
Information	11
Comprehension	7
Arithmetic	6
Digit span	5
Vocabulary	5
Similarities	8
Digit symbol	5
Picture completion	9
Block design	3
Pictures arrangement	3
Objects assembly	1

TABLE I MG's Standard Scores on the WA.I.S.

TABLE II MG's Scores on the Wechsler Memory Scale

	MG	Mean for his age	S.D.
MO	62		
Personal information	5	5.90	0.39
Orientation	3	4.95	0.27
Mental control	5	6.71	2.06
Logical memory	2.5	9.64	4.24
Digits	7	10.35	1.96
Visual reproduction	7	11.48	2.94
Paired associate learning	4	15.44	4.68
Tuned associate learning		15.11	

	MG	Comments
Modified Card Sorting Test <sup>1</sup>		
Categories achieved	1	Controls mean = 5, S.D. $1.6$
Error score	45.5	Controls mean = $6.6$ , S.D. 4
		Cut-off score for frontal lesion patients $\geq 50$
Verbal fluency		
3 categories, each 60 s (total retrieved)	24	Controls mean = $38.2$ , S.D. $12$
$FPL^2$ , each 60 s (total retrieved)	15	Controls mean = $36.5$ , S.D. 11
Cognitive Estimates <sup>3</sup> (error score)	4	Controls mean $=$ 3.6, S.D. 1.92
e v		Mean of left frontals = $8.9$ , S.D. $4.3$
Graphic sequences (total correct) <sup>4</sup>	48	Controls mean $= 78$ , S.D. 15
Trial Making Test <sup>5</sup> (seconds)		
A	66	Controls mean $=$ 30, S.D. 10
В	240	Controls mean = $75$ , S.D. $36$
Stroop Test <sup>6</sup>	20	Normal values $\geq 35$
Anosognosia Index <sup>7</sup>	14.5	Controls mean = $4.3$ , S.D. $2.1$

TABLE III MG's Performance on "Frontal Lobe" Tasks

<sup>1</sup>Nelson, 1976. <sup>2</sup>Ramier and Hécaen, 1970. <sup>3</sup>Shallice and Evans, 1978. <sup>4</sup>Luria, 1966. <sup>5</sup>Davies, 1968. <sup>6</sup>Stroop, 1935. <sup>7</sup>Della Barba et al., 1995.

the item pleasant or unpleasant). He was not told that his memory for the items was tested using a two-alternative forced choice recognition procedure. Two different forms were used: in the first stimuli were words, in the second they were faces. He achieved a score of 39 (mean for his age = 45.5, S.D. 3.1) and of 31 (mean for his age = 44.7, S.D. 3.3) on the words and faces tests respectively.

MG's level of performance in tasks involving various kinds of semantic knowledge, including linguistic, visual, arithmetic, historical and geographical, was normal or only mildly impaired. However, even when he was not able to retrieve the correct information, MG never confabulated in this kind of task.

MG did not show any direct evidence of structural or functional lesions involving the frontal lobe or related structures such as the thalamus or the thalamus-prefrontal projections. However, since a deficit of frontal-executive functions has often been invoked as a prominent factor in confabulation, MG's performance was assessed on tasks thought to be sensitive to frontal lobe lesions. MG's performance on this set of tasks (Table III) was severely impaired with the exception of the Cognitive Estimates (Shallice and Evans, 1978) where he scored well, without producing any extreme response. However, although his performance was severely impaired on these tasks, from a clinical point of view MG did not show a clear frontal syndrome. In particular there was no evidence of grasping and snout reflex, utilization behaviour and changes in mood and self control.

MG was completely unaware of his memory difficulties and he never admitted explicitly to having memory problems, either spontaneously or when specifically requested by the examiner.

MG produced most of his confabulations in informal conversation, both in answering specific questions and spontaneously, mainly referring to autobiographical episodic memory. Confabulation was always plausible and semantically appropriate so that an hypothetical observer not familiar with MG's history, background and present situation could hardly tell whether MG's report were confabulatory or not. For example, while he was waiting to undergo the CT scan, MG told the radiologist that he had accompanied a friend to be admitted to the neurology department that day. The neurologist who was taking care of MG's friend realized that MG also had neurological problems and so decided to refer him to the radiology department for a CT scan. On that occasion the radiologist did not even suspect that MG was confabulating.

Confabulation and intrusions were massively present in learning tasks such as Logical Memory and Paired Associate. For example on the Logical Memory task, the recall of the correct short story, concerning a cleaning woman with four hungry children who was robbed while she was on her way home, was replaced by the following tale: "It is a story about a woman living in a small flat. Somebody rang at her door, she opened it and saw in front of her a man who had first threatened her and then beaten her and stolen her savings. She asked her children to go to the kitchen and not to move from there. She then went to the police where she was asked to usual questions."

To further examine MG's confabulation, we submitted him to the Confabulation Battery (Dalla Barba, 1993a), including questions probing personal, general and linguistic semantic memory, episodic memory, orientation for space and time and questions to which the appropriate response would be "I don't know", both semantic (e.g. "What did Marilyn Monroe's father do?") and episodic (e.g. "Do you remember what you did on March 13, 1985?". It was found that confabulations appeared only in response to questions probing episodic memory and orientation for space and time (see Appendix).

Between three and four weeks after the vascular accident, MG's clinical picture started to improve significantly. Confabulations disappeared first, followed by gradual improvement of the performance on frontal executive tasks. Eventually also memory functions started to improve. By the end of March MG showed only mild learning deficit and some perseveration.

## DISCUSSION

The patient we described had a florid confabulatory syndrome confined to episodic memory tasks. He was impaired on learning tasks and had relatively well preserved semantic knowledge. In addition he was severely impaired on a set of tasks supposed to measure frontally based executive functions, with the exception of Cognitive Estimates where his performance was normal. However, there was no evidence of structural or functional damage to the frontal lobe or to brain structures functionally related to the frontal lobe. Also, from a clinical point of view MG did not show a clear frontal syndrome.

In addition to his impaired memory abilities MG showed a general intellectual impairment as measured by the WAIS. It could be argued that MG's general intellectual and memory impairment, as well as his confabulation, were part of a general confusional state. However, this was clearly not the case. In fact, not only from a clinical point of view MG was not confused, but the type of responses he gave in formal testing did not resemble those usually given by confused patients. Confused patients usually tend to produce bizarre and in some sense confabulatory-like responses in tasks demanding general intellectual abilities and semantic knowledge, such those included in the WAIS. This was never the case for MG. For example on the Vocabulaty subtest of the WAIS, where his performance was very defective, MG produced plausible definitions of words that were scored poorly because they did not meet the scoring criteria of the WAIS. On the Similarities subtest he scored quite well and even for the last three items where he scored zero his responses were plausible. So it seems improbable that MG's general intellectual deficit is due to confusion. MG's general intellectual decline may be the result of a non-specific effect of the lesion upon the cognitive system, so that even in the absence of confusion, a variety of cognitive resources were temporarily not accessible. It must be noted, however, that MG's general intellectual decline as measured by the WAIS was not confirmed by clinical impression or by other bedside tests of semantic and general cognitive abilities (see Neuropsychological Examination).

Confabulation has been associated to lesions involving the ventromedial portion of the frontal lobes and related structures, including the basal forebrain, septum, fornix, cingulate gyrus, cingulum, anterior hypothalamus and the head of the caudate nucleus (Kapur and Coughlan, 1980; Alexander and Freedman, 1984; Wilkki, 1985; Moscovitch, 1989; De Luca and Cicerone, 1991; Irle, Wowra, Kunert et al., 1992), all brain regions fed by the ACoA. However damage to other frontal regions has also been linked to confabulation (Stuss et al., 1978; Baddeley and Wilson, 1986). In view of the fact that all these patients with documented frontal lesions and confabulations also showed poor performance on tests supposed to measure frontal executive functions, it has been suggested that impairment of executive functions may play a special role in confabulation. At least two kinds of observations, however, seem to be in contrast with the hypothesis of a crucial role of executive dysfunction in

confabulation. In fact, on the one hand it has been shown that confabulating patients with frontal lesion may perform normally on tasks supposed to be sensitive to executive dysfunction (Delbecq-Derouesne et al., 1990). On the other hand, confabulation can occur in patients without frontal lobe pathology and with normal performance on tests that are sensitive to frontal damage (Dalla Barba et al., 1990; Dalla Barba, 1993a). The present case casts additional doubts on the association between confabulation and frontal pathology.

Moscovitch (1989, 1995) distinguishes between two components of retrieval. One, associative retrieval, is relatively automatic and independent from frontal functions. The other, strategic retrieval, is self-initiated, goal-directed, effortful and intelligent. Within strategic retrieval processes, two further components are hypothesized. The first involves organising a memory search that uses whatever knowledge is available, whether semantic or episodic. Once knowledge is recovered, a second strategic process is involved in monitoring the output of the memory search and checking whether it is consistent with other information in semantic and episodic memory. Similar proposals have been advanced in the literature on normal (e.g.: Mandler, 1980; Tulving, 1983) and pathological memory (e.g.: Baddeley and Wilson, 1986; Delbecq-Derouesne et al., 1990; Johnson, 1991). According to Moscovitch (1995) the disruption of the monitoring subcomponent of strategic retrieval would be responsible for confabulation. However, there are some difficulties in considering MG's confabulation as the result of a monitoring deficit. If this were the case confabulation would have appeared every time the strategic component of retrieval was involved, i.e. both in episodic and semantic memory tasks. In fact, MG confabulated only in episodic memory tasks, whereas in semantic tasks he either was correct or he responded "I don't know". It could be argued that MG confabulated only on episodic memory tasks because semantic tasks required retrieval that was more associative than strategic. However, apart from the risk of falling in the circularity of considering as strategic retrieval demanding only those tasks where confabulations are observed, there is no good reason to think that a task like the WAIS Vocabulary subtest. where MG did not confabulate, demands less strategic retrieval than the WMS Logical Memory subtest, where he did confabulate.

A further difficulty in considering MG's confabulation the result of a deficit of the strategic component of retrieval comes from his performance on tests considered sensitive to frontal dysfunction. The only one of these tests where MG showed a completely normal performance was that of Cognitive Estimates. This test requires that the subject answer questions which do not have a readily available correct answer (e.g. "How many camels are there in Holland?"), and the subject, in order to provide a plausible answer, is constrained to make a judgment involving the activation, coordination and control of several kinds of semantic information. Thus, strategic retrieval is expected to be prominently involved in the production of a plausible answer in this test, even more so than in other tests considered sensitive to frontal dysfunction.

However, MG's performance on frontal executive tests is interesting in itself because it confirms that performance on tasks supposed to measure executive functions can be dramatically impaired in the absence of frontal pathology. It is well known, for example, that patients with Alzheimer's disease, which involves predominantly posterior brain pathology, are very defective on these kind of tasks (see Spinnler, 1991, for a review). As is the case for any cognitive domain, it is almost impossible to devise tasks which are pure, in the sense that they measure only the cognitive domain for which they were devised. Accordingly, executive tasks can be failed in the absence of a specific, isolated disruption to executive processes. However, it is possible that, whatever its origin, the impairment on executive tasks shares some common underlying mechanism with confabulation. In particular, it is possible that the disruption of the same cognitive resources are responsible both for poor performance on executive tasks and for confabulation in episodic memory tasks. This kind of deficit could occur as a consequence of a frontal lesion or, as in MG, following a brain damage that spares the frontal lobe and in the absence of a confusional state.

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## APPENDIX

Examples of Confabulatory Responses to the Confabulation Battery (Dalla Barba, 1993a)

- Q. What did you do yesterday?
- A. I worked, as usual.
- Q. Who did you meet this morning? A. The Professor... I don't remember his name... with his pupils and nurses.
- O. How did vou spend last Christmas?
- A. I worked, I did nothing special.
- Q. What did you do for your last birthday? A. I had lunch with my parents... nothing special.
- Q. Do you remember the last time you went to see a doctor?
- À. It was when I lost my head... I found my-self in the emergency room of the Salpêtrière.